A Lattice-based Approach to the PSQ Smoking Model

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Abstract

We study the dynamics of smoking behavior of agents with a stochastic lattice-based model, assuming that each agent occupies a node and is influenced by its neighbors. This mechanism is adapted from the PSQ smoking model, which is based on a system of ordinary differential equations. The difference in this model is that, more realistically, potential smokers are only influenced by nearby current smokers, instead of all smokers. In addition, the stochasticity of this model also accounts better for the randomness in real world smoking behavior. It is shown here that the quantitative estimates of this new lattice model are significantly different from the previous numerical results obtained in other works using the ODE model. This suggests that taking locality into account affects the model behavior. The critical exponents of this new lattice smoking model under von Neumann neighborhood condition are calculated and verified to be the same as the classic SIRS epidemic model, which classifies this model as belonging to the directed percolation class. We also consider the model in continuum setting, and solve the system numerically using a particular convolution kernel. To the author's knowledge this is the first time where this widely used and discussed PSQ smoking model is incorporated into the lattice-based setting, and our results show that this changes the quantitative behavior of the PSQ model significantly.

1 Introduction

Smoking is an important issue that affects global health and welfare. According to the World Health Organization [21], tobacco causes more than seven million deaths each year, over 80% of which occur in low- or middle-income countries. It also has a negative environmental impact on deforestation, climate change, and forest fires, to mention a few examples. Over the past years, smoking has drawn a considerable amount of focus and research [1, 3, 10, 19]. One popular smoking model which has been studied significantly is referred to as the PSQ smoking model [1, 19]. This model classifies the whole population to be either potential smokers, current smokers, temporary quitters or permanent quitters. All interactions in the model are assumed to be spontaneous except for the recruitment of potential smokers into current smokers, which is assumed to be proportional to the average number of contacts between potential smokers and current smokers [19].

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The model was first explicitly formulated as a smoking model in [19], and further discussed in [1] and [10], with certain modifications. Models with similar mechanism have also been studied, sometimes in a different context from smoking. For example, Castillo-Garsow et al. (1997, p. 38) [3] discussed a drug abuse and relapse model that is equivalent to the PSQ smoking model after renaming parameters. Thus, the PSQ smoking model is versatile and can be applied to numerous scenarios apart from smoking.

It is worth noting that so far all papers discussing the PSQ smoking model, to our knowledge, have focused on the system of ODEs. On the other hand, similar models in other applications related to public health are often studied through a two-dimensional lattice. For example, Souza and Tomé (2010) [4] studied the SIRS epidemic model on lattice. Other authors have also consider additional mechanisms based on the lattice approach. For example, Rhodes and Anderson (1997) [18] studied a variant of the SIRS model, where not all points on the square lattice are inhabited, and agents have the choice to move onto an uninhabited node near them. Fuks and Lawniczak (2001) [6] studied an SIR type epidemic model on a hexagonal lattice instead of square, where individuals also have the choice to move to different physical locations.

One of the major advantages of the lattice model over its ODE counterpart is that the lattice models take locality into account. In ODE models, the influence a person has on others is independent of their physical distance, whereas in lattice models, the dependence between the influence between individuals and physical distance can be quantified and controlled, by modifying the neighborhood size of interaction, for example. The stochastic and randomized nature of lattice-based models also allow us to better study the inherent uncertainty and randomness in real world smoking behavior, as opposed to the ODE approach which is deterministic. In these regards, lattice models are more realistic than their ODE counterparts. Furthermore, additional mechanisms and interactions can be more conveniently implemented on lattice models than ODE models, especially when these features are not spatially homogeneous.

The main goal of this paper is to introduce the PSQ smoking model into the lattice setting, and examine its difference from the ODE model brought by the consideration of physical distance. We will calculate its critical exponents to classify this model. In the last section, we will consider a continuum model using a particular kernel and examine the results.

2 The ODE System

In this section we provide a brief introduction to the PSQ smoking model as formulated in [10] and [19]. The population is classified to be either potential smokers (P), current smokers (S), past smokers who temporarily quit (Q_t) and past smokers who permanently quit in their lifetime (Q_p). We denote the whole population by N (that is $N = P + S + Q_t + Q_p$). We assume that the total population N remains the same throughout time, and the birth and death rate are universally the same, independent of the smoking status.[1] As time progresses, the system evolves

[1]This simplified assumption of uniform death rate does affect the accuracy of this model. Research [9] shows that life expectancy of current smokers is more than 10 years shorter than people who never smoke.

according to the following system of differential equations:

$$\begin{aligned}
\frac{dP}{dt} &= \mu N - \mu P - \frac{\beta PS}{N}, \\
\frac{dS}{dt} &= -(\mu+\gamma)S + \frac{\beta PS}{N} + \alpha Q_t, \\
\frac{dQ_t}{dt} &= -(\mu+\alpha)Q_t + \gamma(1-\sigma)S, \\
\frac{dQ_p}{dt} &= -\mu Q_p + \gamma\sigma S.
\end{aligned} \tag{1}$$

Here β denotes the rate of potential smokers turning into current smokers. This interaction is also assumed to be proportional to the frequency of contact between the potential smokers and the current smokers, which in this ODE model is measured by PS/N. Note that this term depends only on the total number of potential smokers and current smokers. In other words, it is independent of the physical distance between individuals. γ denotes the rate by which current smokers quit smoking. It is assumed that each quitter has a fixed probability σ to become a permanent quitter, who would never smoke again in their lifetime. The remaining quitters are assumed to be temporary quitters, with α denoting the rate by which these temporary quitters revert back to smoking. This model assumes that the natural birth and death rate μ is constant, independent of smoking status. The newborn population replacing the dead population are assumed as potential smokers. This model also assumes that the smoking behavior is independent of age. In particular, newborn potential smokers are assumed to become smokers at the same rate as other potential smokers.

