Chapter 2
Literature Review

In this chapter, we first give a review of cellular automata in two dimensions. After that, we give a review of differential equation and discrete-time models for epidemics of SIR-type (susceptible-infected-recovered) and SIS-type (susceptible-infected-susceptible). For SIR-type models, we then give a review of the methods of conversion of differential equation and discrete-time models into cellular automata models.

2.1 Cellular Automata in Two Dimensions

Cellular automata (CA) in two dimensions are discrete dynamical systems formed by a finite number of $a \times b$ identical objects called cells which are arranged uniformly in a two dimensional lattice (In this work, we assume that the shape of a cell is a square) [1]. Each cell has a state s (from a finite state set Q), that changes at every time step by a transition function. The state of a cell at time t depends on the state of a set of cells, called its neighborhood, at the time $t-1$.

A CA in two dimensions is defined by the 4-tuple (L,Q,W,f), where L is the lattice:

$$L = \{ (i,j) \mid 1 \le i \le a,\ 1 \le j \le b \}; \tag{2-1}$$

Q is the finite set of states of a cell in the lattice. The state of a cell (i,j) at time t is defined by s_{ij}^t. W is a finite set called the neighborhood of each cell (i,j). In a square lattice, two types of neighborhood are regularly used, namely von Neumann neighborhood and Moore neighborhood. The von Neumann neighborhood of cell (i,j) with a radius r, is defined as

$$W_{i,j} = \{ (k,l) \in L \mid |k-i| + |l-j| \le r \} \tag{2-2}$$

and the Moore neighborhood of cell (i,j) with a radius r, is defined as

$$W_{i,j} = \{ (k,l) \in L \mid |k-i| \le r \wedge |l-j| \le r \}. \tag{2-3}$$

Radius $r = 1$ or $r = 2$ are the most frequently used (see FIGURE. 2-1). There is also one type of neighborhood that has no pattern, called an arbitrary neighborhood (see FIGURE. 2-2);

The function f is the transition function which gives the new state s_{ij}^t of cell (i, j) at time t given the state of the neighborhood $W_{i,j}$ at time $t-1$:

$$s_{ij}^t = f(s_{i+\alpha_1, j+\beta_1}^{t-1}, \ldots, s_{i+\alpha_n, j+\beta_n}^{t-1}) \subset Q. \tag{2-4}$$

In this work, we will use null boundary conditions, that is, $s_{ij}^t = 0$ if $(i, j) \notin L$.

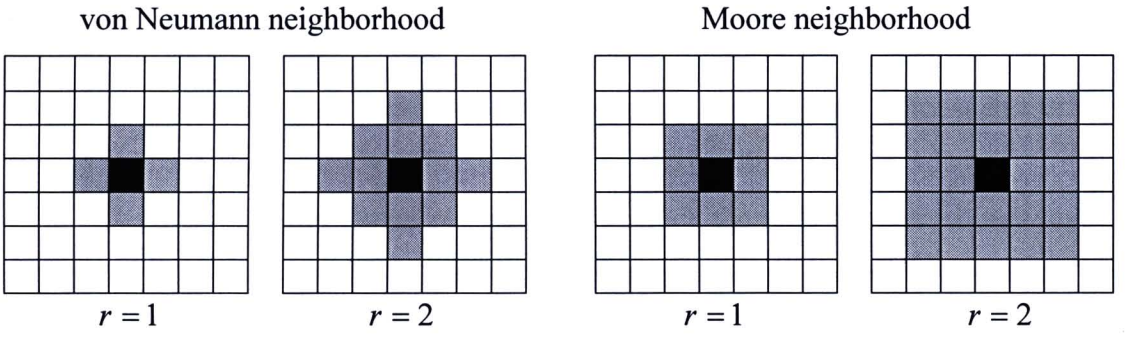

FIGURE 2-1 von Neumann neighborhood and Moore neighborhood of cell (i, j) with radius $r = 1$ and $r = 2$

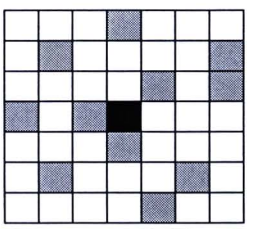

FIGURE 2-2 Arbitrary neighborhood of cell (i, j)

2.2 Epidemic Models

In this section, we first give an introduction to mathematical epidemic models. After that, we give a review of two examples of epidemic model, namely SIR model and SIS model.

