Modeling of surface reactions

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1. GENERAL INTRODUCTION

In this thesis, we analyze mathematical models for the kinetics of chemical reactions occurring on surfaces. Two specific surface reactions are identified and investigated: The monomer-monomer (or $AB$) reaction, and the monomer-dimer (or $AB_2$) reaction. Models are developed, in both cases, using stochastic methods and also using mean-field theory (reaction-diffusion equations). Both methods have advantages and disadvantages which will be discussed in this introduction. The important fact concerning both of these types of mathematical models is that they give somewhat different results (due to fluctuations in stochastic models), but converge in the regime of high diffusion rates. This will be shown in the body of the thesis. First, we will briefly describe this discipline, starting with the idea of applied mathematics in general and gradually becoming more specific.

Historically, mathematics and the sciences have always been closely associated. The mathematician Albert Einstein, when defining physics, wrote [1], “What we call physics comprises that group of natural sciences which base their concepts on measurements; and whose concepts and propositions lend themselves to mathematical formulation. Its realm is accordingly defined as that part of the sum total of our knowledge which is capable of being expressed in mathematical terms” (p. 98). This is clearly no longer an accurate definition, as it encompasses too much. Chemistry, biology, epidemiology, among others, all fit this definition to some extent. Lin and Segal [2] assert that this definition is more a description of applied mathematics. Using this thought, we modify Einstein’s statement into the following definition:

What we call applied mathematics comprises the mathematics\(^1\) used by that group of disciplines which base their concepts on measurements; and whose concepts and propositions lend themselves to mathematical formulation.

\(^{1}\)The reader will supply her/his own definition of mathematics (I have neither the time nor the strength to undertake such an endeavor).
There is seldom confusion between applied mathematics and experimental science, or between theoretical science and pure mathematics, but where is the separation between applied mathematics and theoretical science? The debate is ongoing, and again, Lin and Segal [2] give a reasonable distinction between these two disciplines: "It is often the case that a theoretical scientist, from long study of his particular subject, has a deeper knowledge of a certain discipline. An applied mathematician, by contrast, may work in more than one discipline and cross-fertilize each. Indeed, in these times of increasing specialization, cross-fertilization is one of the most useful and satisfying activities of an applied mathematician" (p. 7)

There are two general approaches to applied mathematics. One approach is to start with a scientific problem and, through limiting assumptions, arrive at a mathematical model for the problem, which can be solved and evaluated as to its relevance. The other approach is create or extend mathematical theory in directions that appear to have applications in science. There are some that claim the former is theoretical science, and others that claim the latter is pure mathematics. We call both approaches applied mathematics, but will use the former approach in this dissertation by formulating and evaluating mathematical models of chemical reactions on surfaces.

A survey of several mathematical modeling texts [3] shows that the process can be described in anywhere from three to seven (or more) steps. While each author has personal biases, three steps appear in each description:

(1) formulation of the model,
(2) solution of the model, and
(3) evaluation of the results.

The last step can often lead to starting over with the first step and the formulation of a new or refined model. Of these three steps, no one step is more important than another. Each contributes to a complete answer of the problem being modeled, and
also gives information that can be used in other problems (often in other disciplines).

**Chemical Kinetics**

Chemical kinetics, or the measurement of chemical reaction rates, has its roots in the work of L. Wilhelmy in 1850. He investigated the inversion of cane sugar in acid solutions, finding that the rate of decrease of sucrose (sugar) was proportional to the amount still present, i.e.

\[ \frac{dS}{dt} = kS \]  

where \( S = S(t) \) is the concentration of sucrose at time \( t \) [4]. This leads to the familiar differential equation for exponential decay. Wilhelmy's reaction is actually modeled quite accurately by the first order reaction

\[ A \rightarrow P, \]

where \( A \) is the reactant and \( P \) is the product of the reaction. This reaction can be approached and solved deterministically, that is, by directly solving the rate equation (1).

This approach works in some reactions, particularly those occurring in a dilute solution. There are several types of reactions, though, where this deterministic approach will not work [5]. Among these are diffusion controlled reactions and adsorption of gases onto solid surfaces [5], both of which are the kind of reactions considered in parts 2 and 3 of this paper.

Chemical reactions involving gases can be divided into three categories:

1. **Homogeneous reactions.** These are reactions which occur entirely (or very nearly so) in the gas phase.
2. **Heterogeneous reactions.** These are reactions which occur entirely (or very nearly so) on a surface.
3. **Reactions which occur partly in the gas phase and partly on the surface.**
The reaction models we develop are for heterogeneous reactions. The catalyst, or surface on which the reactions occur, is often a metal, but not restricted to such.

In 1909 Fritz Haber, a German chemist, discovered a catalytic process to mass produce ammonia from nitrogen and hydrogen gas. Upon his discovery, he tested more than 1,000 materials as catalysts before deciding that iron was the best choice (manufacturers today use a mixture of iron, potassium and calcium as the catalyst) [6]. Since his discovery, catalysts have been used for a myriad of processes: cleaning automotive exhaust using the platinum family of metals, the synthesis of high octane gasoline using zeolites, and the removal of sulfur from fossil fuels using a catalyst consisting of molybdenum, cobalt and sulfur itself [6,7].

In order for a surface reaction to take place, one or more of the reactants must be adsorbed onto the surface. This was first proposed by Faraday in 1825 [4], who postulated that the main effect of the catalyst was to allow the reactant molecules to be much more highly concentrated than in the gas state. This view was of course proved false when it was shown that the same reactants with different catalysts can produce different products of reaction. Thus, specific forces are involved in the adsorption process.

There are two main types of chemical adsorption on a surface. The first type are due to physical forces known as van der Waals forces, corresponding to forces in the van der Waals equation of state of gases. Adsorption of this type is relatively weak and plays a negligible role in surface reactions. The second type of adsorption is known as chemisorption, and is considerably stronger than adsorption due to van der Waals forces. Chemisorption is due to valence forces of the same type as those that bound atoms in molecules have, and are fundamental to the adsorption and reaction stages of surface reactions.

There are also two common theories for the reaction mechanism for surface reactions. The first type is known as the Eley-Rideal mechanism, which is a reaction
of an adsorbed reactant with either gaseous reactant or a reactant adsorbed due to van der Waals forces. The second type is known as the Langmuir-Hinshelwood mechanism, which is a reaction between adsorbed species. The actual chemical reactions being modeled in this paper are motivated from oxidation of carbon monoxide on platinum. Experimental results on these reactions show that the Langmuir-Hinshelwood mechanism is the only clearly consistent mechanism [8].

When the reaction takes place, the reaction product is assumed to have a weak bond with the surface, so it immediately desorbs into the gas phase, and from there it has no effect on future adsorption or reaction. In some models it is appropriate to surface diffusion and/or non-reactive desorption, but these processes vary greatly depending on the reaction and on the catalyst. In parts 3 and 4 we will discuss in detail the effects of surface diffusion on a particular reaction.

