

Reaction-diffusion spatial modeling of COVID-19 in Chicago

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Abstract

We examine an application of the Law of Mass Action to model the outbreak of COVID-19 in Chicago. We begin with a zero-dimension (ODE) compartmental epidemiological model consisting of Susceptible, Infected, and Removed populations (SIR model). We then develop a spatially distributed version of the model in the form of reaction-diffusion equations (PDE).

1 Introduction and Similar Work

2 ODE and PDE Model Setup

We begin by explaining the ODE model which is simply obtained from the full PDE model by removing the diffusion terms in the reaction-diffusion equations.

We start with a population of *susceptibles* (S), which may become *infected* (I) upon the emergence of the virus within the population. Infected individuals can interact with the susceptibles at rate β to draw new members into the group I of individuals infected by the virus. We note that the transmission rate β incorporates the total population size (ODE model) or the total population density (PDE model). A fraction of the infected population recover or die at a rate γ , giving rise to the *removed* (R) population.

These parameters specify the ODE process. They represent processes that occur in a “well mixed” situation when no spatial dependence is assigned, or processes that happen locally at every point in space for the PDE’s. We discuss parameter selection in the next section.

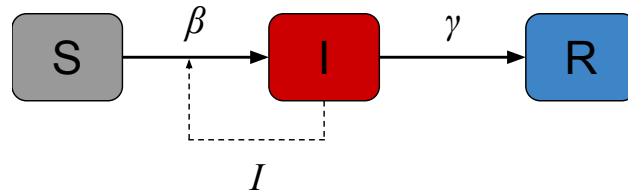


Figure 1: Schematic diagram of the SIR model. The dashed line denotes the interaction of the infectious population with the susceptible population that leads to infection.

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The population model at the PDE level is an autonomous diffusion with a source:

$$S_t = \nabla(\mathfrak{D}_s \nabla S) - \beta SI, \quad (1)$$

$$I_t = \nabla(\mathfrak{D}_I \nabla I) + \beta SI - \gamma I, \quad (2)$$

$$R_t = \gamma I. \quad (3)$$

In principle the R population can have a diffusion, but since we assume this population has immunity we assign $\mathfrak{D}_R = 0$.

As in [4], we have not incorporated any functional forms for directional spreading at the PDE level. In principle, these terms can represent “daily practices” (work commutes, etc.), as well as longer temporal or spatial scales (major thoroughfare traffic, etc.). We simply allow diffusion to perform the relevant spreading. Arriving infected individuals are assumed to form local hotspots within the susceptible population. This initial seeding roughly approximates long-range transportation.

The next step is to identify the parameters at the ODE level. To do this, we use a nonlinear optimization algorithm. We determine the optimal parameters by minimizing the Euclidean distance between the time series generated by the model (num) and the corresponding observed (obs) time series,

$$\mathcal{N} = \sum_i^{t_{\text{fit}}^{\text{end}}} \left(|\log(C_{\text{num}}(t_i)) - \log(C_{\text{obs}}(t_i))|^2 + |\log(D_{\text{num}}(t_i)) - \log(D_{\text{obs}}(t_i))|^2 \right) \quad (4)$$

where the index i identifies a point in the time series. The parameters are optimized to reproduce the reported total number of infected cases $C(t) = I(t) + R(t)$, and the total number of deceased $D(t) = R(t)$.

Since the transition rate γ is an “individual” parameter, and thus acts “on average”, we preserve the same value at the PDE level as at the ODE level. The transmission rate β is more complicated, since at the PDE level the quantities S , I , and R are densities rather than populations, which integrate over the spatial surface to the true population of each category.

3 Results

3.1 ODE Model

We begin by discussing the zero-dimensional model for Chicago. Data for the progression of the pandemic in Chicago were obtained from the Chicago Data Portal [1]. The relevant results are given in Figure 3. We obtained the optimal ODE model parameters for Chicago from 1,000 optimizations (implemented in MATLAB via the `fminsearch` function) that compared model predictions to jointly the number of cumulative infected and the deceased, as in Equation 4. For each optimization, the initial guess for each parameter was uniformly sampled within a pre-specified range. The parameter ranges were determined from epidemiological information.

The COVID-19 pandemic had three major impact waves in Chicago, as seen clearly in Figure 2. For simplicity, we analyze only the first major wave, which ends at approximately October 30, 2020. Additionally, we begin the analysis at March 17 instead of the first day of data collection (March 1) because of reporting errors early in the pandemic.