This model has a obvious absorbing state when $P = N, S = Q_t = Q_p = 0$, which is called the smoking free limit (*SFE*) in [19]. For β small enough, this limit is the only equilibrium state. However, there is another equilibrium state for large β, which is called smoking present limit (*SPE*). In this case, *SPE* is stable while *SFE* is unstable. If all other parameters are fixed, there is a β_0 such that when $\beta \leq \beta_0$ there is only one equilibrium state, and when $\beta > \beta_0$ there are two equilibria. The value β_0 is called the critical value, which is also a bifurcation of this ODE system. The system will be called subcritical if $\beta \leq \beta_0$, and supercritical if $\beta > \beta_0$. For the ODE system, Sharomi and Gumel (2008, p. 479-480) gave an exact formula for determining whether the system is in subcritical or supercritical state. The system is in subcritical state if $R_s \leq 1$, and in supercritical state if $R_s > 1$, where R_s is given by:

$$R_s = \frac{\beta(\mu+\alpha)}{\mu(\mu+\alpha) + \gamma(\alpha\sigma+\mu)},$$

with the critical value occurring when $R_s = 1$. This gives a expression for the critical value β_0 for the ODE model, given other parameters fixed:

$$\beta_0^{ode} = \frac{\mu(\mu+\alpha) + \gamma(\alpha\sigma+\mu)}{\mu+\alpha} = \mu + \frac{\gamma(\sigma\alpha+\mu)}{\mu+\alpha}. \tag{2}$$

In both supercritical and subcritical states, there is only one stable equilibrium for the ODE

system 1, where the number of potential smokers is given by

$$P^* = \begin{cases} N \text{ if } R_s < 1, \\ N/R_s \text{ if } R_s \geq 1, \end{cases} \tag{3}$$

with R_s defined as above.

3 Description of the Lattice Model

From the ODE model above, we intend to produce a lattice model that has the same mechanism except for the interaction between potential smokers and current smokers. We will implement the lattice model in three different ways depending on the interaction between potential and current smokers.

Throughout this section, space is assumed to be a finite square two-dimensional lattice, where each vertex represents a single agent. In the global version, at each time step, an agent is randomly chosen, and possible changes are made depending on the agent's smoking status. Each potential smoker has probability $\beta S/N$ to become a smoker and $1 - \beta S/N$ to remain unchanged. Here S denotes the total number of smokers on the whole lattice, and N denotes the total number of all agents. Each smoker has probability μ to be replaced by a potential smoker, $\gamma(1 - \sigma)$ to become a temporary quitter, $\gamma\sigma$ to become a permanent quitter, and $1 - \mu - \gamma$ to remain unchanged. A temporary quitter has probability μ to be replaced by a potential smoker, α to become smoker again and $1 - \mu - \alpha$ to remain unchanged. A permanent quitter has probability μ to be replaced by a potential smoker and $1 - \mu$ to remain unchanged. Note that the requirement that all probabilities being non-negative imposes certain constraints on the parameters, namely $\beta < 1, \mu + \gamma < 1$ and $\mu + \alpha < 1$.

All rules of mechanisms in the global version are defined in the same way as the ODE system given by (1) above, and the interaction between potential smokers and smokers are global, ignoring locality. This variant of the lattice model is designed to simulate the ODE model.

Compared to this global version, there are also local versions of the lattice model. In the local versions of the lattice model, all changing probabilities remain the same as the global version, except that now potential smokers look only at smokers in their neighborhood. Both von Neumann neighborhood (4 nearest neighbors) and Moore neighborhood (8 nearest neighbors) are used. Now a potential smoker has probability $\beta S_{vn}/4$ to become a smoker in the von Neumann neighborhood case, where S_{vn} is the number of smokers among his 4 nearest neighbors. In the Moore neighborhood case the probability is $\beta S_m/8$, where S_m is the number of smokers among his 8 nearest neighbors. All these models are very similar, except for the degree of locality in the interaction between potential smokers and smokers. A potential smoker in each version look at either smokers in his neighborhood (von Neumann type or Moore type) or all smokers on the whole lattice. A diagram illustrating the mechanism of these smoking models is given in Figure 1.

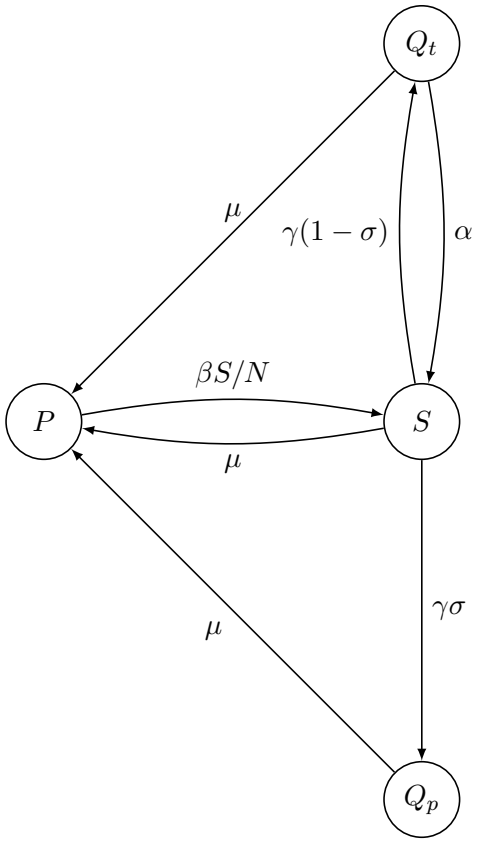

Figure 1: Interaction diagram of the PSQ smoking model.

This lattice model can be viewed mathematically as a more complicated variant of the standard SIRS model in epidemic studies ([4]). We draw its diagram of interaction in Figure 2, using our notation for parameters when applicable. In particular, we use letters P, S, Q to denote susceptible individuals, infected individuals and recovered individuals, respectively. The total population is again denoted as N.

As we can see, this PSQ smoking model depicted in Figure 1 has an additional class of temporary quitters Q_t who may revert back to smoking, while in SIRS model depicted in Figure 2, all recovered individuals are considered permanent, in the sense that they cannot directly become infected again without becoming susceptible first. The other difference is that in the smoking model, a smoker at each time step also has probability μ to become a potential smoker like the quitters, whereas in the SIRS epidemic model the infected individuals cannot directly become susceptible individuals.

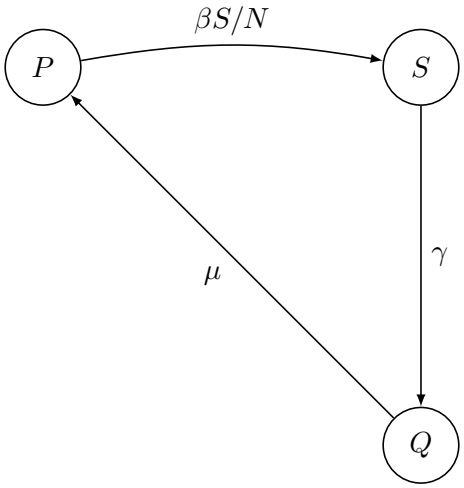

Figure 2: Interaction diagram of the SIRS epidemic model.

