Modeling epidemic spread with spike-based models

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ABSTRACT

The Susceptible-Infected-Recovered/Removed model is a standard model for epidemiological spread of disease through vulnerable populations. In this paper we show how SIR network dynamics can be implemented using spiking neurons.

CCS CONCEPTS

• **Hardware** → *Neural systems*; • **Theory of computation** → *Theory and algorithms for application domains*.

KEYWORDS

epidemiological modeling, spiking neural networks, neuromorphic algorithms

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1 INTRODUCTION

Contagious pandemic diseases like COVID-19, Zika, Ebola and H1N1 Swine Flu severely disrupt lives of people globally [24]. Epidemiological models are needed for modeling and simulation of the impact of contagious diseases. There are many such models but there are a number of common pitfalls such as: reliability, robustness or scalability [7, 9]. The reasons for this are two-fold:

- These models were not designed to work with real-world data in real-time, and are not very adaptive or robust [2].
- As new information pertaining to the spread of a disease

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becomes available, these epidemic simulations must be re-run from scratch.

- These models are either agent-based or equation-based and use differential equations at their core.

Real-world populations are inherently heterogeneous with varying ranges of susceptibility for different demographics. If the underlying system of differential equations grows in dimension and problem density, then modeling becomes increasingly infeasible on even the largest and fastest supercomputers like Summit [29].

We believe that epidemiological models that leverage neuromorphic computing could prove to be more effective than the traditional epidemiological models that leverage conventional computers. Neuromorphic computing is a computing paradigm that performs computations by emulating the human brain. Akin to the human brain, neuromorphic systems can perform computations fast and consume orders of magnitude less energy than CPUs or GPUs. Neuromorphic systems have been shown to perform machine learning tasks in a fast and energy-efficient manner, and excel in applications involving real-time analysis of real-world data. Neuromorphic systems like Intel Loihi [5], IBM TrueNorth [6] and SpiNNaker [21] are highly scalable by design and have been scaled to millions of neurons at present. Neuromorphic systems are composed of neurons which are connected to each other through synapses. The intrinsic parameters of neurons (like spiking threshold, refractory period) and synapses (like synaptic weights, synaptic delay) are governed by differential equations and as we show in this paper, translate fluently to traditional epidemiological SIR model.

We focus on the Susceptible-Infected-Removed (SIR) model which has been developed and utilized since the early 20th Century [20] and commonly employed in epidemiological studies [17] and also utilized in network science studies [25, 26]. We present proof of concept studies that demonstrate a spiking neuron approach for simulating SIR dynamics. We use synthetically generated graphs of social interactions to show that SIR dynamics modeled through spike-based simulation capture epidemic growth that can be controlled through the probability of successful disease transmission.

2 RELATED WORK

SIR models use a variety of tools to study the spread of a wide range of diseases. They are highly adaptable, from epidemics where vaccines are available (such as measles [3] and influenza [8]) and where they are not (such as Zika [22] and H1N1 [4]). Various mathematical frameworks have been utilized, such as numerical analysis [23] and graph theory [30]. The latter is a highly interpretable model where nodes can represent a host of objects such as individuals, cities, or

airports; the edges can represent personal interaction, road linkages, or flight routes. Neuromorphic systems provide low-power, robust and resilient deep learning frameworks, yet their highly graph-theoretic architectures have provided useful in effective implementations of graph algorithms [15] [27] [19] which carry low overhead. Thus, SIR models which have a graph theoretic nature coupled with neuromorphic computing provide an ideal framework for analysis of the spread of new epidemics with a variety of properties (vaccinations, varying R_0 values, etc).

3 DEFINITIONS

To construct our spike-based SIR model we make the following analogy: the spread of spikes throughout a system of neurons mimics the spread of a disease through a population. The basic elements of our model are: 1) an undirected network to represent social contacts in our population and 2) a system of spiking neurons. The spread of spikes throughout the network will depend on the neuronal and synaptic parameters, as well as the underlying network connectivity.

Our representative social network was generated using a code library introduced for the study of community structure in graphs [10, 11]. The population we studied contains 1000 vertices representing 1000 individuals connected by a network of over 7500 social interactions that can spread infection - the graph consists of several densely connected communities that are sparsely interconnected (see Fig. 1).

Figure 1: The social interactions of a population with 1000 individuals and $\langle d \rangle = 15.064$.

We construct the spiking neural system (SNS) by mapping each individual (graph vertex) to the standard leaky-integrate and fire neuron model [12]. The neuronal spiking threshold (v_{th}) is used to represent the susceptibility of each individual to becoming infected. Once an individual has recovered from an infection, the neuronal refractory period represents immunity of an individual—a high refractory period resembles high immunity to the disease, and a small

refractory period implies that the individual would be susceptible to the disease in the future. Each social interaction (graph edge) is mapped to a pair of symmetric weighted synapses. The rate at which infections spread within a population is dependent on the synaptic weights.