The surface reactions modeled in this paper are motivated by the oxidation of carbon monoxide on platinum. In their review, Razon and Schmitz list 93 distinct models for this reaction [9]. These and more general reaction models can often be characterized as follows: We adopt a convention of calling the reactants $A$ and $B$, and consider $A_mB_n$ reactions described by

$$A_m + mE \overset{y_A}{\rightarrow} mA(ads), \quad B_n + nE \overset{y_B}{\rightarrow} nB(ads),$$

$$A(ads) + B(ads) \xrightarrow{k} AB + 2E.$$  

Here $E$ represents an empty site on the square lattice of adsorption sites, and $ads$ denotes adsorbed species (as opposed to gas-phase species). The impingement rates\(^2\) of $A$ and $B$ are $y_A$ and $y_B$, respectively, and the reaction rate is $k$. Adsorption occurs only if $m$ (or $n$) adjacent sites are empty.

\(^2\)We adopt the standard convention of normalizing these so that $y_A + y_B = 1$.  

Mean-Field Theory

In a well mixed system (e.g., in surface reaction systems with high surface diffusion rates), a mean-field theory can be adopted for analysis. Starting with the standard $A_mB_n$ reaction, we illustrate the development of such mean-field rate equations and reaction-diffusion equations. We use the notation $\theta_J$ as the probability that the configuration $J$ is present (or in other words the coverage of the configuration $J$). We are thus fundamentally concerned with the evolution of $\theta_A$ and $\theta_B$ due to adsorption and reaction. We define $R_A$ and $R_B$ as the rates of change of $\theta_A$ and $\theta_B$, respectively, due to adsorption and reaction.

Growth of $\theta_A$ (or $\theta_B$) occurs when an attempt is made to adsorb $A$ (or $B$) and the sites where the attempt is made are a string of $m$ (or $n$) adjacent empty sites denoted $E^m$ ($E^n$). Decline of $\theta_A$ (or $\theta_B$) occurs when a reaction takes place (since the $AB$ molecule immediately desorbs). This occurs at rate $k$ for each neighboring $AB$ pair. We restrict our studies at this point to a square lattice, and allow only nearest-neighbor pairs to react. Quantifying this discussion, we get

$$R_A = my_A\theta_{E^m} - 4k\theta_{AB}$$
$$R_B = ny_B\theta_{E^n} - 4k\theta_{AB}.$$  \hspace{1cm} (2)

where the factor of 4 is required since each $A$ ($B$) could potentially react with a $B$ ($A$) on any of 4 neighboring sites (for a square lattice).

Since we are assuming a well mixed system, we conclude that there are no correlations between sites, so that for the mean field theory we can assume that $\theta_{E^m} = (\theta_E)^m$ and $\theta_{AB} = \theta_A\theta_B$. In the case of a spatially homogeneous system, i.e.

$$\frac{\partial \theta_A}{\partial t} = R_A \quad \text{and} \quad \frac{\partial \theta_B}{\partial t} = R_B,$$

\footnote{This is not true in general. Consider, for example, a reaction that leads to the development of clusters of like reactants and, for simplicity, consider a $10 \times 10$ finite lattice. If the $A$ and $B$ reactants cluster completely, i.e. all $A$'s on one side and all $B$'s on the other, then $\theta_A\theta_B = 0.25$ but $\theta_{AB} = 0.1$ (or 0.2 if we assume periodic boundary conditions).}
analyses can be made concerning the steady state. By setting $\frac{\partial \theta_A}{\partial t} = \frac{\partial \theta_B}{\partial t} = 0$, we get

$$my_A (\theta_E)^m = ny_B (\theta_E)^n.$$  

We can solve this equation for $\theta_E$ (assume $n > m$; other cases have similar derivations):

$$\theta_E = \left( \frac{my_A}{ny_B} \right)^{\frac{1}{n-m}} \text{ (if } my_A < ny_B \text{) or 0 (poisoned cases } \theta_A = 1, \theta_B = 1).$$  

Due to conservation of probability, we can substitute $\theta_B = 1 - \theta_A - \theta_E$ into the original equation for $R_A$, and get

$$\frac{\partial \theta_A}{\partial t} = 0 = m y_A (\theta_E)^m + 4 k \theta_A \theta_B$$  

$$= m y_A (\theta_E)^m + 4 k \theta_A (1 - \theta_A - \theta_E).$$  

Solving this quadratic equation for $\theta_A$ we have

$$\theta_A = \frac{1}{2} (1 - \theta_E) \pm \frac{1}{2k} \sqrt{k^2 (1 - \theta_E)^2 - km y_A \theta_E^m}$$  

where $\theta_E$ is determined above. For values of $y_A$ that give a positive value for the terms inside the square root sign above, we have 2 reactive steady states in addition to the 2 poisoned states. When those terms are zero, we are at what is called the spinodal point.

In an inhomogeneous system, we can adopt the standard diffusion terms

$$\frac{\partial \theta_A}{\partial t} = R_A + D_A \nabla^2 \theta_A$$  

$$\frac{\partial \theta_B}{\partial t} = R_B + D_B \nabla^2 \theta_B$$

where $R_A$ and $R_B$ are the adsorption/reaction terms defined above, and $D_A$ and $D_B$ are the diffusion coefficients associated with $A \leftrightarrow E$ and $B \leftrightarrow E$ place exchange,
respectively. These equations, however, do not guarantee solutions with \( \theta_A + \theta_B \leq 1 \). We must add corrective terms to these equations [10]:

\[
\frac{\partial \theta_A}{\partial t} = R_A + D_A \nabla^2 \theta_A + (D_{AB} - D_A) (\theta_B \nabla^2 \theta_A - \theta_A \nabla^2 \theta_B)
\]

\[
\frac{\partial \theta_B}{\partial t} = R_B + D_B \nabla^2 \theta_B + (D_{AB} - D_B) (\theta_A \nabla^2 \theta_B - \theta_B \nabla^2 \theta_A),
\]

where \( D_{AB} \) is the diffusion coefficient associated with \( A \leftrightarrow B \) place exchange.

Insight into the behavior of these equations can be found by studying a field of interdisciplinary research called synergetics. Synergetics deals with how disordered states of complex systems may lead to ordered states through self-organization. The basic concepts of this have applications in a broad spectrum of fields, including physics, chemistry, biology and sociology [11]. Often these systems exhibit bistability. In this case their basic behavior can be described by a simple one-component model where each individual element \( u \) can be described by

\[
\frac{\partial u}{\partial t} = f(u) + D \nabla^2 u.
\]

where \( f(u) \) is shown in Fig. 1.

The values \( u_1, u_2 \) and \( u_3 \) correspond to possible homogeneous steady states since at those points \( f(u) = 0 \). An analysis of small perturbations from these points shows that \( u_1 \) and \( u_3 \) are stable steady states and \( u_2 \) is an unstable steady state. While the stable states are stable with respect to small deviations, it is possible to have larger deviations that lead to transitions between states.
Figure 1. The function $f(u)$. 
The characteristic form of the solutions of these types of equations is a trigger, or traveling wave. Trigger waves are so named because they trigger a transition from one stable steady state to another.

When seeking a solution in the form of a traveling wave, we look for a solution of the form

$$u = u(z)$$

where

$$z = x - ct.$$  

Here $c$ is the wave speed, and $u$ satisfies the boundary conditions

$$u \to u_3, \quad z \to -\infty,$$
$$u \to u_1, \quad z \to \infty.$$  

Substituting $u(z)$ into equation (5) yields the ordinary differential equation

$$Du_{zz} + cu_z + f(u) = 0,$$  

whose solution has been shown to exist and to be unique [12]. A sample trigger wave is displayed in Fig. 2.