4 Direct Simulations and Results

We implement the above lattice model directly on a finite lattice and do some basic simulations. Throughout this section, the lattice size is set to be 50 times 50, corresponding to a total population $N = 2500$. Its boundary is assumed to periodic. Each point on the lattice represents a person who belongs to a certain smoking group, namely potential smokers, smokers, temporary quitters and permanent quitters. Time step is set to be 1. Initially everyone is set to be a smoker. Some trials of simulations are run to verify that this configuration does not affect the long time equilibrium limit.

For the global and local lattice models, we run simulations varying β. 50 simulations are executed for each β value and neighborhood type (global, von Neumann or Moore), and the average and standard deviation of potential smokers at time $T = 10^8$ are recorded as the approximated equilibrium for each set of parameters, rounding to nearest integer. These results will be compared to the ODE stable limits obtained by the formulas 3 given in [19] stated previously.

The list of parameters is summarized in Table 1.

N	α	β	γ	μ	σ	T
2500	0.9	vary	0.3	0.05	0.5	10^8

Table 1: List of parameters in the PSQ smoking model.

We also state the procedure of checking whether initial conditions for the lattice models affect the equilibrium. In this simulation β is fixed to be 0.8. For each neighborhood type (global, von Neumann and Moore), 50 trials are respectively implemented for the initial condition that all people are smoking at first, corresponding to $S = N, P = Q_t = Q_p = 0$, and for the case where only one randomly placed person is smoking at first, corresponding to $P = N - 1, S = 1, Q_t = Q_p = 0$. Again, all results are rounded to nearest integer. Note that in the second scenario when

initially only one smoker is present, the system has a substantial probability of degenerating into the *SFE* smoke-free state where $P = N$ and all other classes are 0. These trials that entered *SFE* are deleted when calculating the average. As we can see from Table 2, the lattice model has only one stable equilibrium that is independent of initial condition.

Neighborhood type	von Neumann	Moore	Global
All smoking initial avg	1449	1038	646
One smoker initial avg	1451	1029	648

Table 2: Comparison of results after changing initial condition, $\beta = 0.8$.

The main results for the simulations under the all smoking initial conditions are summarized in Table 3 and Figure 3.

β value	von Neumann nbd avg	Moore nbd avg	Global avg	ODE limit
0.2	2500 ± 0	2500 ± 0	2500 ± 0	2500
0.3	2500 ± 0	2500 ± 0	1739 ± 65	1732
0.4	2500 ± 0	2500 ± 0	1287 ± 66	1299
0.5	2500 ± 0	1819 ± 104	1043 ± 50	1039
0.6	2500 ± 0	1445 ± 100	867 ± 35	866
0.7	1720 ± 115	1188 ± 73	743 ± 36	742
0.8	1449 ± 89	1038 ± 59	646 ± 35	650
0.9	1284 ± 89	909 ± 60	578 ± 34	577
1.0	1140 ± 74	828 ± 61	529 ± 28	520

Table 3: Simulation results of average number of potential smokers at approximated equilibrium, with $\pm$ denoting standard deviation.

As we can see on the graph, the global version of the lattice model agrees very well with the ODE limit, suggesting that the global version of the lattice model simulates the ODE system very well. Although the local models also exhibit the general behavior of the transition between subcritical (*SFE* only) into supercritical states where two equilibria exist, the actual critical value β_0 and the number of potential smokers at stable equilibrium differ significantly from the global model. This suggests that under the locality and stochasticity assumption, the predictions given by the ODE model is different from the predictions given by other models that take account into these aspects, including our lattice model.

Note that the global version of our lattice model is equivalent to taking the neighborhood to be the whole lattice, since we assume periodic condition. Therefore, as shown on the graph, when the neighborhood size decreases, the number of potential smokers in the stable equilibrium increases. The critical β value, i.e. the transition point of the model from having only one smoking-free stable state *SPE* into having two equilibria, increases as neighborhood size shrinks.