Upon performing the optimizations we find that the fitting yields the results summarized in Table 1. We show the median parameters, as well as the interquartile range, and the range of variation used to sample the parameters. Model predictions (with median parameter values, solid blue lines) are compared graphically to data (black dots) in Figure 3.

The optimal parameters were determined for a scenario in which restrictive measures (quarantine, lockdown) were not enforced. Therefore, we do not impose a time dependence on the transmission rate β . The transmission rate represents the rate of contact of susceptible individuals

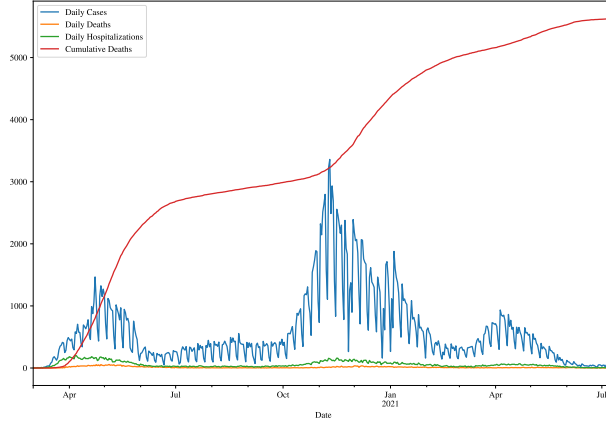


Figure 2: Observed data for Chicago showing the three impact waves.

with infectious individuals that leads to infection by the virus. It may be expressed as the product of daily average contacts and the probability of infection given a contact [2]. Thus, restrictive measures such as mobility restrictions, social-distancing, face-mask wearing, and gathering limitations are expected to decrease the transmission rate, an effect not accounted for in this model.

Table 1: ODE parameters for Chicago: optimal (best-fitting), median and interquartile range, and variation range used in the optimization algorithm. Initial parameter guesses were uniformly sampled within these ranges.

		Median (interquartile range)	Initial value
Population	N	2,695,598	
Initial population	(I_0, R_0)	(127, 2)	
Transmission rate, $S \rightarrow I$ [per day]	β^1	0.38206(0.38204-0.38209)	$c \in U[0, 1]$
Transition rate, $I \rightarrow R$ [per day]	γ	0.39656(0.39654-0.39659)	$c \in U[0.25, 0.75]$
Diffusivity, S [km ² /day]	\mathfrak{D}_S	10	
Diffusivity, I [km ² /day]	\mathfrak{D}_I	100	

The two top panels of Figure 3 show how well we capture the data for the Chicago outbreak. The model reproduces reasonable well the number of cases (top left panel) but not the number of deaths (top right panel). The model fails to capture the characteristic S-shape in the logarithmic plot of the top right panel (deaths).

3.2 PDE Model

A ODE Dynamics

Here we briefly discuss some dynamics of the ODE system. Note that for the simply SIR model as we have here, R is completely determined when S and I are known, so we need only consider

$$\begin{aligned} S_t &= -\tilde{\beta}SI, \\ I_t &= \tilde{\beta}SI - \gamma I, \end{aligned}$$

where $\tilde{\beta} = \beta/N$.

¹The transmission rate β must be divided by N when used in the ODE model.