If $c > 0$ the state with $u = u_3$ displaces that with $u = u_1$ (we say the former is more stable than the latter). If $c < 0$, then stability is reversed. This phenomena is strikingly similar to first-order phase transitions in equilibrium physical systems [13]. If a system is in a less stable state, a strong perturbation may possibly force the system to the more stable state.

Consider a system in a less stable state except for a finite hyperspherical nucleus (an interval in $1-D$, a circle in $2-D$, etc.) which is in a more stable state. If the region is too small it will shrink, but if it is sufficiently large it will expand. There is then, a size at which the region neither shrinks nor expands. We call this the critical size, and correspondingly define a critical nucleus as a region of critical size. Also, the radius of this hyperspherical critical nucleus is called the critical radius.
Figure 2. An example of a typical trigger wave. The width $l$ of the transition layer is shown.
In a two-dimensional system, we can investigate the critical nucleus as follows. We choose polar coordinates with the center of the circular nucleus at the origin. Thus, the problem reduces to a one-dimensional equation

\[ \frac{\partial u}{\partial t} = f(u) + \frac{D}{r} \frac{\partial u}{\partial r} + D \frac{\partial^2 u}{\partial r^2}. \] (7)

The trigger wave solutions of this equation exhibit the same characteristic shape as shown in Fig. 2 for the planar interface. From Fig. 2 it is clear that \( \partial u/\partial r \) is negligible except in the transition layer. Thus, if the radius \( R \) of the circular wave front is large compared to the width \( l \) of the transition layer, we can make an approximation by replacing \( r \) by \( R \) in the \( D/r \) term, giving

\[ \frac{\partial u}{\partial t} = f(u) + \frac{D}{R} \frac{\partial u}{\partial r} + D \frac{\partial^2 u}{\partial r^2}. \] (8)

We let the velocity of the circular trigger wave be denoted by \( c(R) \), and assume a solution of the form

\[ u = u(z) \]

where

\[ z = r - c(R)t. \]

Substituting the solution into equation (8), we get

\[ Du_{rr} + \left( c(R) + \frac{D}{R} \right) u_r + f(u) = 0. \] (9)

Putting \( c = c(R) + \frac{D}{R} \) where \( c = c(\infty) \) is the flat trigger wave velocity (see equation (6)), equation (9) coincides with equation (6). Thus, at the critical radius \( R^* \), we need \( c(R) = 0 \), which gives

\[ R^* = \frac{D}{c}. \]
Comparison of Mean-Field and Lattice Gas Models

As was previously mentioned, there are many instances when a mean-field treat­ment of a reaction is not exact [5], in particular the case of surface reactions with low surface diffusion. In these cases, due to the presence of correlations which produce deviations from the mean-field theory, a lattice gas approach is necessary.

In this approach we start with a lattice (in all of our cases it is a square lattice), and through Monte Carlo simulations mimic the reaction. At each discrete time step (called an attempt), a lattice site is randomly selected. For reaction rate $k$ with $0 < k < \infty$ a decision is then made as to whether to attempt to adsorb or to attempt to react with the appropriate weight. The attempt is made, and the lattice is modified depending on the outcome of the attempt.$^4$ A natural time scale arises which assigns one unit of time for every $n^2$ attempts on an $n \times n$ lattice. Thus, as the size of the lattice increases, the number of attempts for one time unit increases, and thus the accuracy of the simulation increases.

The first reaction model treated herein is the so-called monomer-monomer, or $AB$ model. In our studies of this reaction we ignore surface diffusion and non-reactive desorption effects. The monomer-monomer surface reaction is shown schematically as

$$A + E \xrightarrow{y_A} A(ads), \quad B + E \xrightarrow{y_B} B(ads),$$

$$A(ads) + B(ads) \xrightarrow{k} AB + 2E.$$

This system has the exact rate equations

$$\frac{d\theta_A}{dt} = y_A \theta_E - 4k\theta_{AB} \quad (10)$$

$$\frac{d\theta_B}{dt} = y_B \theta_E - 4k\theta_{AB},$$

$^4$It is possible for nothing to occur, e.g. an attempt to adsorb on a filled site will result in no change, and the reaction attempt picks a neighboring site at random. If this site cannot react nothing changes, even if another neighboring site is capable of reacting.
where $\theta_A$, $\theta_B$ and $\theta_{AB}$ are as previously defined.

For $y_A \neq y_B$, poisoning follows immediately from a study of these rate equations. We naturally turn our studies to the case $y_A = y_B$.

In the case $y_A = y_B = 1/2$, we look at the rate equations:

$$
\begin{align*}
\frac{d\theta_A}{dt} &= \frac{1}{2} \theta_E - 4k\theta_{AB} \\
\frac{d\theta_B}{dt} &= \frac{1}{2} \theta_E - 4k\theta_{AB},
\end{align*}
$$

(11)

If we start with an empty lattice ($\theta_A = \theta_B = 0$), we see that there is no mechanism to make $\theta_A \neq \theta_B$. Using a mean-field approximation (i.e., no correlations, so $\theta_{AB} = \theta_A\theta_B$), we solve these equations for the homogeneous steady state. Note that, since $\theta_A = \theta_B$ and $y_A = y_B$, the equations in (11) are identical. Solving, we get:

$$
\frac{d\theta_A}{dt} = 0 = y_A\theta_E - 4k\theta_{AB} = \frac{1}{2} (1 - \theta_A - \theta_B) - 4k\theta_A\theta_B,
$$

$$
\frac{d\theta_B}{dt} = 0 = y_B\theta_E - 4k\theta_{AB} = \frac{1}{2} (1 - 2\theta_A) - 4k\theta_A^2.
$$

Solving this quadratic, we have,

$$
\theta_A = \frac{\sqrt{1+8k} - 1}{8k} = \frac{1}{\sqrt{1+8k} + 1}
$$

This gives the steady state concentrations of $AE$ and $BE$ as

$$
\theta_{AE} = \theta_{BE} = \theta_A\theta_E = \theta_A (1 - 2\theta_A)
$$

Therefore, as $k \to 0$, the mean-field theory has $\theta_A \to 1/2$ which shows that pair approximations for $\theta_{AB} \to 1/4$, and also that $\theta_{AE} \to 0$, as $k \to 0$. In contrast, we

\footnote{We normalize the total adsorption rate to $y_A + y_B = 1$, so $y_A = y_B = 1/2$.}
know from the lattice gas simulations that\(^6\) \(\theta_{AA'} = \theta_{AB} + \theta_{AE}\) decays in time by the form \(\tau^{-w}\) where \(w = 0.063 \pm 0.02\) and \(\tau = O(10^3)\).

The clustering can be observed directly from simulations (see Fig. 3) on a small surface lattice (50 \(\times\) 50 grids). As time evolves, one sees clusters of both reactants forming, then one reactant completely overcoming the other. However, is it possible, at some time, for these clusters to stop growing, and thus reach a reactive steady state?