2.2.1 Introduction

Epidemic means a large number of cases of the same infectious disease at the same time. Many scientists have developed mathematical models for predicting transmission of a disease transmission beginning with W. O. Kermack and A. G. McKendrick [8], who created an SIR model for a closed population.

Two types of epidemic model are of interest, namely stochastic model [10] and deterministic model. A stochastic model is a mathematical model which includes random variations in one or more parameters and inputs over time. The random variation is usually based on fluctuations observed in historical data. The predictions of the model therefore do not give a single point estimate but give a probability distribution of possible estimates. A deterministic model is a mathematical model in which the parameters and variables are not subject to random fluctuations, so that the system is at any time entirely defined by the initial conditions chosen.

When we consider closed populations in which the total number of individuals in a population does not change, deterministic or compartmental models are used. In a compartmental model, the total population can be divided into many classes. For example, in an SIR model [1, 2, 8] the total population is divided into three classes: Susceptible (S) (people who have not had the disease), Infected (I) (people who have the disease) and Recovered (R) (people who have recovered from the disease and cannot become infected again). The model of spreading of a disease in a closed population depends on time or space and time. In a model in which the change is assumed to occur only with time, a model can be created using ordinary differential equations [2, 8] (continuous-time) or difference equations [5] (discrete-time). We will consider examples of time-dependent models in the next section. In space and time, a model can be created using partial differential equations [12] (continuous-time with local transition function of disease transmission in space) or network model [9] (discrete-time with non-local transition function in space) or Cellular Automata [1] (discrete-time and the transition function can be local or non-local in space).

In the next section, we will give a review of two examples of epidemic model, namely SIR model and SIS model. For definitions of the terminology that is used in this work see Table 1.

2.2.2 SIR-type epidemics models

In 1927, W. O. Kermack and A.G. McKendrick created an SIR (susceptible – infected – recovered) model [8, 11]. The population that they considered was a closed population; they assumed the total population was fixed, for example, no births, no deaths by disease, no deaths by natural causes and no immigration or emigration. They divided the population into three classes: Susceptible (S), Infected (I) and Recovered (R). They assumed $S(t)$ was the number of individuals who did not have the disease at time t but who could become infected; $I(t)$ was the number of individuals who were infected with the disease and were able to spread the disease to susceptible people; $R(t)$ was the number of individuals who had been infected with the disease, but then recovered from the disease and could not infected again because they had become immune. Two positive parameters α and β described an infection rate between a susceptible individual and an infected individual, and a recovery rate of an infected individual, respectively. For a constant total population, we have:

$$S(t)+I(t)+R(t)=N \tag{2-5}$$

where N is population size.

The SIR model can be described by the following system of ODEs:

$$\frac{dS(t)}{dt}=-\alpha S(t)I(t) \tag{2-6}$$

$$\frac{dI(t)}{dt}=\alpha S(t)I(t)-\beta I(t) \tag{2-7}$$

$$\frac{dR(t)}{dt}=\beta I(t) \tag{2-8}$$

with the initial condition: $S(0)=S_0$, $I(0)=I_0$ and $R(0)=R_0=0$, so that

$$S(0)+I(0)+R(0)=S_0+I_0=N. \tag{2-9}$$

It can be seen that the equilibrium points of this system are $(S^*,0,N-S^*)$, where $0\le S^*\le N$.

The stability of the equilibrium points can be checked using standard linearization methods. The eigenvalues of the linearized system at $(S^*,0,N-S^*)$ are 0, 0 and $\alpha S^*-\beta$. Therefore, if $\alpha S^*-\beta>0$, the equilibrium point $(S^*,0,N-S^*)$ is

unstable. In all other cases, the linearization tests fail because of the 0 eigenvalues. However, the stability can be determined by direct analysis of the equations. It can be seen that if $I > 0$, then S must decrease and R must increase. Therefore the only stable equilibrium point will be $(0,0,N)$. However, if the system is not at this stable equilibrium point, then we have the following results. Consider Eq. (2-7). If $S_0 < \frac{\beta}{\alpha}$ then $\left(\frac{dI}{dt}\right)_{t=0} < 0$ and infection decreases. If $S_0 > \frac{\beta}{\alpha}$ then $\left(\frac{dI}{dt}\right)_{t=0} > 0$ and infection increases. If $S_0 = \frac{\beta}{\alpha}$ then $\left(\frac{dI}{dt}\right)_{t=0} = 0$ and infection does not change and can be maximum or zero. In this case, if $I=0$, then S_0 and $R_0 = N - S_0$ will not change, but $I > 0$ will cause S to decrease and R to increase.

