Exploring Disease Spread Dynamics: The SCIR Model and the Role of Asymptomatic Carriers

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Abstract

This paper presents the SCIR model, an adaptation of the traditional SIR framework, incorporating a new compartment for Asymptomatic infectives (C) to highlight the impact of asymptomatic carriers on disease spread. We explore the dynamics of infectious diseases within and across multiple populations, emphasizing the significance of asymptomatic transmission in our globally connected society. By applying the SCIR model, we aim to offer better insights for public health strategies, stressing the importance of including asymptomatic transmission in modeling efforts. Our study contributes to enhancing the understanding and management of infectious diseases, suggesting a nuanced approach to public health interventions.

1 Introduction

In our project, we explored a modification of the traditional SIR model for infectious diseases by incorporating a new group called Asymptomatic infectives (C), leading to our SCIR model. This addition reflects our understanding of how diseases can spread through individuals who don't show symptoms but can still transmit the infection. We recognized the role of asymptomatic carriers, especially in the context of recent global health challenges, and aimed to see how this factor might affect disease dynamics within and across communities.

We extended our model to consider the movement of people between different population groups, acknowledging our world's interconnected nature. By doing so, we hoped to gain insights into how diseases could spread more comprehensively, not just within isolated populations but across a network of interacting communities.

Our work is a small step towards better understanding the complex dynamics of infectious diseases. We recognize that our model, while offering some new perspectives, is just one of many tools researchers can use to tackle public health challenges. Through this exploration, we've learned a lot about modeling disease spread and the importance of considering various factors, including asymptomatic transmission. We hope our project can contribute in some way to the broader conversation on public health and disease management, and we look forward to seeing how these models evolve with future research.

2 Background

Infectious diseases modeling has become a vital tool in understanding and managing the spread of contagious illnesses, particularly highlighted by the onset of the COVID-19 pandemic. The emergence of the novel coronavirus and its swift global transmission in early 2020 necessitated an urgent response from the scientific community. The strategic importance of mathematical models was underscored as they offered insights into the potential trajectories of the virus's spread. These models, rooted in the classical SIR framework[1], have provided a foundation for predicting the dynamics of disease spread and the potential impact of public health interventions.

Given the complex and multifactorial nature of infectious disease spread, adaptations of the conventional SIR model were developed to incorporate additional relevant dynamics. For example, recent works have integrated vaccination into the SIR model, resulting in SIRV variations[2][3] that take into account the proportion of vaccinated individuals within a population. Such modifications allow for more accurate predictions and strategic planning in public health policy and vaccination campaigns.

This paper attempts to improve the SIR model from another perspective. We introduce the SCIR model, an adaptation of the traditional SIR framework, which includes a crucial compartment: the Asymptomatic infectives (C). The SCIR model emphasize the significant role that asymptomatic carriers play in the spread of infectious diseases.

The introduction of the C compartment is crucial because:

- A significant portion of the population infected with many communicable diseases may not show symptoms. These individuals are asymptomatic carriers.
- Asymptomatic infected individuals can transmit the disease, just like their symptomatic counterparts. This characteristic is especially critical for diseases where asymptomatic transmission plays a significant role in the spread of the infection.
- Although it is not problematic to categorize both symptomatic and asymptomatic infected individuals as infected if they have the same infectious capacity in a single population, in the case of multiple interconnected groups, asymptomatic individuals may bridge the gap between population groups and unknowingly spread the disease between these population groups.

Our study extends the SCIR model to multi-population scenarios, examining the interconnectedness of various population groups and the role of asymptomatic carriers in bridging these communities. This approach is particularly relevant in our globally connected world, where movement between population groups can unknowingly catalyze the spread of disease. By incorporating multiple populations into the SCIR model, we can explore the dynamics of disease spread within and between communities, providing valuable insights for targeted interventions and containment strategies.

3 Model

3.1 SCIR model for a single population

We divide infectives of the classic SIR model into two groups: symptomatic infectives (I) and asymptomatic carriers (C). In our model, as shown in Diagram X, these two groups play very similar roles—they indeed carry the virus, can infect susceptibles (S), and can recover over time to become part of the recovery (R). This is one reason why they are not distinguished in the classic SIR model. Like the classic SIR model, we have the pathway $S \to I \to R$. Correspondingly, we also have $S \to C \to R$. Moreover, as most infectious diseases have a latent phase where some infectives are initially asymptomatic but develop clear symptoms after a period, our model includes the transition $C \to I$ to represent this transformation from asymptomatic to symptomatic.