One simple explanation of the steady coarsening, and thus against a reactive steady state, is given for the case of infinitesimal reaction rate (referred to as \(k = 0^+\)), and is shown in Fig. 4. [14] The idea is that with an infinitesimal reaction rate, each site is always occupied by either an \(A\) or a \(B\). When a reaction occurs, the two reactive sites are immediately filled. Looking at a single site, we are concerned initially with only one of the surrounding sites (i.e., the site that will be tested for reaction). If that site is another \(A\), we have started the formation of a cluster, and now consider one of the three remaining sites. If that site is also \(A\), our cluster has grown. If, at any time, the adjacent site being considered is a \(B\), our original site reacts with it, and both sites are repopulated, effectively starting the entire process over again. While not rigorous, this analysis demonstrates the mechanism which causes clustering.

A compelling argument against a reactive steady state comes from looking at a model for a different, yet similar, reaction: The dimer-dimer, or \(A_2B_2\) surface reaction model (with no surface diffusion or non-reactive desorption). This reaction is shown schematically as

\[
A_2 + 2E \xrightarrow{y_A} 2A(ads), \quad B_2 + 2E \xrightarrow{y_B} 2B(ads),
\]

\[
A(ads) + B(ads) \xrightarrow{k} AB + 2E.
\]

\(^6\)\(A'\) corresponds to a 'not \(A\)' site, i.e. a site containing either a \(B\) or an \(E\). For \(k = 0^+\), \(\theta_{AA'} = \theta_{AB}\), for \(0 < k < \infty\), \(\theta_{AA'} = \theta_{AB} + \theta_{AE}\), and for \(k = \infty\), \(\theta_{AA'} = \theta_{AE}\).
Figure 3. \( AB \) lattice gas model for a finite lattice.

Slow poisoning due to clusters is shown as \( \tau \) increases.
Figure 4. Schematic diagram showing the mechanism for clustering in the monomer-monomer surface reaction with infinitesimal reaction rate.
Here we consider only the case \( y_A = y_B = \frac{1}{2} \) (for other cases, one can show that the system "rapidly" poisons).

This surface reaction model, when \( k \) is infinitesimal, has been solved analytically. In fact, the \( A_2B_2 \) surface reaction model with \( k = 0^+ \) is equivalent to a model for spatial conflict. The voter model, as it is now commonly known, was originally a model of two opposing forces on a lattice in one or more dimensions [15]. If two adjacent sites are occupied by opposing forces, a conflict or invasion takes place where one species occupies both sites (each species has an equal probability of conquering the other). The paper gave a rigorous proof that, in two dimensions, one species will eventually completely conquer the other (with probability 1). This means that in the \( A_2B_2 \) model slow poisoning through clustering always occurs (for \( y_A = y_B = 1/2 \)). A logical direction is to compare the voter model with the monomer-monomer surface reaction model. If they evolve in an equivalent manner, this would thus indicate that the same type of slow poisoning is also certain in the monomer-monomer case. Simulations show this similarity, so we conclude that slow poisoning is indeed certain in the monomer-monomer surface reaction model (for \( y_A = y_B = 1/2 \)).

The second reaction model that is treated is the so-called monomer-dimer, or \( AB_2 \) model. In our studies of this reaction we include surface diffusion of adsorbed species, but still ignore non-reactive desorption effects. The monomer-dimer surface reaction is shown schematically as

\[
\begin{align*}
A + E & \xrightarrow{y_A} A(ads), \\
B_2 + 2E & \xrightarrow{y_B} 2B(ads), \\
A(ads) + B(ads) & \xrightarrow{k} AB + 2E.
\end{align*}
\]
The exact rate equations of the homogeneous system are

\[ \frac{d\theta_A}{dt} = y_A \theta_E - 4k\theta_{AB} \]  \hspace{1cm} (12)

\[ \frac{d\theta_B}{dt} = 2y_B \theta_{EE} - 4k\theta_{AB} \]

In this model, the exact rate equations do not easily give information into the nature of the reaction. There is one calculation that is possible, and that is to look at the homogeneous steady state and find a relationship between \( \theta_E \) and \( \theta_{EE} \):

\[ \frac{d\theta_A}{dt} = 0 = y_A \theta_E - 4k\theta_{AB} \]
\[ - \frac{d\theta_B}{dt} = 0 = 2y_B \theta_{EE} - 4k\theta_{AB} \]

\[ 0 = y_A \theta_E - 2y_B \theta_{EE} \]

This shows that in a reactive steady state,

\[ \theta_{EE} = \frac{y_A}{2y_B} \theta_E, \]

in contrast to the mean-field theory which assumes \( \theta_{EE} = (\theta_E)^2 \).

We follow the same strategy as with the monomer-monomer model, that is we start by analyzing the homogeneous steady state using a mean-field theory. With this assumption the rate equations are

\[ \frac{d\theta_A}{dt} = y_A \theta_E - 4k\theta_A \theta_B \]
\[ \frac{d\theta_B}{dt} = 2y_B \theta_E^2 - 4k\theta_A \theta_B \]

We have the solution of these equations in the general case (see equation (3)). By setting \( k = 1 \) for example one can sketch the coverage of \( A \) for the steady state vs. \( y_A \) (see Fig. 5). There is a region of bistability, where there are two stable steady states separated by one unsteady stable state.
In contrast, the lattice gas model always supports a unique stable steady state. The lattice gas model has a second-order transition from a reactive steady state to a B-poisoned state as $y_A$ becomes less than $y_1$ (see Fig. 6). It also has a first-order transition to an A-poisoned state as $y_A$ becomes more than $y_2$. For the diffusionless case, values of $y_1$ and $y_2$ have been obtained for $k = \infty$ [16,17] and for $k > 0$ [17]; for the $k = 0^+$ case the model becomes the voter model with $y_A = \frac{y_B}{4}$ [18].

By adding surface diffusion to these models, we observe changes in their behavior, although they are small for low levels of surface mobility. The behavior of the lattice gas models vary with the degree of mobility, and approach a mean-field behavior in the limit of extreme high diffusion.

**Thesis Organization**

This thesis is organized into 3 papers showing different approaches to mathematical modeling of surface reactions. Two different reactions are discussed.

In Paper 1 we present a detailed analysis of the monomer-monomer surface reaction model without diffusion and without nonreactive desorption. We use a lattice gas approach. We show that for $y_A = y_B = 1/2$ there are no non-trivial reactive steady states, and that slow poisoning occurs due to a clustering of like reactants. This is done through direct comparison with the voter model, which is known to have no non-trivial reactive steady states. This part is a reprint of an article which appeared in *Physical Review E, Volume 47, Number 2, pages 1018-1025*. It is reproduced here with the kind permission of the American Physical Society.

Paper 2 is a lattice gas analysis of the monomer-dimer surface reaction model for both zero and nonzero surface diffusion (but still without nonreactive desorption). We investigate the behavior of the survival probability of a non-poisoned patch embedded in a poisoned surface at $A$ impingement rates just below the $A$-poisoning transition. This part is an article that will be submitted to a journal.

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This is the so-called ZGB model, named for the authors.
Paper 3 is a mean-field theory approach to nucleation of the monomer-dimer surface reaction model in the regime of high diffusion. We investigate critical sizes for nucleation at various reaction rates and at several A-impingement rates, extrapolating the critical radii to $k = \infty$. We also relate the velocity of planar interfaces to the critical radii, and comment on the connection between the mean-field approach to critical size and the lattice gas approach.