The previous model is based on continuous-time. When we consider discrete time, the model can be described by a difference equation [5] that is the following:

$$S(t+1) = S(t)(1 - \alpha \Delta t I(t)) \tag{2-10}$$

$$I(t+1) = I(t)(1 - \beta \Delta t + \alpha \Delta t S(t)) \tag{2-11}$$

$$R(t+1) = R(t) + \beta \Delta t I(t) \tag{2-12}$$

with the same meaning for parameters and variables as in the ODEs. The solutions for S, I and R must be non-negative. The behavior of infection in a population is as follows:

From Eq. (2-11), the number of infected individuals will increase at time t if

$$I(t+1) > I(t), \text{ i.e., if } \frac{\alpha}{\beta} S(t) > 1 \tag{2-13}$$

the number of infected individuals will decrease at time t if

$$I(t+1) < I(t), \text{ i.e., if } \frac{\alpha}{\beta N} S(t) < 1 \tag{2-14}$$

and the number of infected individuals will not change at time t if

$$I(t+1) = I(t), \text{ i.e., if } \frac{\alpha}{\beta} S(t) = 1 \tag{2-15}$$

These conditions for the discrete model are the same as for the continuous model.

2.2.3 SIS-type epidemics models

The SIS model [5] describes an epidemic model in which the population is divided into two classes: susceptible individuals (S) and infected individuals (I). A susceptible individual can be infected by an infected individual with the infection rate α and an infected individual can recover with the recovery rate β. However, in this model individuals do not become immune to the disease and therefore they return to the S class. Assume the population is a constant:

$$S(t)+I(t)=N \tag{2-16}$$

where N is total population size.

The model that considers only the time can be described by the following system of ODE for continuous time:

$$\frac{dS(t)}{dt}=-\alpha S(t)I(t)+\beta I(t) \tag{2-17}$$

$$\frac{dI(t)}{dt}=\alpha S(t)I(t)-\beta I(t) \tag{2-18}$$

with the initial condition: $S(0)=S_0$, $I(0)=I_0$

From Eq. (2-16), $I=N-S$, and substituting into Eq. (2-17) then gives the equation

$$\frac{dS}{dt}=\alpha(N-S)\left(\frac{\beta}{\alpha}-S\right). \tag{2-19}$$

When $\frac{\beta}{\alpha}>N$, Eq. (2-19) has a unique equilibrium $S=N$ on the interval $(0,N]$. This equilibrium is asymptotically stable, that is, the solution of $S(t)$ starts from S_0 and increases to N as t tends to infinity and the solution of $I(t)$ decreases to zero. That means the infection dies out.

When $\frac{\beta}{\alpha}<N$, Eq. (2-19) has two equilibrium points $S=N$ and $S=\frac{\beta}{\alpha}$, where $S=N$ is unstable, and $S=\frac{\beta}{\alpha}$ is asymptotically stable. The solution of $S(t)$ starts from S_0 approaches to $\frac{\beta}{\alpha}$ as t tends to infinity and the solution of $I(t)$ tends to $N-\frac{\beta}{\alpha}>0$.

From Eq. (2-17) and (2-18), when we consider discrete time [6], the model is the following:

$$S(t+1) = S(t) - \alpha \frac{\Delta t}{N} S(t)I(t) + \beta \Delta t I(t) \tag{2-20}$$

$$I(t+1) = (1 - \beta \Delta t)I(t) + \alpha \frac{\Delta t}{N} S(t)I(t) \tag{2-21}$$

with the same meaning of parameters and variable as in SIS continuous model. Assume the population of each cell is constant and normalized to 1, i.e., $S(t) + I(t) = 1$. So, $S(t)$ is the proportion of susceptible individual and $I(t)$ is the proportion of infected individuals.

From Eq. (2-21), we obtain:

$$I(t+1) = f(I(t)) = \left(1 - \beta \Delta t + \alpha \Delta t - \alpha \frac{\Delta t}{N} I(t)\right) I(t) \tag{2-22}$$

Since the population of infectious individuals cannot be negative and cannot be greater than the total population, from Eq. (2-22), we must have that:

$$0 \le I(t+1) = f(I(t)) = \left(1 - \beta \Delta t + \alpha \Delta t - \alpha \frac{\Delta t}{N} I(t)\right) I(t) \le 1 \tag{2-23}$$

Since $0 \le I(t+1) \le 1$, the function f in Eq. (2-23) must also satisfy $0 \le f(I) \le 1$ for $0 \le I \le 1$. Using calculus to find the maximum value of $f(I)$ gives the condition $\alpha \le \left(1 + \sqrt{\beta}\right)^2$.