3.1 SIR model parameters

We focus on the Kermack-McKendrick model [20] which is defined by a system of differential equations which determine the temporal evolution of 3 population classes: the susceptible (S), the infected (I) and the recovered or removed (R);

$$\begin{aligned}\frac{dS}{dt} &= -\beta IS, \\ \frac{dI}{dt} &= \beta IS - \gamma I, \\ \frac{dR}{dt} &= \gamma I.\end{aligned}\tag{1}$$

The constants β, γ^{-1} define the contact rate (the average number of interactions that are able to spread infection per unit time) and the latent period (the time that an individual is infectious).

In this work we present results where spike dynamics are used to simulate the time dynamics of these 3 classes with the following constraints:

- An individual can be part of only one class during each decoded time step.
- The sizes of the 3 classes are constrained $S + I + R = N$. We do not include births or deaths in these preliminary studies.
- The underlying structure of social interactions do not change as the epidemic spreads.

The spread of an epidemic is largely driven by the reproduction number R_0 . This defines the number of secondary infections that are caused by an infected individual and can be defined as $R_0 = \beta/\gamma$. For comparison influenza can have $R_0 \approx 1.7 - 2.0$ [8]. Since R_0 is dependent on the social contacts of an individual it can vary significantly based on the social behavior in a population.

3.2 Spiking-SIR model definitions

On a neuromorphic platform, information is transmitted via discrete electrical pulses and an algorithm design is defined by a spiking neural network. Previous studies have established that graph algorithms can be executed using neuromorphic platforms [1, 14, 15, 28]. In this section we define our mapping between graph notation, spiking neuron notation and SIR notation.

The underlying network structure is defined by a graph, and it is the individual neuron and synaptic parameters which are tuned in order to simulate different epidemic outcomes. The spiking neurons are characterized by leaky-integrate and fire neurons implemented in the Brian2 Python Library [13]. Each neuron is initialized at a voltage v_0 , fires a spike if its potential exceeds a threshold voltage v_{th} and if a neuron fires then its voltage is reset to v_r and the neuron enters a refractory period of length (t_R). The synaptic connections are parameterized by a weight s_w , and a delay δ . Since we assume that the network of social interactions remain fixed during the growth of an epidemic we do not require the use of synaptic plasticity. We initialize each neuron at $v_0 = 0$. with $v_{th} = 0.8$. The

synaptic connection are initialized with $s_w = 1.0$, $\delta = 10$. This means that any spike traveling between neurons is sufficient to propagate the infection.

For a basic SIR model there are 3 population classes [17]: susceptible individuals (S) are those who may become infected, infected individuals (I) are those spreading disease, and recovered individuals (R) are those who have been infected but are no longer infectious. We define our mapping between spiking neuron dynamics and SIR model classes:

- Neurons that have never fired a spike are in class (S). Their voltage is the initial voltage (v_0) and they are not in a refractory period.
- Neurons that are firing are in class (I). Their voltage has exceeded the spike threshold value (v_{th}).
- Neurons that are in the refractory period are in class (R). They were previously in class I but have recovered.

The synaptic weights are the primary driver of the epidemic dynamics. If a synaptic weight is weak ($s_w < v_{th}$) then any spike that is transmitted along this synapse cannot cause a secondary neuron to fire (cause a secondary infection). On the other hand, if a synaptic weight is strong ($s_w > v_{th}$) then any spike that arrives along this synapse will cause the secondary neuron to fire (become infected).

Our approach to SIR modeling relies on a static graph of social interactions (Figure 1), but the connectivity is not uniform. We expect the structure of the graph to affect the growth of any epidemic. With the underlying network connections fixed, we generate multiple epidemic curves: for each simulation we choose a specific neuron to act as the site of initial infection. When we report the results of multiple epidemic simulations we use the mean relative population sizes $\langle s(t) \rangle = \langle S(t) \rangle / N$, $\langle i(t) \rangle = \langle I(t) \rangle / N$, and $\langle r(t) \rangle = \langle R(t) \rangle / N$. Additionally when we compute the time to peak infection (T_{peak}), size of peak infection ($|E_{max}|$) we use the mean relative populations. For comparison we also include the “worst case” or largest infected population ($\max |E|$) for an epidemic started at a single vertex.