Following Paper 3 we sum up the results with General Conclusions.
Figure 5. Plot of the homogeneous steady states for the $AB_2$ surface reaction model - mean-field approach.
Figure 6. Plot of the homogeneous steady states for the $AB_2$ surface reaction model - lattice gas approach.
PAPER 1

KINETICS OF THE MONOMER-MONOMER SURFACE REACTION MODEL
KINETICS OF THE MONOMER-MONOMER SURFACE REACTION MODEL

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ABSTRACT

The two-dimensional monomer-monomer \((AB)\) surface reaction model without diffusion is considered for infinitesimal, finite and infinite reaction rates \(k\). For equal reactant adsorption rates, in all cases, simulations reveal the same form of slow poisoning, associated with clustering of reactants. This behavior is also the same as that found in simulations of the two-dimensional voter model studied in Interacting Particle Systems theory. The voter model can also be obtained from the dimer-dimer or monomer-dimer surface reaction model with infinitesimal reaction rate. We provide a detailed elucidation of the slow poisoning kinetics via an analytic treatment for the \(k=0^+\) \(AB\) reaction and the voter model. This analysis is extended to incorporate the effects of place-exchange diffusion which slows, but does not prevent poisoning. We also show that the \(k=0^+\) \(AB\) reaction with no diffusion is equivalent to the voter model with diffusion at rate 1/2. Identical behavior of the monomer-monomer reaction and the voter model is also found in an "epidemic" analysis, where one considers the evolution of a surface poisoned by one species, except for a small patch. Finally we apply our findings to elucidate the behavior of the monomer-dimer surface reaction model for small reaction rates.
I. INTRODUCTION

In this contribution, we shall consider the monomer-monomer (or AB) surface reaction model,

\begin{align}
A(g) + * &\rightarrow A(ad), \quad B(g) + * \rightarrow B(ad), \\
A(ad) + B(ad) &\rightarrow AB(g) + 2 * ,
\end{align}

on a square lattice of adsorption sites. Here 'g' and 'ad' denote gas-phase and adsorbed species, and * denotes an empty surface site. Adsorbed species are immobile unless otherwise stated. Adsorption rates, \( p_A \) and \( p_B \), for \( A \) and \( B \), respectively, are normalized so that \( p_A + p_B = 1 \), and the reaction rate is denoted by \( k \). We shall consider the reaction-limited regime with infinitesimal reaction rate, \( k=0^+ \), the regime of finite reaction rates, \( 0<k<\infty \), and the adsorption-limited regime with infinite reaction rate, \( k=\infty \).

The basic behavior of these models follows from inspection of the appropriate exact rate equations for the evolution of concentrations or coverages of \( A(ad) \) and \( B(ad) \) with time, \( t \). Here we let \([A]\) and \([B]\) denote the concentration (i.e., coverage) of \( A(ad) \) and \( B(ad) \), \([E]=1-[A]-[B]\) denote the concentration of empty sites, \([AB]\) denote the probability of finding an adjacent \( A(ad)B(ad) \)-pair as distinct from a \( B(ad)A(ad) \)-pair, \( A' \) denote a site not filled by \( A \) and \([A'] = 1-[A] \) the corresponding probability, etc.. The presence of correlations implies that \([AB] \neq [A][B], [EE] \neq [E]^2 \), etc.. For a square lattice, one immediately obtains for \( 0<k<\infty \),

\[
\frac{d[A]}{dt} = p_A[E] - 4k[AB], \quad \frac{d[B]}{dt} = p_B[E] - 4k[AB],
\]

so

\[
\frac{d}{dt} ([A] - [B]) = (p_A - p_B)[E].
\]

For \( k=\infty \), clearly one has \([AB]=0 \). Here it is convenient to let
\[ D(A) = p_A \begin{bmatrix} B & A' \\ E & B \\ B & A' \end{bmatrix}, \quad D(B) = p_B \begin{bmatrix} A' \\ E & A' \end{bmatrix} \]

denote non-reactive adsorption or deposition rates for \( A \) and \( B \), respectively. The quantities in the square parentheses denote the probabilities of finding an empty site with none of the neighboring sites occupied by \( B \)'s for \( D(A) \), or by \( A \)'s for \( D(B) \). Then \( T(J) = p_j[E] \) and \( R(J) = T(J) - D(J) \) denote total and reactive adsorption rates for \( J = A \) or \( B \), respectively. Thus, for \( k = \infty \), one has

\[
\frac{d[A]}{dt} = D(A) - R(B), \quad \frac{d[B]}{dt} = D(B) - R(A),
\]

so

\[
\frac{d}{dt} ([A] - [B]) = (p_A - p_B)[E], \tag{3}
\]

the last result being identical to (2). Finally let \( t = kt/(1+k) \) and consider the limit \( k = 0^+ \) where one obtains\(^1\)

\[
\frac{d[A]}{d\tau} = -\frac{d[B]}{d\tau} = 4(p_A - p_B)[AB]. \tag{4}
\]

In all cases, it is immediately clear that if \( p_A \neq p_B \), then the only steady state is a trivial "adsorbing" poisoned state with \([E] = [AB] = 0\) and \([A] = 1\) or \([B] = 1\). Note that adding diffusion to these models does not change rate equations (2-4) for the species concentrations, so the same conclusion applies. Although it does not rigorously follow from (2-4) alone, one might expect that poisoning by the species with the larger pressure occurs exponentially with rate proportional to \((p_A - p_B)^{-1}\). This claim is supported by simulations\(^2\).

Henceforth, our attention naturally focusses on the case where \( p_A = p_B = 1/2 \), and where (2-4) provide no direct information about the steady state. In an important early simulation study of the reaction-limited regime, \( k = 0^+ \), Wicke et al\(^3\)
noted a propensity for reactant clustering or segregation, and gave a simple argument for this propensity. A detailed study of analogous reactant segregation for the adsorption-limited regime, $k=\infty$, was given by Ziff and Fichthorn. Although an reactive steady state appears to form in these systems, more detailed studies revealed that reactant clusters continue to slowly grow or coarsen, while the reaction rate correspondingly slowly decreases to zero. We discuss this behavior in detail below. It is however appropriate to note here that ben-Avraham et al. argue that this slow poisoning is driven by concentration fluctuations of a diffusive nature. They also show that the mean poisoning time for these models, on a finite (two-dimensional) lattice of $N$ sites, increases roughly linearly with $N$. (The precise form is like $N \ln(N)$.) This contrasts the behavior of processes with a true reactive steady state (on an infinite lattice), where the mean poisoning time increases exponentially with $N$.

A primary focus of this contribution is to compare the above behavior for the monomer-monomer reaction with that of the voter model studied extensively in Interacting Particle Systems theory. In the voter model, sites have two states or "opinions", $A$ or $B$, say. Each site waits an exponential time, with parameter one, say, at which time it changes its opinion to that which it sees on a randomly chosen neighboring site. Another realization of this model is as a dimer-dimer surface reaction with infinitesimal reaction rate: imagine a surface completely covered with $A$ and $B$; the reaction $A(\text{ad}) + B(\text{ad}) \rightarrow AB(\text{g}) + 2^*$ occurs at a randomly chosen $A(\text{ad})B(\text{ad})$-pair, and the resulting empty pair is immediately filled with an $A_2$-dimer, $A_2(\text{g}) + 2^* \rightarrow 2A(\text{ad})$, or a $B_2$-dimer, $B_2(\text{g}) + 2^* \rightarrow 2B(\text{ad})$, with equal probability. The feature that the voter model has only trivial "poisoned" steady states in $d \leq 2$ dimensions (i.e., consensus is eventually reached) has been established rigorously. This was achieved by relating the voter model to an auxiliary problem of coalescing random walks, and then using the recurrence property of random walks. The lack of existence of non-trivial steady states indicates that clustering must occur. We emphasize however that the kinetics of clustering, e.g., the time decay of the
concentration, $[AB]$, of neighbors with opposite opinions, apparently has not been well characterized to date. We shall comment further on the properties of the voter model below.