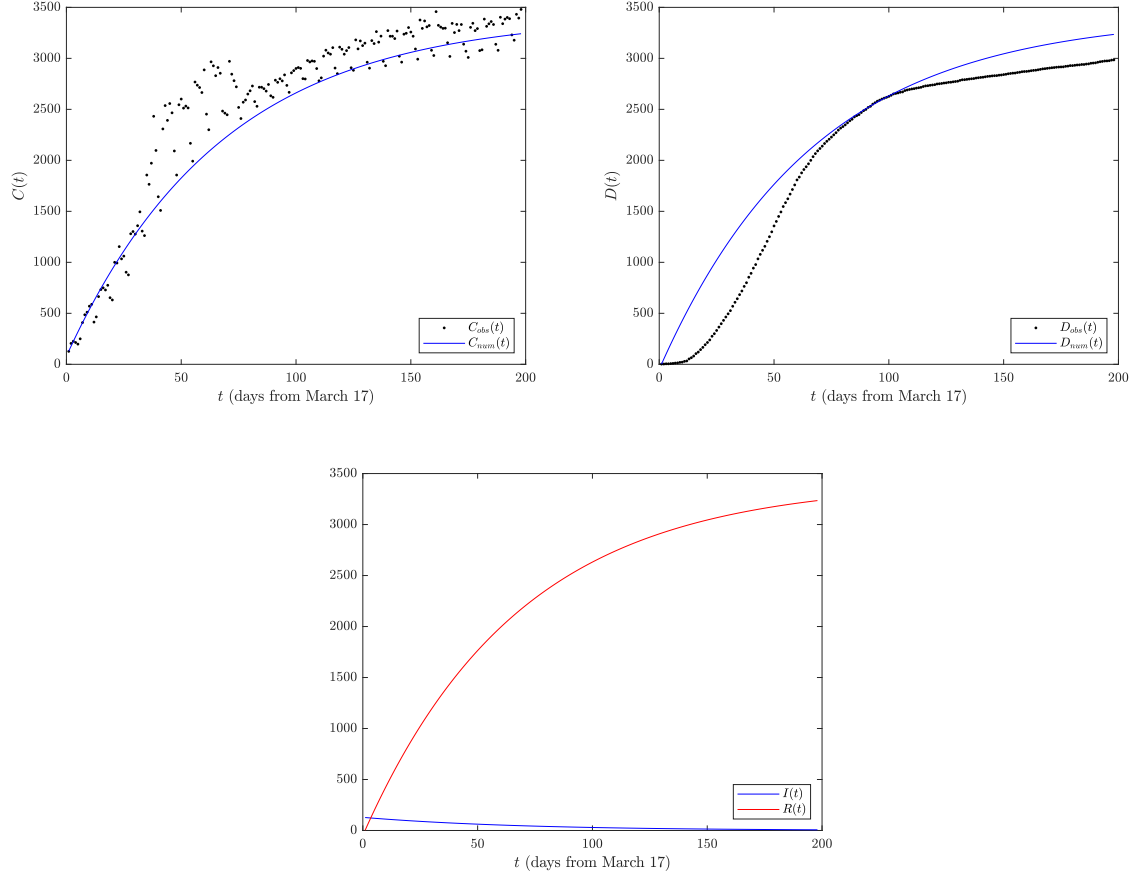


Figure 3: ODE model for Chicago with fitting to official data from March 17, 2020 ($t = t_{\text{init}} = 1$) to September 30, 2020 ($t = t_{\text{fit}}^{\text{end}} = 198$). Left panel: Confirmed cases $C(t) = I(t) + R(t)$; Right panel: Number of deaths $D(t)$. Bottom panel shows the other populations: infected $I(t)$ and recovered $R(t)$.

Immediately we see there are infinitely many non-isolated fixed points of the form $(S, 0)$. Linearizing, we get

$$\mathbf{A}_{(S,0)} = \begin{bmatrix} 0 & -\tilde{\beta}S \\ 0 & \beta S - \gamma \end{bmatrix}.$$

Thus, as seen in Figure 4, for $S < \frac{\gamma}{\beta}$ the nodes are stable, and for $S > \frac{\gamma}{\beta}$ they are unstable. We

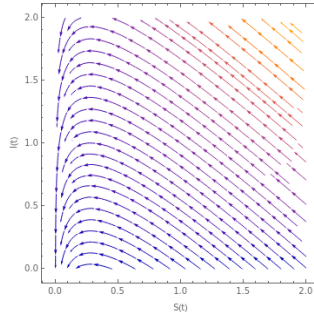
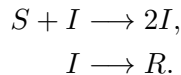


Figure 4: Stream plot for the local ODE dynamics. Note the transcritical bifurcation around $S_0 = 0.4$.

can then conclude that an epidemic will occur only if $S_0 = S(t=0) > \frac{\gamma}{\beta}$ [5]. Hence, a transcritical bifurcation occurs at $\mathcal{R}_0 = \tilde{\beta}S_0/\gamma$, which is known as the *basic reproduction number*, defined as the average number of secondary infections caused by a single infected individual [2].

Note that the ODE model can be viewed as a generalized chemical reaction



However, these two reactions are not reversible, so we are not able to write the energy-dissipation law for the ODE model [3, 6]. Indeed, taking $\frac{I}{S} = -1 + \frac{\gamma}{\beta S}$ and solving this differential equation gives $I = -S + \frac{\gamma}{\beta} \ln(S) + C$. Thus,

$$E(S, I) = S + I - \frac{\gamma}{\beta} \ln(S) \tag{5}$$

is a conserved quantity for the system.

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