The parameters are defined as follows:

- β: The transmission rate at which susceptibles become infected by symptomatic infectives.
- α: The transmission rate at which susceptibles become infected by asymptomatic carriers.
- γ: The recovery rate for symptomatic infectives.
- ω: The recovery rate for asymptomatic carriers, which can be different from γ to reflect a distinct recovery process for asymptomatic individuals.
- δ: The rate at which asymptomatic carriers develop into symptomatic infectives.

Also, several assumptions are made to simplify the dynamics of disease transmission and recovery in our SCIR model:

- The total population is constant; the model does not incorporate birth or natural death rates.
- The contact rate among individuals in the population is uniform, meaning that every individual has an equal chance of coming into contact with others, which is a simplification of real-world social interactions.
- There's no distinction between the different routes of disease transmission. The model assumes a single mode of transmission that applies uniformly across the population.
- Individuals in the recovered category are assumed to have immunity and are not susceptible to reinfection.
- Both symptomatic infectives and asymptomatic carriers can infect susceptibles with the same capacity for transmission.
- Upon infection, susceptibles can develop into either symptomatic infectives or asymptomatic carriers, with different likelihood of becoming an infective and a carrier.

- Both I and C can recover and transition to the recovery class, but they do so at different rates.
- Carriers have the potential to progress to infectives.
- All rates described, such as transmission, recovery, and progression, are considered to be constant over time, implying that the parameters are constants with respect to time.
- The effect of potential interventions, such as vaccination or quarantine measures, is not included in this basic model structure.

The dynamics of the model are described by the following set of ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta S(I+C) - \alpha S(I+C) \\
\frac{dI}{dt} &= \beta S(I+C) - \gamma I + \delta C \\
\frac{dC}{dt} &= \alpha S(I+C) - \omega C - \delta C \\
\frac{dR}{dt} &= \gamma I + \omega C
\end{aligned}$$

These equations describe the change in the number of individuals in each compartment over time, with each term corresponding to a specific epidemiological process such as infection, recovery, and transition from asymptomatic to symptomatic states.

3.2 SCIR model including travelling

Now we extend the model to multiple populations and take travel between populations into account. For each population, we apply the single population SCIR model in section 3.1. In this model, each population has its own S, C, I, and R. For generality, we will allow the parameters to vary from population to population.

Also, several assumptions are made for this model as follows:

- Different populations have their own S, C, I, and R compartments, along with corresponding parameters which may differ based on real-world conditions.
- All assumptions made in the single population SCIR model are applied to this model.
- Populations are connected through a network that defines the potential paths of travel between them, and this network structure is fixed throughout the simulation period.
- Different populations implement their own rules for allowing or restricting the movement of individuals between groups, without "entry-only" or "exit-only" scenarios—movement is either permitted or not.
- Within the same group of the same population, all individuals are subject to the same movement regulations without discrimination.
- There is no direct transmission of the disease between populations without travel; that is, the disease spreads from one population to another solely through the movement of infected individuals.

The dynamics of the model are described by the following set of ordinary differential equations:

$$\begin{aligned}
\frac{dS_i}{dt} &= -\beta_i S_i(I_i+C_i) - \alpha_i S_i(I_i+C_i) + \sum_j T^S_{j,i} S_j - \sum_j T^S_{i,j} S_i \\
\frac{dI_i}{dt} &= \beta_i S_i(I_i+C_i) - \gamma_i I_i + \delta_i C_i + \sum_j T^I_{j,i} I_j - \sum_j T^I_{i,j} I_i \\
\frac{dC_i}{dt} &= \alpha_i S_i(I_i+C_i) - \omega_i C_i - \delta_i C_i + \sum_j T^C_{j,i} C_j - \sum_j T^C_{i,j} C_i \\
\frac{dR_i}{dt} &= \gamma_i I_i + \omega_i C_i + \sum_j T^R_{j,i} R_j - \sum_j T^R_{i,j} R_i
\end{aligned}$$

Using this model, we can employ data obtained from real-world scenarios to estimate the parameters within the model, thereby simulating various situations. This allows us to explore factors influencing the outbreak of infectious diseases.

4 Results

4.1 Basic reproduction number

The classic SIR model has a parameter R_0, which represents the infectious ability. The larger R_0 is, the stronger their ability to infect and the easier it is for epidemics to break out. After the epidemic, the proportion of R will be higher and the proportion of S will be smaller.