In Sec.II, we present simulation results characterizing and comparing the slow poisoning kinetics for the monomer-monomer reaction and the voter model (without diffusion). Analytic elucidation of this behavior is presented for the $k=0+$ monomer-monomer reaction and for the voter model. In Sec.III, we consider the evolution of a surface which is initially covered or poisoned by one species, $B$ say, except for a small patch. We then explore how the probability that the system is not completely poisoned by $B$ varies (decreases) with time. This type of question is familiar in theories of “critical epidemics”[11], and has been considered previously within the context of surface reaction models[5,12]. The effect of adding diffusion to the $k=0+$ monomer-monomer reaction and the voter model is considered analytically in Sec.IV. We apply the findings of Sec.III to elucidate the behavior of the monomer-dimer surface reaction for low reaction rates[5,13,14,15] in Sec.V, and provide some concluding remarks in Sec.VI.
II. POISONING KINETICS

We first present results for poisoning kinetics for the monomer-monomer reaction with $p_A=p_B=1/2$ and for voter model (without diffusion). We start from an initially empty lattice for $k>0$, and from a random distribution with $[A]=[B]=1/2$ for $k=0+$. In order to present a unified description, we examine the behavior of the concentration, $[AA']=[AB]$, of adjacent pairs of sites, one filled with $A$ (ad) and the other not. Thus one has $[AA']=[AB]$ when $k=0+$, $[AB]+[AE]$ when $0<k<\infty$, $[AE]$ when $k=\infty$. A unified and well-defined time-scale is provided by $\tau=kt/(1+k)$ for $0+<k<\infty$.

Simulations were performed involving about 40 trials on a 200x200 lattice up to $T=10^3$. A typical time per trial on a Silicon Graphics machine is 3 hours. To test the relationship $[AA']\sim\tau^\omega$, as $\tau\to\infty$, we plot $\log_{10}([AA'])$ against $\log_{10}(\tau)$ in Fig.1. From the data for $O(10^2)\leq\tau\leq O(10^3)$, we find effective exponent values of $\omega=0.04$ to $0.08$ for the monomer-monomer reaction with various $k$ (Table I), and a voter model value of $\omega=0.096$. These values are consistent with previous estimates for special cases[2,5]. It has also been proposed[2] that $[AA']\sim\log_{10}(\tau)^{-\sigma}$, as $\tau\to\infty$, so in Fig.2 we plot $\log_{10}([AA'])$ against $\log_{10}(\log_{10}(\tau))$. We thus find for $O(10^2)\leq\tau\leq O(10^3)$, effective exponent values of $\sigma=0.25-0.5$ for the monomer-monomer reaction (Table I), and $\sigma=0.59$ for the voter model.

Thus from these simulation studies, it is reasonable to conclude that the monomer-monomer reaction model for various $k$, and the voter model, exhibit fundamentally the same behavior. However the precise form of the asymptotic decay is unclear. Fortunately further elucidation is possible via analytic treatment for the $k=0+$ monomer-monomer reaction and for the voter model. It is, of course, possible to write down an exact hierarchy of rate equations for various subconfiguration probabilities either directly[1] (accounting for all possible ways of creating and destroying the subconfiguration), or by first mapping the process onto a spin-system[8,16]. The latter approach has been applied only to the $k=0+$ monomer-monomer problem treating $A$'s ($B$'s) as spin +1 (-1), but it can also be applied to the
Fig. 1  Simulated poisoning kinetics for the monomer-monomer reaction model, for various $k$ shown in the legend, and for the voter model. Plotted is the logarithm of the concentration of $AA'$-pairs versus $\log_{10}(\tau)$; slopes give the exponent $-\omega$. 
Fig. 2  Simulated poisoning kinetics for the monomer-monomer reaction model, for various $k$ shown in the legend, and for the voter model. Plotted is the logarithm of the concentration of $AA^\prime$-pairs versus $\log_{10}(\log_{10}(\tau))$; slopes give the exponent $-\sigma$. 
Table I. Effective exponents describing poisoning kinetics for the voter model and for the monomer-monomer (AB) reaction model for various k.

<table>
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<tr>
<th>Model Type</th>
<th>$\frac{k}{1+k}$</th>
<th>$\omega$</th>
<th>$\sigma$</th>
</tr>
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<tr>
<td>Voter</td>
<td>0+</td>
<td>0.096</td>
<td>0.59</td>
</tr>
<tr>
<td>$AB k=0+$</td>
<td>0+</td>
<td>0.078</td>
<td>0.51</td>
</tr>
<tr>
<td>$AB k=1/7$</td>
<td>1/8</td>
<td>0.071</td>
<td>0.38</td>
</tr>
<tr>
<td>$AB k=1/5$</td>
<td>1/6</td>
<td>0.083</td>
<td>0.47</td>
</tr>
<tr>
<td>$AB k=1/3$</td>
<td>1/4</td>
<td>0.083</td>
<td>0.46</td>
</tr>
<tr>
<td>$AB k=3/5$</td>
<td>3/8</td>
<td>0.070</td>
<td>0.33</td>
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<td>0.052</td>
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<tr>
<td>$AB k=3$</td>
<td>3/4</td>
<td>0.058</td>
<td>0.31</td>
</tr>
<tr>
<td>$AB k=\infty$</td>
<td>1</td>
<td>0.043</td>
<td>0.26</td>
</tr>
</tbody>
</table>
voter model. Previous studies have focussed on the case of equal adsorption rates, $p_A=p_B=1/2$. The key observation here was that the single-site probabilities satisfy a closed set of equations, as do the pair probabilities, except for coupling back to the single site quantities\cite{8,16}. In fact n-point probabilities couple only to themselves and to (n-1)-point probabilities. Just as for the Glauber model\cite{17}, the randomly hopping lattice-gas\cite{18}, or the equilibrium single-step model\cite{19}, it is this feature that facilitates analytic treatment of the model. We consider first the translationally invariant $k=0+$ monomer-monomer reaction (without diffusion) on a square lattice, and directly develop a set of equations for the probabilities of $A$-filled pairs of sites (cf. Ref.\cite{1}). One finds that

$$\frac{d}{dt}[AA] = 2p_A^2[AB] + 2p_A[AAB] + 2p_B[A][B]$$

where, as previously, square parentheses indicate probabilities or "concentrations" of the configurations enclosed. Here the first two gain terms describe the transformation $AB \rightarrow AA$ for a specific pair of sites under consideration: in the first term, the $A$ and $B$ in the pair under consideration react and are replaced by deposition of two $A$'s; in the second term, the $B$ reacts with an $A$ other than the one in the pair under consideration, so deposition of just one $A$ is required to form an $AA$-pair on the pair of sites under consideration. The last loss term describes the transformation $AA \rightarrow AB$ for a specific pair of sites under consideration by reaction of the right $A$ with an adjacent $B$ and subsequent replacement by a $B$. The 2's are symmetry numbers. Similarly, for a separated pair of $A$'s, one has
\[
\frac{d}{d\tau} [A --- A] = 2p_A \left( [A --- AB] + [A --- BA] + [A --- B] \right) - 2p_B \left( [A --- BA] + [A --- A] + [A --- AB] \right) + \left( \begin{array}{c} A \\ B \end{array} \right) + \left( \begin{array}{c} B \\ A \end{array} \right) + \left( \begin{array}{c} A \\ A \end{array} \right) + \left( \begin{array}{c} A \\ B \end{array} \right)
\]