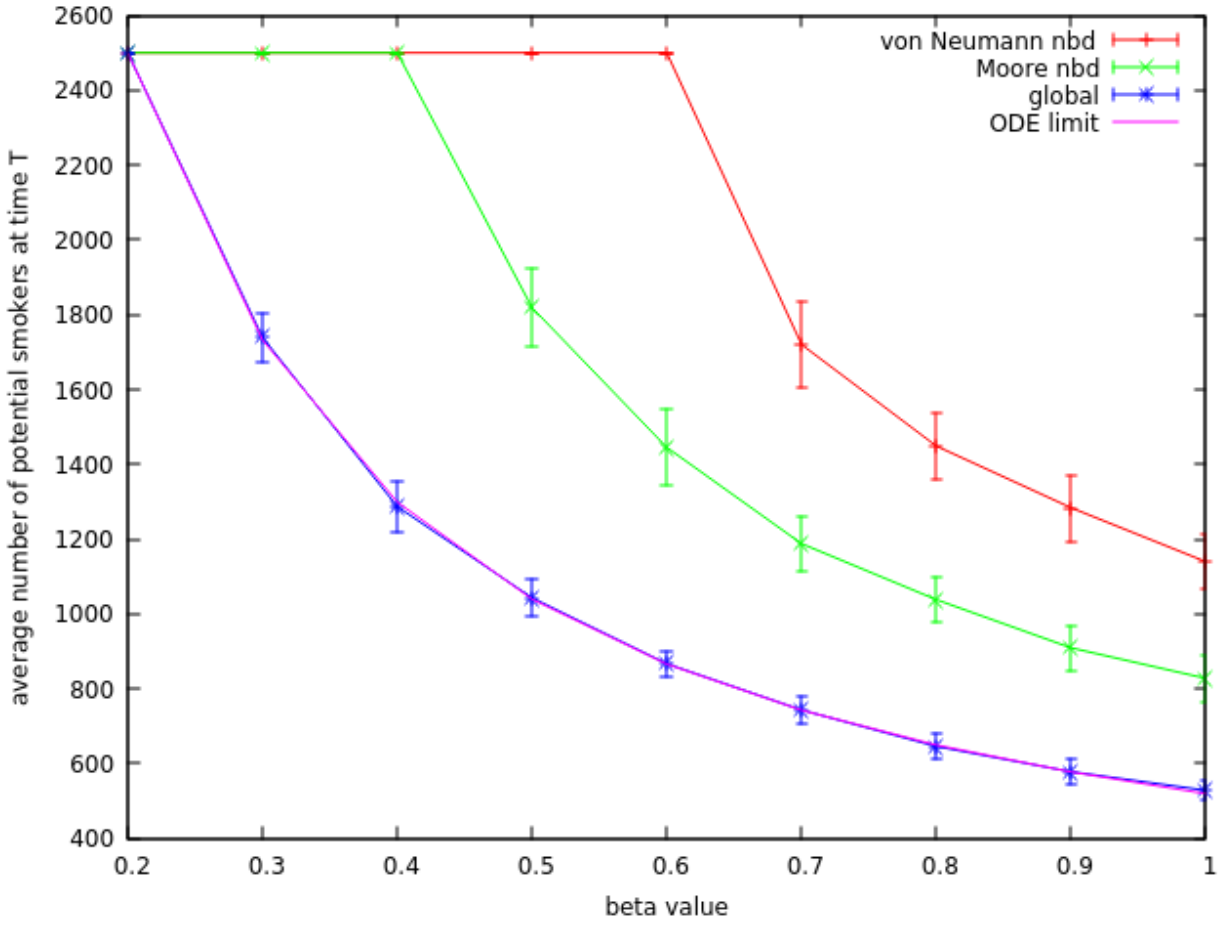

Figure 3: Plot of average number of potential smokers at approximated equilibrium, with error-bars denoting standard deviation.

5 Critical Exponents

The simulations in the previous section are not very precise, and they are in particular not suited to estimate the exact stable equilibrium and critical value β_0 given a set of parameters, since when $\beta - \beta_0$ is small, all trials have very high probability of entering the smoking-free absorbing state, especially over long term, despite the fact that $\beta - \beta_0 > 0$, and the system is in supercritical state. In fact, as long as the lattice used in the simulation is finite, the system is an absorbing Markov chain with only one absorbing state. Therefore, the probability of the system becoming smoke-free converges to 1 as time goes to ∞, no matter how large β is.

A better way to estimate the equilibrium is to use the tool called critical exponents, which also offers other insights to the lattice model. Throughout this section we fix the neighborhood type to be von Neumann type, since most research in critical exponents is done in von Neumann neighborhood settings, allowing us to compare our results with other models with known critical exponents.

It is known that a large class of lattice models exhibit great similarity in their asymptotic be-

havior near the phase transition. These lattice models can be classified into several categories, for example direct percolation and dynamic percolation ([4, 7, 15]), based on the behavior of several quantities near criticality.

For this smoking model in particular, the phase transition denotes the critical value β_0 which marks the transition from the subcritical state with one equilibrium into the supercritical state with two equilibria. If the PSQ lattice model has critical exponents, then as β approaches critical value β_0 from above and t becomes large, some quantities of the model are, on average, expected to asymptotically behave as

$$\begin{aligned} M(t) &\sim t^{\eta}, \\ B(t) &\sim t^{-\delta}, \\ R^2(t) &\sim t^{z}. \end{aligned} \tag{4}$$

The constants η, δ, z are called critical exponents, and they remain invariant as model parameters vary reasonably. Here M denotes the expected number of smokers; B denotes the expected survival probability, i.e. the probability that the system is not in the smoke free absorbing state; R^2 denotes the expected mean distance of smokers from origin. To eliminate possible bias, both Manhattan and Euclidean distances will be used.

To find out the critical exponents of the smoking model, we use a new simulation procedure based on the mechanism above. This technique is also used in other similar scenarios in [7] and [13] when estimating critical exponents of various lattice models. The β values are chosen near the critical value, after some initial probes. A list of all individuals who are not potential smokers and their physical locations is maintained. Initially the list only contains one smoker at the origin, and therefore has size 1. At each simulation step, a random individual from this list is chosen, and the overall time elapses by $\Delta t = 1/H(t)$, where $H(t)$ is the length of the list, i.e. the current total number of smokers, temporary quitters and permanent quitters at that time. Apart from the smoker-potential smoker interaction, the system evolves in a spontaneous manner in the same way as the lattice simulation, and the mechanisms are unchanged. Namely, if the chosen person is a temporary quitter, then he has probability α to become a smoker, μ to become a potential smoker, $1 - \alpha - \mu$ to remain unchanged. The permanent quitter case is entirely similar.

The smoker-potential smoker interaction is implemented as follows. If the chosen agent from the list is a smoker, then with probability β that agent attempts to influence a randomly chosen neighbor. In other words, the agent randomly chooses one out of 4 nearest neighbors, and add this person to the list as a smoker if he/she is not originally one the list, i.e. the chosen neighbor was a potential smoker before. With probability $\gamma(1 - \sigma)$ the smoker becomes a temporary quitter, $\gamma\sigma$ a permanent quitter, and μ a potential smoker. Note that this imposes a new constraint on the parameters being $\beta + \gamma + \mu < 1$. The simulation runs as long as there are still smokers or quitters, either temporary or permanent, remaining on the list. It terminates when the list becomes empty, which corresponds to the smoke-free limit.

In the calculation, 10000 trials are run for each set of parameters. Since the time steps in

all trials are different, the data for each trial is linearly interpolated at integer time, in order to add the data from different trials together and take the averages. All parameters are kept the same as in Table 1 excluding T. All trials, including those which become empty, are taken into the average of $M(t)$. For R^2, only the existing smokers in all trials are taken into account. The log-log plot for each quantity is shown in Figure 4 through 7, with initial data $t < 100$ removed to show long time behavior only. From the equation 4 above, the critical value β_0 should give a straight line on the log-log plot. So the slopes of these log-log plots can be used to estimate the critical exponents η, δ, z. To make quantitative estimates of the slope, we take $\log_2$ of all the raw data with t from 100 to 10000. Then the data are linearly interpolated (using interp1 command in Matlab) at evenly spaced points from $\log_2(100)$ to $\log_2(10000)$ (using linspace command in Matlab). Then the linear regression results using these interpolated data are recorded in Table 4.