Like the classic SIR model, our SCIR model has its own R_0 to represent the ability of infectives and carriers to transmit the virus, which is obtained from the equilibrium of the dynamic ODEs.

In order to find the equilibrium of the ODE, we need to set $\frac{dI}{dt}$ and $\frac{dC}{dt}$ to zero:

$$\frac{dI}{dt} = \beta S(I + C) - \gamma I + \delta C = 0$$
$$\frac{dC}{dt} = \alpha S(I + C) - \omega C - \delta C = 0$$

After some rearranging, we get:

$$\frac{I}{C} = \frac{\delta + \beta S}{\gamma - \beta S} = \frac{\omega + \delta - \alpha S}{\alpha S}$$

Solve for S, we get:

$$S = \frac{\gamma(\omega + \delta)}{\alpha(\gamma + \delta) + \beta(\omega + \delta)}$$

Setting $R_0 = \frac{\alpha(\gamma+\delta)+\beta(\omega+\delta)}{\gamma(\omega+\delta)} S_0$, where S_0 is the number of susceptibles at the beginning time, we have that $\frac{S_{eq}}{S_0} = \frac{1}{R_0}$. In this case, the larger R_0 is, the proportion of R will be higher and the proportion of S will be smaller after epidemic. It is a well defined parameter to measure the infectious ability of the disease.

4.2 Scenario simulation

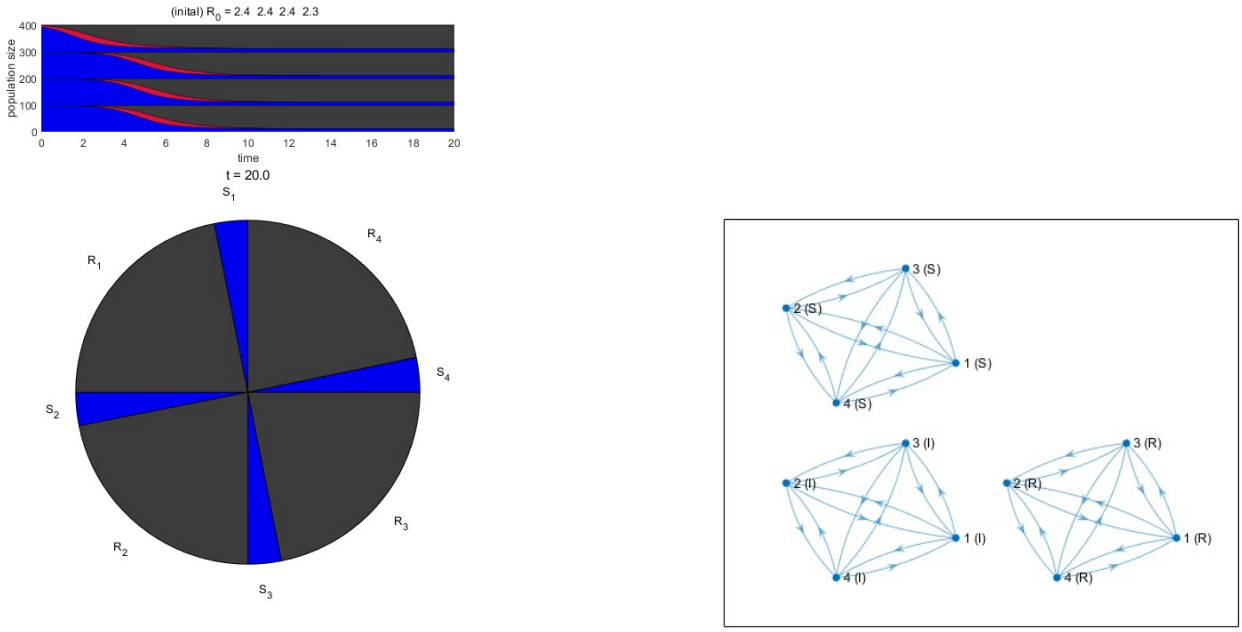
In our study, we modeled disease transmission in four different population groups of people. All people can travel between any populations, except symptomatic infectives. To ensure comparability between the two models, we set the basic regeneration number (R_0) to the same value for both the SIR model and the SCIR model as a way of controlling for the variable of the rate of viral transmission, thus comparing the effects of the control measures under the different models.