Henceforth we consider only the case \( p_A = p_B = 1/2 \) which facilitates fundamental reduction of (5) and (6). First it is necessary to use conservation of probability relationships to convert all configurations appearing in (5) and (6) to ones which involve only \( A \)'s. Thus, for example, we use the identities \( [AB] = [A] - [AA] \), \( [ABA] = [A - A] - [AAA] \), \( [AAB] = [AA] - [AAA] \), \( [A-AB] = [A - A] - [A - AA] \), etc.

Substitution into (5) and (6) shows that all three-site probabilities cancel out in the special case where \( p_A = p_B \) (but in no other case). Before writing out the resulting equations, it is convenient to introduce a more compact notation. Thus we let \( P_{ij} \) denote the concentration of pairs of \( A \)'s separated by \((i,j)\), so by symmetry \( P_{i,j} = P_{j,i} \) and \( P_{i,j} = P_{j,i} \). Let denote the discrete Laplace operator, \( P_{ij} = P_{i+1,j} + P_{i-1,j} + P_{i,j+1} + P_{i,j-1} - 4P_{ij} \). Then after the above mentioned cancellation for \( p_A = p_B \) (5) and (6) become

\[
\begin{align*}
\frac{d}{d\tau} P_{10} &= P_{20} + P_{11} + P_{-11} - (3 + \chi)P_{10} + \chi[A], \\
\frac{d}{d\tau} P_{ij} &= \Delta P_{ij}, \text{ for } |i| + |j| \geq 2,
\end{align*}
\]

with \( \chi = 1/2 \). These equations are consistent with Ref.[8], where the focus was on analysis of the increase of the total correlation, and subsequent estimation of the poisoning time for finite systems. Here instead we focus on the poisoning kinetics, and specifically the concentration, \([AB]\), of adjacent \( AB = AA' \) pairs which gives the reaction rate. Thus we let \( Q_{ij} \) denote the concentration of \( AB \)-pairs of separation
\( (i, j) \), i.e., \( Q_{ij} \) gives the probability of finding an \( A \) at \((0,0)\) and a \( B \) at \((i, j)\). Then one has \( Q_{ij} = [A] - P_{ij} \) and \( Q_{10} = [AB] \). For an initially random distribution of \( A \)'s and \( B \)'s, from (7) it is easy to see that \( Q_{ij} = [A](1-[A])S_{ij} = [B](1-[B])S_{ij} \), where the \( S_{ij} \) are independent of \( A \) and satisfy

\[
\frac{d}{d\tau} S_{ij} = S_{20} + S_{11} - S_{-11} - (3 + \chi) S_{10},
\]
\[
\frac{d}{d\tau} S_{ij} = \Delta S_{ij}, \text{ for } |i| + |j| \geq 2.
\]

The random initial condition corresponds to \( S_{ij} = 1 \), for all \( i \) and \( j \).

The above analysis can be repeated for the voter model. The key difference is that \( p_A^2 \) is replaced by \( p_A \) in the first term of (5). Consequently, one finds that equation (8) still applies but with \( \chi = 1 \).

Numerical solution of these equations is possible after truncation, e.g., by setting \( S_{ij} = 1 \), for \( |i| \) and \( |j| \) sufficiently large. (It is easy to check during integration of (8) that errors introduced by such truncation are insignificant over the time range considered; we truncate at \(|i|, |j| > 350\) to determine \( S_{ij} \) for \( \tau < 8000 \).) This allows determination of the behavior of \([AA'] = [AB] = [A](1-[A])S_{10} \) consistent with, but more precise than, above simulation estimates. Results from integration of (8) for the effective exponents

\[
\omega(\tau) = \frac{d \log_{10}([AB])}{d \log_{10}(\tau)}
\]
and

\[
\sigma(\tau) = \frac{d \log_{10}([AB])}{d \log_{10}(\log_{10}(\tau))}
\]
are shown in Fig.3 for both models. It appears that \( \sigma(\tau) \) increases monotonically, approaching a value between 0.8 and 1, as \( \tau \to \infty \) (a limit of unity is certainly consistent with our data), but this behavior is achieved very slowly. If \( \sigma(\tau) \) approaches any finite value, as \( \tau \to \infty \), then one must conclude that \( \omega(\tau) \to 0 \), as \( \tau \to \infty \). This is also consistent with our data. It is also clear that it is practically impossible to determine the true asymptotic behavior of the poisoning kinetics via simulation.
In Sec.IV, we show that for the one-dimensional versions of these models, rate equations analogous to (8) can be solved completely via standard techniques\cite{18,20} to recover, e.g., the well known result $[AA'] \sim t^{1/2}$.

Finally we mention a relevant previous study. For the voter model, it has been shown\cite{10,21} that the (probability distribution for) the side length, $l$, of the largest square containing just one species, and including the origin, scales like $\log_{10}(t)$. One might then expect that $[AA']$ should scale like $\log_{10}(\tau)^{-1}$. However if the growing clusters have an “active zone” with $O(1)$ defect density which scales like $\mathcal{L}$, then $[AA']$ would scale like $\log(\tau)^{\mathcal{L}-1}$. [See Ref.\cite{22} for a discussion of similar issues applied to multi-Eden-cluster growth.] Thus our results for $\sigma(\tau)$ in Fig.3 above can be used to extract values for an effective $\nu(\tau)=1-\sigma(\tau)$. Our data is consistent with the expectation that $\nu(\tau) \leq 1/2$ for large clusters, and allow that $\nu(\tau)$ may vanish, as $\tau \to \infty$. 
Fig. 3 Analytic estimates of the $\tau$ dependence of effective exponents, $\omega(\tau)$ and $\sigma(\tau)$, describing poisoning kinetics. Results for both the voter model and the $k=0+$ monomer-monomer ($AB$) reaction model are shown.
III. EPIDEMIC ANALYSIS

Here we present results of an "epidemic analysis" for the monomer-monomer reaction with $p_A=p_B=1/2$ and for the voter model (without diffusion). In such an analysis, the system is initially completely covered or poisoned by species $B$, say, except for a small patch. We determine the behavior of the "survival" probability, $P_g$, that the system is not completely poisoned, as a function of time. This type of analysis is commonly used to determine the critical behavior of models with non-equilibrium phase transitions to adsorbing or poisoned states\cite{5,11,12}. In such studies, right at the transition, one finds that $P_g \to t^{-\delta}$, where $\delta$ is a non-trivial exponent and provides information on the universality class. For the voter model, however, if one exploits the connection with coalescing random walks\cite{10,23}, it follows that $P_g \sim \log \left( \frac{t}{\ln t} \right)$, so one should find $\delta=1$.