The analysis of the infected Eq. (2-21) can be simplified by making a change of variable to transform it into the logistic difference equation [6]:

$$x(k+1) = F(x(k)) = ax(k)(1 - x(k)). \tag{2-24}$$

The changes of variable are:

$$x(t) = \frac{\alpha I(t)}{1 - \beta + \alpha}, \tag{2-25}$$

where $a = 1 - \beta + \alpha$

Since $0 \le \beta \le 1$ and $0 \le \alpha \le \left(1 + \sqrt{\beta}\right)^2$, the possible range of $a = 1 - \beta + \alpha$ is $0 \le a \le 4$. The equilibrium point $x^* = F(x^*) = ax^*(1 - x^*)$ are: $x^* = 0$ and $x^* = 1 - \frac{1}{a}$.

The logistic equation shows the bifurcation path to chaos [6, 7].

2.3 A Partial Differential Equation Model for an SIR-type Epidemic

The SIR model in section 2.2.2 considers only the time and does not include space. When we consider spread of a disease that depends on space and time, we can use a partial differential equation (PDE) to create a model [12, 11]. Schneckenreither et al [12] assume that $S(t,x,y)$ is susceptible population density at position (x,y) at time t, $I(t,x,y)$ is infected population density at position (x,y) at time t, and $R(t,x,y)$ is recovered population density at position (x,y) at time t. They also assume that there is no movement of people in space and time and that the total population density is initially uniform in space. Then the total population density at (x,y) is independent of time. It is assumed that a susceptible individual can get a disease from an infected individual at a different position, where the probability of infection is a function of the distance between the susceptible and infected individuals. The spread of the infection is therefore through a non-local interaction in space. Schneckenreither et al [12] then approximate the non-local interaction by a second-order Taylor series expansion and obtain a formula for the rate of infection of susceptible individuals of the form: $-\alpha S(t,x,y)I(t,x,y)-\gamma S(t,x,y)\nabla^2 I(t,x,y)$ where α and γ are constant infection rates. After recovery from the disease, a recovered individual cannot be infected again because they are assumed to be immune. The SIR model can be written as a system of PDE's as follows [12]:

$$\begin{aligned}
\frac{\partial S(t,x,y)}{\partial t} &= -\alpha S(t,x,y)I(t,x,y)-\gamma S(t,x,y)\nabla^2 I(t,x,y) \\
\frac{\partial I(t,x,y)}{\partial t} &= \alpha S(t,x,y)I(t,x,y)+\gamma S(t,x,y)\nabla^2 I(t,x,y)-\beta I(t,x,y) \qquad (2\text{-}26) \\
\frac{\partial R(t,x,y)}{\partial t} &= \beta I(t,x,y)
\end{aligned}$$

where α is an infection rate between a susceptible individual and an infected individual in the same position

γ is an integrated infection rate between a susceptible individual and infected individuals in neighboring positions

β is a recovery rate

To approximate the solution, we can discretize the PDE model (2-26) using a finite difference approximation for the time and space derivatives (see Appendix A).

Schneckenreither use a standard finite-difference approximation for the Laplacian $\nabla^2 I(x,y)$ of the form:

$$\nabla^2 I(x,y) \approx \frac{1}{\varepsilon^2}\left(I(x+\varepsilon,y)+I(x-\varepsilon,y)+I(x,y+\varepsilon)+I(x,y-\varepsilon)-4I(x,y)\right) \quad (2\text{-}27)$$

Schneckenreither et al [12] state that the use of a finite difference approximation for a PDE gives a cellular automata model. We will examine the connection between PDE and CA models in chapter 3.