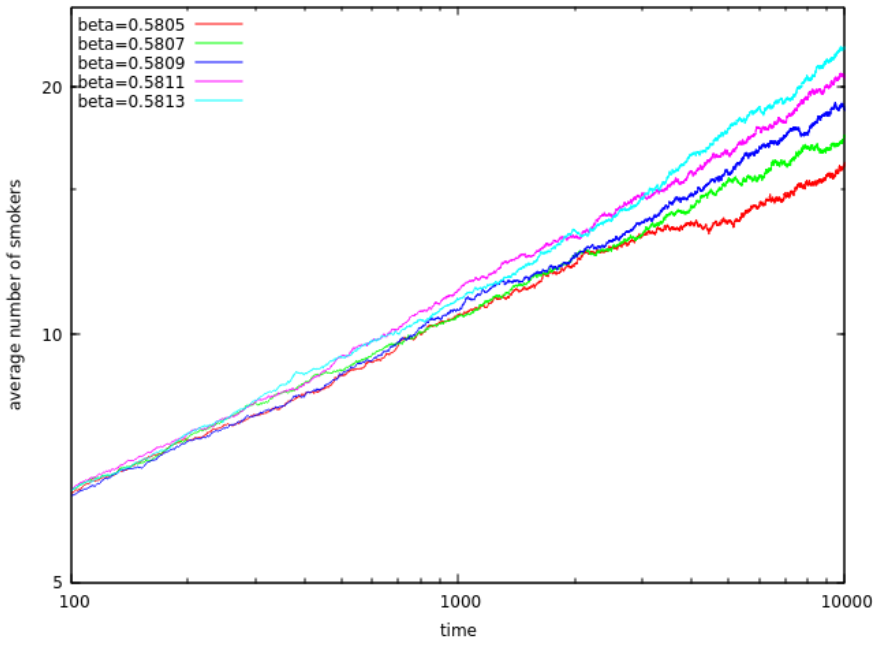

Figure 4: Log-log plot of expected number of smokers $M(t)$, varying β.

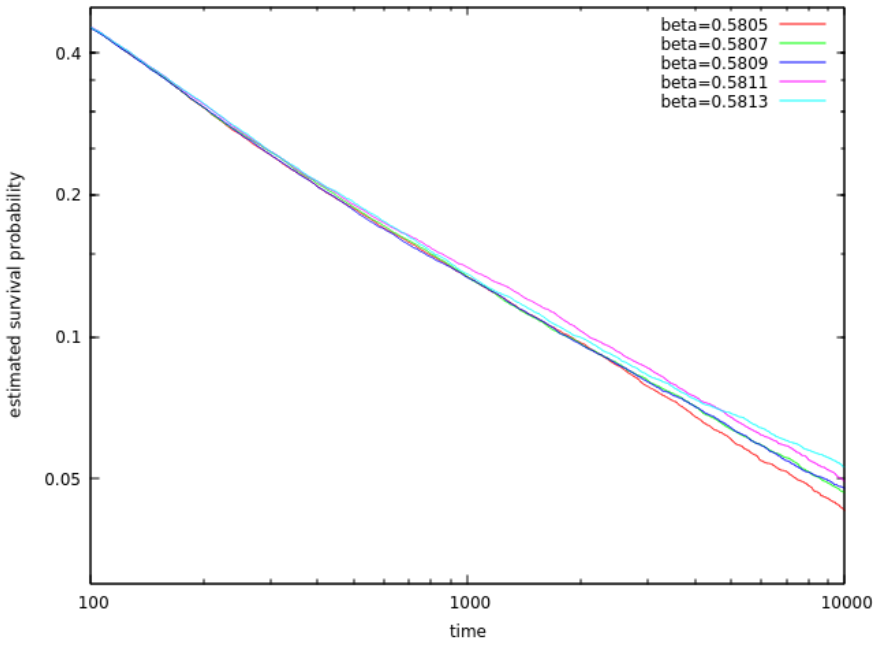

Figure 5: Log-log plot of estimated survival probability $B(t)$, varying β.

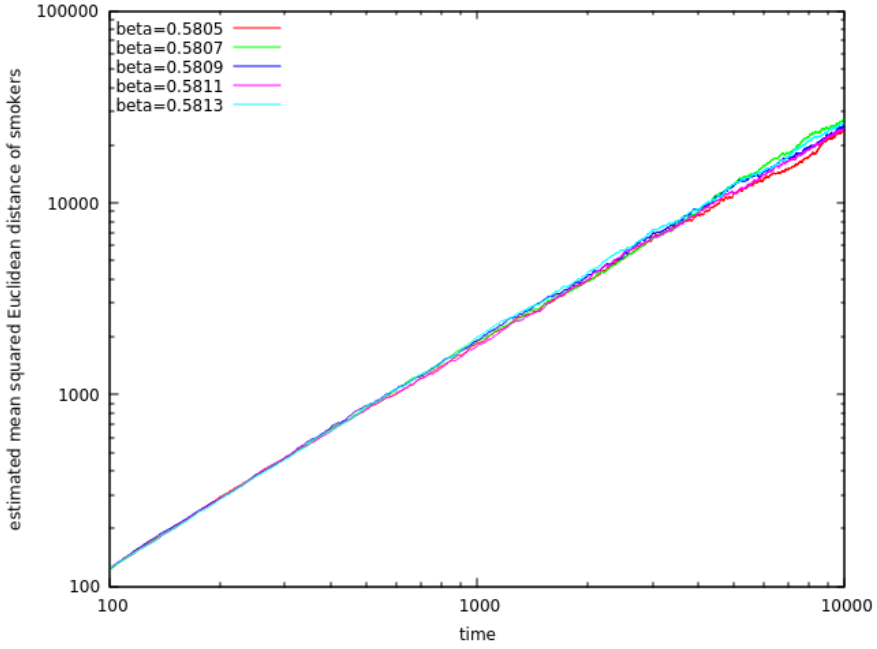

Figure 6: Log-log plot of estimated mean squared Euclidean distance of smokers $R^2_{ec}(t)$, varying β.