Simulation of the SIR model in figure 1a shows a rapid increase in the number of infected people in the absence of control measures. The high proportion of final recoverds reflects that most people gets infected and there's severe outbreak.

In figure 2a, implementation of control measures on infectives significantly reduces the proportion of final recovereds which means much less people get infected. This reflects effective disease management and control strategies.

In figure 3, under the SCIR model, the presence of asymptomatic infected persons, despite the same control measures as in the SIR model, implies that controlling only symptomatic infectives is not sufficient to effectively control the spread of disease. The greater proportion of final recoverds in the pie chart suggests that asymptomatic infectives are a non-negligible factor in the spread of disease.

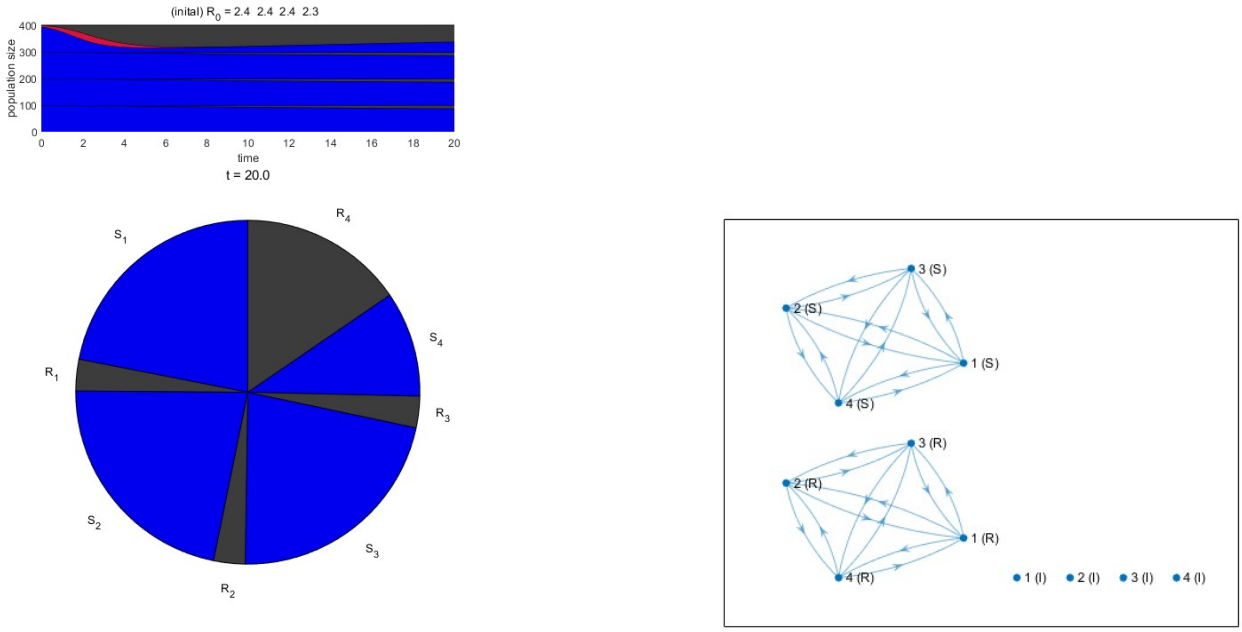
Our findings emphasize that asymptomatic infected individuals are capable of sustaining disease transmission even when symptomatic infectives are effectively controlled, which poses new challenges for public health intervention strategies. Therefore, more comprehensive measures are needed in the response to COVID-19 and similar diseases, which include, but are not limited to, increasing the frequency of testing to facilitate the detection and isolation of asymptomatic infectives , as well as increasing vaccination efforts to reduce the proportion of susceptible individuals. In addition, this also implies that models should focus more on the consideration of asymptomatic transmission when predicting and guiding policy decisions.