2.4 A cellular automata model for an SIR-type epidemics

White et al [1] have developed a cellular model for an SIR-type epidemic. They suppose that the ground where the epidemic is spreading is modeled as the lattice of a CA with a cell being a square of side length 1. Different cells can have different populations and the lattice is considered to be large enough to ensure that the epidemic spreading affects only the central region. They used null boundary conditions. The assumptions of White et al for their CA model are as follows:

- The total population of each cell and the total population in the lattice are constant, i.e., no births or deaths, and no net immigration or emigration between cells. People can move from their home cell to another cell during a time step $t-1$ to t, but will return to their home cell before time t.
- It is supposed that when an infectious individual arrives at a cell, the number of susceptible individuals contacted by him/her is the same independently of the total population of the cell.
- The set of states for cell (i,j) is $Q = K \times K \times K$ where

$$K \in \{0.00,0.01,0.02,...,0.99,1.00\}, \quad (2\text{-}28)$$

and the state of cell (i,j) at time t is given by

$$s_{i,j}^t = \left(S_{i,j}^t, I_{i,j}^t, R_{i,j}^t\right), \quad (2\text{-}29)$$

where $s_{i,j}^t \in Q$.

In Eq. (2-29), the populations $\left(S_{i,j}^t, I_{i,j}^t, R_{i,j}^t\right)$ will represent the fractions of the population of cell (i,j) that are susceptible, infected and recovered at time t. Then,

$$S_{i,j}^t + I_{i,j}^t + R_{i,j}^t = 1. \quad (2\text{-}30)$$

where,

$S_{i,j}^t \in [0,1]$ is the proportion of susceptible individuals of the cell (i,j) at time t, $I_{i,j}^t \in [0,1]$ is the proportion of infected individuals of the cell (i,j) at time t and $R_{i,j}^t \in [0,1]$ is the proportion of recovered individuals of the cell (i,j) at time t.

The main goal of the model is to compute the factors S_{ij}^t, I_{ij}^t and R_{ij}^t. The local transition function used is the following:

$$\begin{aligned} S_{i,j}^{t+1} &= S_{i,j}^t - \alpha \cdot S_{i,j}^t \cdot I_{i,j}^t - S_{i,j}^t \cdot \sum_{(k,l)\in W^*} \frac{N_{i+k,j+l}}{N_{i,j}} \cdot \mu_{k,l}^{(i,j)} \cdot I_{i+k,j+l}^t \\ I_{i,j}^{t+1} &= (1-\beta) I_{i,j}^t + \alpha \cdot S_{i,j}^t \cdot I_{i,j}^t + S_{i,j}^t \cdot \sum_{(k,l)\in W^*} \frac{N_{i+k,j+l}}{N_{i,j}} \cdot \mu_{k,l}^{(i,j)} \cdot I_{i+k,j+l}^t \\ R_{i,j}^{t+1} &= R_{i,j}^t - \beta \cdot I_{i,j}^t \end{aligned} \tag{2-31}$$

where W^* is an index set of the neighborhood, for example, for a von Neumann neighborhood

$$W^* = \{(0,1),(0,-1),(1,0),(-1,0)\}. \tag{2-32}$$

The real parameter $\mu_{k,l}^{(i,j)}$ is defined by the product of three factors:

$$\mu_{k,l}^{(i,j)} = c_{k,l}^{(i,j)} \cdot m_{k,l}^{(i,j)} \cdot \alpha, \tag{2-33}$$

where $c_{k,l}^{(i,j)}$ is a connection factor and $m_{k,l}^{(i,j)}$ is a movement factor between the main cell (i,j) and its neighborhood cell $(i+k, j+l)$.

White et al assume that there are three ways of transport between cells: by airplane, by train and by car or bus. This connection is given by the coefficients $c_{k,l}^{(i,j)}$ such that:

$$c_{k,l}^{(i,j)} = \begin{cases} 1, & \text{if there exist the three ways of transport between the cells,} \\ 0.6, & \text{if there are two ways of transport between the cells,} \\ 0.3, & \text{if there is only one way of transport between the cells,} \\ 0, & \text{if there is no way of transport between the cells} \end{cases} \tag{2-34}$$

The movement factor $m_{k,l}^{(i,j)} \in [0,1]$ stands for the probability of an infected individual belonging to the neighbor cell $(i+k, j+l)$ to be moved to the main cell (i,j).

2.5 Summary

In this chapter, we have reviewed some basic definitions and notation that are used in cellular automata in two dimensions. We have described the SIR-type and SIS-type of epidemics models based on continuous-time and discrete-time that depend on time only. In space and time, we have described the SIR model and given examples of a PDE model and a CA model.

In this thesis, we aim to develop an SIS-type epidemics model based on cellular automata. We also aim to show the conversion for SIR-type epidemics from a PDE model to a CA model. After that, we aim to develop a computer program to simulate cellular automata epidemics models. In chapter 3 we describe our methodology for creating SIS-type epidemics based on cellular automata and also the methodology for converting PDE models to CA models.