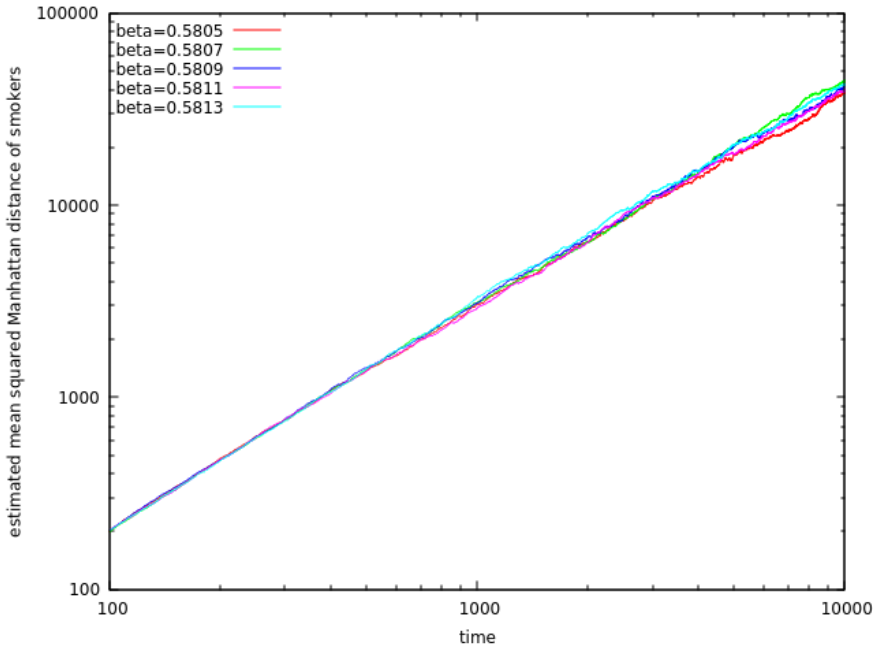

Figure 7: Log-log plot of estimated mean squared Manhattan distance of smokers $R^2_{mh}(t)$, varying β.

β value	η		δ		z			
	slope	R^2	slope	R^2	EC slope	R^2	MH slope	R^2
0.5805	0.1997	0.9937	0.5026	0.9991	1.1298	0.9996	1.1268	0.9996
0.5807	0.2162	0.9991	0.4885	0.9981	1.1640	0.9996	1.1651	0.9996
0.5809	0.2373	0.9974	0.4866	0.9976	1.1510	0.9997	1.1509	0.9997
0.5811	0.2490	0.9982	0.4726	0.9980	1.1442	0.9998	1.1444	0.9997
0.5813	0.2619	0.9932	0.4697	0.9954	1.1668	0.9997	1.1666	0.9996
Dir. Perc.	0.2295	–	0.4505	–	1.1325	–	1.1325	–

Table 4: Linear regression results for critical exponents

As we can see, the log-log plots in Figure 4 through 7 as well as the numerical results in Table 4 indicate that the critical exponents do exist for the model. The values of these exponents, as recorded in the table, are close to the critical exponents of directed percolation class lattice models ([15]), to which the SIRS epidemic model belongs, according to [4]. The data obtained from using Euclidean and Manhattan distance show that this choice does not affect the calculation of the relevant critical exponent z. This experiment also tells us that the critical value of β for this local lattice model, given other parameters the same as in Table 1, is around 0.58. Using the equation 2 on page 3 we can also directly calculate the critical value for the ODE model. In our set of parameters, it is calculated to be 0.2079, vastly different from 0.58. This also illustrates the quantitative difference between two models in terms of their respective critical value β_0.

6 A Continuum Model in One Dimension

Now, we attempt to generalize this model into continuum space. Since the interaction in this model is short-ranged, the most common way of formulating the continuum model is to use a convolution kernel ([11]). There are also other methods and techniques. For example, Postnikov and Sokolov in [16] used the Laplacian instead of a convolution kernel to formulate the model. However, in actual computation pertaining to our particular model, the Laplacian will still be computed with finite difference method, which then becomes equivalent to the convolution kernel method, as we will see later in this section.

Throughout this section, the space is assumed to be one-dimensional due to the high computational cost. We first consider the one-dimensional counterpart of the lattice model and study its basic behaviors. This model will use the two nearest neighbors for smoking-potential interaction, i.e. each potential smoker has probability $\beta S_{nn}/2$ to change into a smoker, where S_{nn} denotes the number of smokers among his two nearest neighbors. Other mechanisms remain the same as stated in section 3. In particular, please see Figure 1 for the interaction diagram.

We simulate 10000 trials for each β, with the initial condition of only one smoker at origin, and record the average number of smokers with time. It turns out that in our set of parameters listed in 4, the one-dimensional lattice model always has only the smoke free limit, and the system always belongs to the subcritical state. As shown in Figure 8, even when β is equal to the maximum value $1 - \gamma - \mu = 0.65$, the expected number of smokers goes to 0 fast, as compared to Figure 4 above in the two-dimensional case.

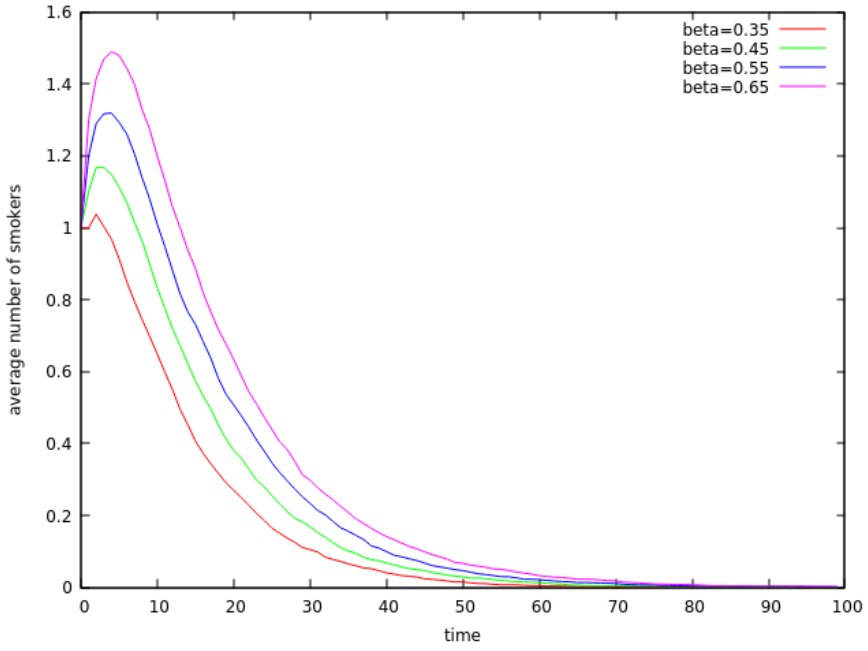

Figure 8: Plot of average number of smokers in the one-dimensional lattice model, varying β