4 SPIKE-BASED KERMACK-MCKENDRICK MODEL

We use a system of spiking neurons with ($v_{th} = 0.8$, $v_0 = 0.0$, $v_R = -1.0$) to simulate the Kermack-McKendrick model. Starting from Eq. 1 we derive a spike-based implementation of the SIR model using a phenomenological mapping of R_0 into synaptic parameters. In this section we fix the contagious period to a generic unit time step $\gamma = 1$ and the SIR model is reduced to one parameter: (β). We use a probabilistic mapping $\beta = p(s_w > v_{th})$ such that β is now the probability that a social interaction, represented by a weighted synaptic connection (s_{ji}), is successful at transmitting infection from individual (n_i) to individual (n_j) during a unit time step. We implement the probabilistic map by first initializing all synapses with weight $s_w = 1.0$, then with probability $1 - \beta$ we choose a subset of weights to be reduced to $s_w = 0.1$, too weak to transmit infection throughout the network. To translate this probabilistic mapping into an estimate for R_0 , we use the average degree of the graph: $\langle d \rangle = 15.064$. The estimated reproduction number for our studies is given by: $R_0 = p\langle d \rangle / \gamma$.

Table 1: Time to peak infection, and size of epidemic for non-recurrent infections ($t_R = |E(G)|$, $\beta = [0.25, 0.2, 0.15, 0.1]$).

R_0	T_{MAX}	$ E $	$\max E $
$R_0 = 3.77$	9.0	0.13 ± 0.08	0.282
$R_0 = 3.01$	9.0	0.12 ± 0.07	0.226
$R_0 = 2.26$	10.0	0.07 ± 0.05	0.178
$R_0 = 1.51$	14.0	0.02 ± 0.02	0.087

Our first study considers a population that will gain immunity to the disease after infection - we model this using $t_R = |E(G)|$ a refractory period that is equal to the size of the underlying graph. Using this mapping imposes the assumption that the following happen on short time scales: once an individual is exposed to an infection (a spike arrives at a neuron) the individual immediately becomes infectious (the neuron fires a spike); once an individual spreads an infection (the spike is fired) the individual immediately becomes recovered (the neuron enter the refractory period).

We use population decoding to assign neurons to an appropriate SIR class. Each simulation is run for a minimum number of clock steps defined by the graph order: $T = |V|$. In this case the spike dynamics are self-terminating and will end well before this time. Once the final spike raster is generated, we partition the total simulation time into n discrete non-overlapping windows each of width Δt and we count the number of neurons which belong to each class.

We simulated 4 sets of epidemic curves ($[S(t), I(t), R(t)]$) using $\beta = 0.25, 0.2, 0.15, 0.1$. We plot the relative populations $s(t) = S(t)/N$, $i(t) = I(t)/N$, and $r(t) = R(t)/N$ in Figs. 2, 3, 4, respectively. The relevant metrics for each epidemic curve (time to peak infection, size of outbreak at the peak, etc.) are reported in Table 1.

Figure 2: Mean relative populations of susceptible individuals $s(t)$ as β is varied from $\beta = 0.25$ (black, circles), $\beta = 0.20$ (grey, squares), $\beta = 0.15$ (blue, diamonds), $\beta = 0.10$ (red, triangles). The mean is taken over the 150 different initial infection locations, the width of the shaded region is determined by the standard deviation of the mean.

Figure 3: Mean relative populations of infected individuals $i(t)$ as β is varied from $\beta = 0.25$ (black, circles), $\beta = 0.20$ (grey, squares), $\beta = 0.15$ (blue, diamonds), $\beta = 0.10$ (red, triangles). The mean is taken over the 150 different initial infection locations, the width of the shaded region is determined by the standard deviation of the mean.

Figure 4: Mean relative populations of recovered individuals $r(t)$ as β is varied from $\beta = 0.25$ (black, circles), $\beta = 0.20$ (grey, squares), $\beta = 0.15$ (blue, diamonds), $\beta = 0.10$ (red, triangles). The mean is taken over the 150 different initial infection locations, the width of the shaded region is determined by the standard deviation of the mean.

5 CONCLUSIONS

There is a clear parallel between the growth of a giant connected component on a graph and the growth of the infected population in a network. We would expect that there is a critical value for the parameter β above which the epidemic fails to reach all individuals in the population. For the case where immunity is assumed, the large connected component is restricted to grow along non-backtracking paths, which have been used to establish lower bounds on percolation thresholds for sparse, tree-like graphs [16, 18]. These bounds predict that the critical value of $\beta = 0.039$, corresponding to $R_0 = 0.59$. While this agrees with the SIR model dynamics, where $R_0 > 1$ must be satisfied for an epidemic to spread, we note that this bound is not tight for the graph used in this study as it contains many cycles and is far from tree-like.

The initial studies reported in this paper have introduced the use of spiking neurons in SIR model simulation. The goal of this

work was to show the parallels between SIR models and spiking neuron dynamics with static synapses. To use spiking neurons as an alternative approach to solving SIR equations of motion we have shown that basic parameters such as β , γ can be mapped to neuron and synaptic parameters. While the calculated R_0 values are high, this is likely due to the use of unit time steps to define γ . Future work will develop more detailed methods of transmission, and explore the effects of underlying graph connectivity on the epidemic curves. For these initial studies we have considered populations with uniform disease immunity characteristics (v_{th} constant), however these studies are easily extensible to the case of populations with diverse characteristics.

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