(a) SIR model without control on infectives

(b) SIR model with control on infectives

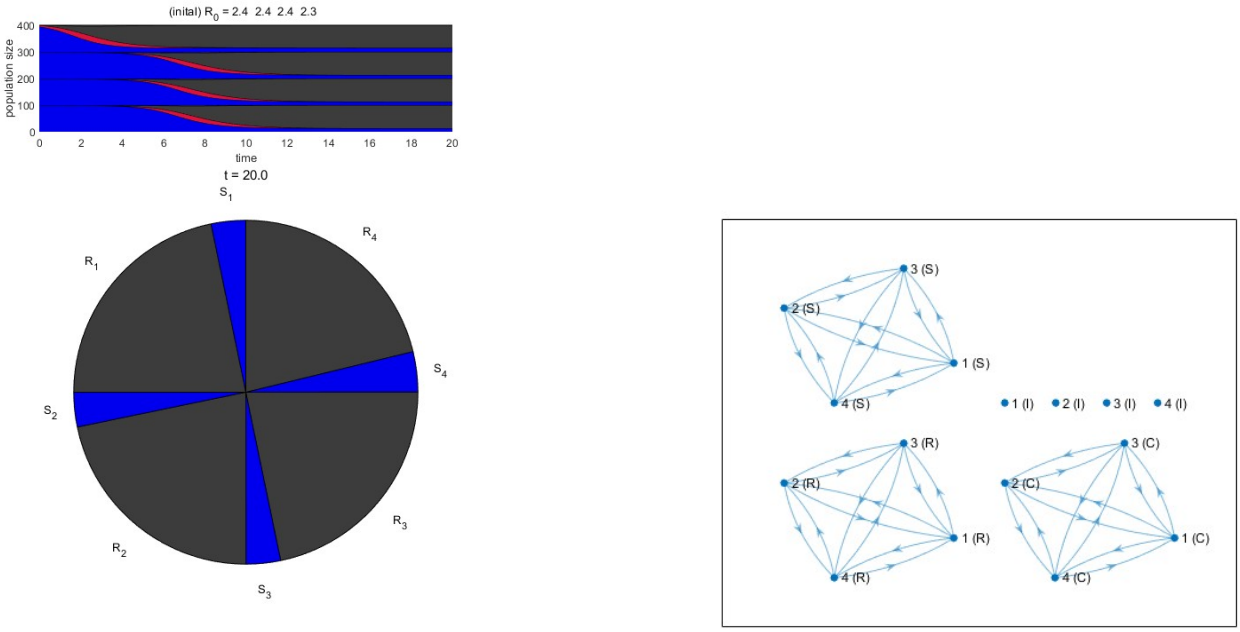
Figure 1: Result of SIR model simulation



(a) SIR model without control on infectives

(b) SIR model with control on infectives

Figure 2: Result of SIR model simulation



(a) SIR model without control on infectives

(b) SIR model with control on infectives

Figure 3: Result of SIR model simulation

5 Discussion

5.1 Limitations

The use of static parameters in the model presents a limitation in accurately predicting the course of an epidemic over time. Real-world infectious disease dynamics are influenced by a multitude of factors that change over time, including seasonal variations, changes in human behavior in response to the epidemic, and the implementation of public health interventions. Defining parameters as constant values does not account for these temporal variations, potentially leading to inaccuracies in the model's predictions. Utilizing real-world datasets to define parameters as functions of time t could provide a more accurate and responsive model that reflects the changing nature of epidemic conditions.

Also, the current assumptions of the model may oversimplify the complex interactions involved in the spread of infectious diseases. Real-world epidemics are influenced by a wide range of factors, including social, economic, and environmental conditions, which are not fully encapsulated by the model. For instance, the model does not differentiate between different population groups that may have varying susceptibility to the disease, nor does it account for the impact of healthcare system capacity on the epidemic's outcome. Improving the model to include a richer set of assumptions could lead to a more nuanced understanding of disease transmission and the effectiveness of control measures.

5.2 Future researches

Based on the model, we can define parameters using various sets of real-world data to simulate different scenarios. For instance, we can utilize epidemiological datasets from different cities to define the corresponding parameters for different populations, thereby creating a differentiated urban cluster scenario to simulate the spread of the epidemic. This approach may allow us to observe the impacts of the epidemic on different cities. We can also implement different isolation policies for different populations to verify the effectiveness of various quarantine measures in containing the spread of the epidemic. Indeed, even with the same model, choosing different datasets to define parameters can lead to research in different directions and yield diverse conclusions.

Moreover, additional assumptions and variations can be incorporated into the model. For example, we can include vaccination and secondary infections. In fact, vaccines and secondary infections have been attempted and

preliminarily included in our model but did not yield any meaningful conclusions. Due to the challenge of fitting a vaccine function over time through data, as well as the difficulties that secondary infections introduce to the ODE equilibrium in the short term, we ultimately abandoned modeling these aspects. Perhaps in future research, these elements can be added to the model and further refined.

Reproducibility

All code are available at: https://github.com/Hhnxxxxxx/SCIR-Model.git

References

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