For the one-dimensional continuum model, the presence of agents is replaced by consider-

ing its probability density function at each point on the real line. Using the convolution kernel method, the evolution of the system is defined via a set of integro-differential equations:

$$\begin{aligned} P &= 1 - S - Q_t - Q_p, \\ \frac{\partial S}{\partial t} &= -(\mu+\gamma)S + \alpha Q_t + \beta P(S * K), \\ \frac{\partial Q_t}{\partial t} &= -(\mu+\alpha)Q_t + \gamma(1-\sigma)S, \\ \frac{\partial Q_p}{\partial t} &= -\mu Q_p + \gamma\sigma S. \end{aligned} \tag{5}$$

Here each P, S, Q_t, Q_p are considered as functions of x and t, representing the probability density function of each smoking class at any given location and time. Given the constraints that all probability functions are non-negative, we ensure that the initial conditions ($t = 0$) for this system are all strictly positive functions for all P, S, Q_t, Q_p. $S * K$ is the convolution of two functions $S(x,t)$ and $K(x)$ over the spatial coordinate:

$$(S * K)(x,t) = \int_u S(u,t)K(x-u)\,du.$$

Notice that the one-dimensional lattice model described above can also be defined via this paradigm, with underlying space and time discretized with step size 1. All partial derivatives in equation 5 then become discretized as

$$\frac{\partial S}{\partial t}(x_0,t_0) = S(x_0,t_0+1) - S(x_0,t_0),$$

and so on. The β term in this case can be explicitly written as follows:

$$\beta P(x,t)\frac{S(x+1,t)+S(x-1,t)}{2},$$

corresponding to the one-dimensional lattice model where the agent is affected by his two nearest neighbors. If we compare this expression to the β term using convolution $\beta P(x,t)(S * K_{disc})(x,t)$, we see that the kernel used for the lattice version can be written as

$$K_{disc} = (\mathbb{1}_{\{-1\}} + \mathbb{1}_{\{1\}})/2,$$

where $\mathbb{1}_S$ denotes the indicator function that takes value 1 on S and 0 otherwise. Besides convolution kernel, another method is to use the Laplacian, for example in [16]. Using the Laplacian, all equations except the second are the same as above. The equation governing the change of current smokers becomes

$$\begin{aligned} \frac{\partial S}{\partial t} &= -(\mu+\gamma)S + \alpha Q_t + \beta P(S + \frac{\Delta S}{2}), \\ &= -(\mu+\gamma)S + \alpha Q_t + \beta P(S + \frac{\partial^2 S}{2\partial x^2}), \end{aligned} \tag{6}$$

since the space is one-dimensional. In actual computation, the second derivative of the probability density function S will be computed by a finite difference method of a certain order. This will

make the computation equivalent to the convolution kernel method. For example, if we choose to use the second order finite difference to compute the second derivative as

$$\frac{\partial^2 S}{\partial x^2}(x,t) \approx \frac{S(x-h,t) + S(x+h,t) - 2S(x,t)}{h^2},$$

where h is the spatial discretizing step, then after spatial discretization, the equation (6) is equivalent to equation (5) after choosing the convolution kernel in the actual computation to be

$$K_{lapl} = \frac{1}{2h^2}\begin{bmatrix} 1 & -2+2h^2 & 1 \end{bmatrix},$$

with the same discretizing step h. Therefore, in this section we will only concern ourselves with the convolution kernel method.

The convolution kernel K must satisfy the normalization condition $\int K(u)\,du = 1$. After some initial probes and simulations, we found that kernels with large spatial variation, including the kernel K_{lapl} above derived from Laplacian, would make the computation stiff and ill-conditioned.

We therefore choose a kernel that varies slowly in space. In this experiment we choose the kernel to be

$$K(x) = \begin{cases} (3-|x|)/9 & \text{if } |x| < 3, \\ 0 & \text{otherwise.} \end{cases}$$

This kernel has the advantage that it is easy to compute, and it gradually decays as the distance from origin increases.

We solve the system numerically using forward Euler method. Time and space are discretized with $\Delta t = 0.01$ and $h = 0.05$. The initial condition probability density is assumed to be $S(x,0) = 0.5$ if $x \in [-1,1]$, and 0 for all other places. Q_t and Q_p are assumed to be 0 initially.

We make the hypothesis that this continuum model behaves in the long run like the ODE model rather than the lattice model. So we run the numerical simulation at β close to ODE critical value 0.2079. The result, as shown in Figure 9, confirms this hypothesis.

Here the number of smokers is interpreted as the integral of probability density function $\int_{-\infty}^{\infty} S(x,t)\,dx$, or the corresponding discrete sum in the numerical computation. After the rapid initial decrease of the smokers, its long term behavior is determined by whether the β value is above the ODE critical value or not. If $\beta > \beta_0 \approx 0.2079$, the expected number of smokers will eventually increase, and vice versa. As the probability density of smokers sufficiently diffuse across the space, the spatial component no longer plays a big role, and the model in the long term behaves similarly to the spatial-independent ODE model, i.e. governed by the same critical value. Due to the computational limitation and difficulties, we do not know whether other convolution kernels also exhibit this kind of behavior.

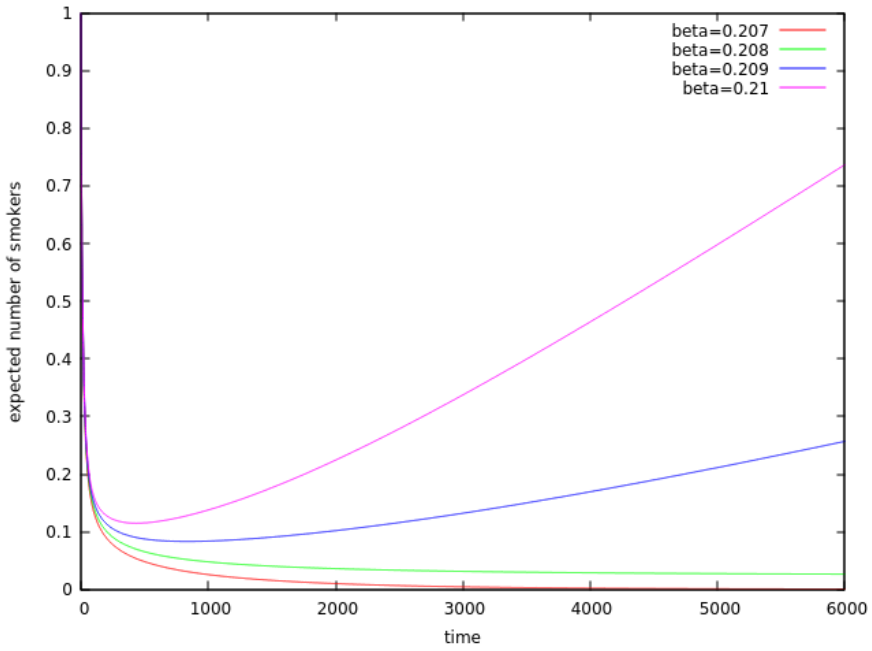

Figure 9: Number of smokers in the continuum model near ODE critical value, using convolution kernel method.

7 Comparing with Existing Works

In this section we directly compare our result with existing works on PSQ model where locality is not taken into account. We will mainly use the numerical results obtained by Sharomi and Gumel in [19] on page 483. In this section we use the same parameters as the simulation in their paper. Due to the nature of our agent-based lattice model, we scale all parameters (except σ) by 1/10 to make them less than 1. We run the simulation on a square lattice with size $n = 40$, which gives the same total population 1600 as in [19], listed in table 2. Given the scale factor of 1/10, in addition to the fact that at each time step in our model, only 1 out of n^2 agents is selected, $10n^2$ time steps in our simulation would correspond to the timespan of 1 year in [19]. In our simulation, we run 50 trials for each β value, and stop after 250 years, corresponding to 4 million time steps in each trial. We record the average number of potential smokers P_{lat} for each β, classify the β values based on the equilibrium type, and compare with the number of potential smokers at ODE equilibrium P_{ode}, listed in Table 2 in [19]. We also record the stable equilibrium (SE) type for each version.

N	α	β	γ	μ	σ	T
1600	0.3	vary	0.03	0.005	0.5	4×10^6

Table 5: List of parameters in the simulation for direct comparison, after scaling.

β	R_s	P_{lat}	P_{ode}	lattice SE type	ODE SE type
0.2	0.7896	1600	1600	smoking free	smoking free
0.3	1.1844	1600	1351	smoking free	smoking present
0.5	1.9741	1580	811	smoking present	smoking present
1	3.9482	762	405	smoking present	smoking present

Table 6: Comparison of average number of potential smokers at approximated equilibrium between our lattice model and the ODE model.

It can be seen from Table 6 that in some cases, namely when $\beta = 0.3$ in our set of parameters, the smoking free equilibrium is not stable in the lattice model even if the ODE counterpart with the same set of parameters is stable in the smoking free state, namely with $R_s < 1$. As mentioned in section 2,

$$R_s = \frac{\beta(\mu + \alpha)}{\mu(\mu + \alpha) + \gamma(\alpha\sigma + \mu)}$$

is the threshold for the ODE system, and is called the "smokers-generation number" in [19]. Sharomi and Gumel stated that "the number of smokers in the community will be effectively controlled (or eliminated) at steady-state" whenever $R_s < 1$. However, our result suggests that this is not the case for the lattice model, where locality of interaction is taken into account. Namely, $R_s < 1$ is not a good threshold for smoking officials to effectively control the smoking behavior in the long term, when potential smokers are affected only locally by their smoking neighbors. Our numerical results here are more optimistic, in the sense that when all other factors remain the same, the contact rate between potential smokers and current smokers could be a little higher for the smoking behavior to be still effectively under control.

Our result also implies that their analysis and results for their extended model may also need modifications when the interaction between potential smokers is local. This also applies to other existing works that use ODE model approach, including [1],[10], and so on.

8 Discussion and Further Steps

This paper explores the lattice version of the PSQ smoking model, and shows that its behavior is significantly different from the ODE version. Under the assumption that interaction between people with different smoking behavior is stochastic and dependent on spatial vicinity, this result implies that many implications and numerical estimates obtained from the ODE model might need reconsideration in order to take these aspects into account.

The lattice model studied in this paper can be extended in a number of ways. Some additional mechanisms that can be implemented include setting death rate to be different based on smoking status, allowing agents to move to different location, changing grid shape, introducing random noise in each simulation step, and so on. This paper studies only the basic model, and

gives some additional analysis about the critical exponents and a continuum model, in section 5 and 6.

Many results in these two sections can be strengthened by additional computations. In particular, we only state that the critical exponents of the PSQ lattice model are close to direct percolation constants, but have not given a more definitive answer. In addition, every trial in the direct simulation as well as the simulation for critical exponents behaves very different from each other. Some trials terminate very early, whereas some others grow really big. Thus, the variation is very high, and increasing our simulation accuracy would require much more computational power.

In addition to numerical simulation, there are other ways to study these types of lattice models and determine critical value, critical exponents, effect under perturbation, and so on. One approach is to use operators and series expansions. Jensen and Dickman [8] used this method to study the one-dimensional generalized contact process model. However, our original PSQ lattice model is two-dimensional, and the mechanism in our model is also much more complicated compared to the mechanism of generalized contact process, in which there are only two states for each individual. Therefore, it is unclear whether series expansion is an effective method in our situation.

Under the current configuration of the lattice model, we made the remark in section 7 that unlike its ODE counterpart, $R_s < 1$ is not a good criterion for the effective control of smoking behavior for our model. We suggested that the criterion will be less strict for the lattice model, but did not give a quantitative characterization of it. More work is needed to find an analytic formula that exactly or approximately determines when the smoking free equilibrium is stable, i.e. the smoking behavior is effectively controlled.

For the continuum model, we only studied the numerical results using discretization of both time and space. Further mathematical analysis remains to be done for both differential equation systems 5 and 6.

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