In our simulations, we somewhat arbitrarily start with a $B$-covered lattice except for an empty pair of sites for $k>0$ monomer-monomer reaction models, or an $A$-filled pair if $k=0+$ and for the voter model. Results are obtained from $O(10^4)$ trials. Fig. 4 shows our simulation results for $\log_{10}(P_g)$ versus $\log_{10}(t)$, and corresponding $\delta$-values are shown in Table II. These $\delta$-values are typically slightly below unity, but certainly consistent with a true asymptotic value of $\delta=1$. (Any logarithmic correction of the form suggested above would produce lower effective values.) In Table II, we have also given slopes obtained from $\log_{10}(P_g)$ versus $\log_{10}(t)$, and versus $\log_{10}(t/\ln t)$, for the $k=0+$ monomer-monomer reaction and voter model, and versus $\log_{10}(t/\ln t)$, for $k>0$ monomer-monomer reaction models. These slopes are also consistent with a true asymptotic value of $\delta=1$. From these results, it is reasonable to assert that the monomer-monomer reaction model for various $k$, and the voter model, exhibit fundamentally the same behavior characterized by $\delta=1$. 
Fig. 4 Simulation results for the decay of the survival probability, $P_s$, with reduced time, $\tau$, for epidemic analyses of the monomer-monomer reaction model, for various $k$ shown in the legend, and for the voter model. Slopes of these $\log_{10}(P_s)$ versus $\log_{10}(\tau)$ plots give the exponent $-\delta$. 
Table II. Effective exponents describing the decay of the survival probability $P_s$ for the voter model and for epidemic analyses of the monomer-monomer (AB) reaction model for various $k$. We have fit to $P_s \sim t^{-\delta}$, and $\log_{10}(P_s) \sim \hat{\delta}\log_{10}(t)$, for $k > 0$, or $\log_{10}(P_s) \sim \hat{\delta}\log_{10}(\tau)$, for voter and $AB$ $k=0+$ models.

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<th>Model Type</th>
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<th>$\delta$</th>
<th>(\hat{\delta})</th>
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<td>1.08</td>
</tr>
<tr>
<td>$AB$ $k=0+$</td>
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</table>
IV. MODEL EXTENSIONS: DIFFUSION

It should be noted that various natural extensions of these monomer-monomer reaction models (with $p_A=p_B$) might be considered. If one introduces desorption in a finite system of N sites, one observes a transition from bistability to monostability as the desorption rate increases above $O(N^{-1})$. This has been demonstrated analytically for infinitesimal reaction rate using appropriate rate equations of the form (5-8) or the corresponding spin representation\cite{16,26}.

One can also introduce diffusion, the randomizing effect of which works against the clustering propensity of the reaction. This competition has been studied to date only for one case of finite reaction rate\cite{27}. Here we consider the $k=0+$ monomer-monomer ($AB$) reaction and the voter model with diffusion corresponding to random $A$-$B$ place exchange at rate $h=h_{AB}$ or $h_v$ (on a time scale $\tau$), respectively. Recall that here the surface is completely covered with $A$ and $B$. Again analytic treatment is possible by simply augmenting (7) or (8) with the appropriate random hopping terms. See Ref.\cite{18} for a detailed discussion of these terms. Specifically, one adds a term $2h(S_{20}+S_{11}+3S_{10})$ to $(d/d\tau) S_{10}$, and $2hS_{ij}$ to $(d/d\tau) S_{ij}$ for $|i|+|j|\geq2$. Setting $\tau'=\tau(1+2h)$ and $\varepsilon=h/(1+2h)$ (so $0<\varepsilon\leq1$ for the $k=0+$ $AB$ model, and $0<\varepsilon\leq1$ for the voter model), one obtains

$$\frac{d}{d\tau'} S_{10} = (S_{20}+S_{11}+3S_{10}) - \varepsilon S_{10} ,$$

$$\frac{d}{d\tau'} S_{jj} = \Delta S_{jj} , \text{ for } |i|+|j|\geq2 .$$

(9)

It thus becomes apparent from (9) that the $k=0+$ $AB$ model with $h_{AB} \geq 0$ is equivalent to the voter model with $h_v=(1+4h_{AB})/2 \geq 1/2$ at the level of the pair probabilities (after a simple rescaling of time). The same equivalence is also found for $n$-point probabilities with $n \geq 3$. In particular, the $k=0+$ $AB$ model with no diffusion is equivalent to the voter model with $h_v=1/2$.

This last result can be rationalized directly by considering the possible
transitions from an $AB$ pair of sites. For the diffusionless $k=0^+$ $AB$ model, it is clear that the state of these sites can change to one of $AA$, $BA$ or $BB$ with equal probabilities of $1/4$ (or remain as $AB$ with probability $1/4$). This is not the case for the diffusionless voter model where $AB$ changes to either $AA$ or $BB$ with equal probability. However introducing exchange diffusion at rate $h_v=1/2$ in the voter model guarantees that one of $AA$, $BA$ or $BB$ is again chosen with equal probability.

The evolution equations (9), which are of the same form as (8), have a simple interpretation in terms of random walks. Consider a particle undergoing a random walk between neighboring sites $(i,j)
eq(0,0)$ on a square lattice with a hop rate of unity, and with the additional possibility of irreversible adsorption at $(0,0)$ from neighboring sites with "small" rate $\varepsilon$. Then $S_{ij}$ represents the probability of finding the particle at site $(i,j)$. The recurrence property for two-dimensional random walks suggests that even for our unnormalized initial conditions, one will find that $S_{ij}\to0$, as $\tau'\to\infty$, no matter how small $\varepsilon$. Thus we conclude that for any $b>0$, the process still poisons, i.e., there is no reactive steady state. Fig.5 shows numerical evidence to support this claim. Specifically, the effective exponent

$$
\sigma(\tau') = d \log_{10}(|AB|) / d \log_{10}(\log_{10}(\tau'))
$$

is shown to increase monotonically as $\tau'\to\infty$, for various $h\geq0$, leading to the conclusion that $\sigma(\infty)>0$ which confirms that poisoning must occur.

Finally, it is instructive to consider the one-dimensional versions of these $k=0^+$ monomer-monomer reaction and voter models with diffusion. Let $Q_i=[A](1-[A])S_i$ denote the concentration of $AB$-pairs of separation $i$. (Thus $Q_i$ gives the probability of finding an $A$ at the origin, and a $B$ at site $i$.) Then it is easy to show that (9) is replaced by

$$
\frac{d}{d\tau}S_i = (S_2 - S_i) - \varepsilon S_i, \quad \frac{d}{d\tau}S_i = \Delta S_i, \text{ for } i \geq 2,
$$

(10)
Fig. 5 Analytic estimates of the $\tau'$-dependence of the effective exponent, $\sigma(\tau')$, describing the poisoning kinetics of the $k=0+$ $AB$ model with place-exchange diffusion for various rates $h \geq 0$, shown.
where $\tau'$ and $\varepsilon$ are exactly as above, but now $S_i = S_{i+1} - 2S_i + S_{i-1}$. The random initial condition corresponds to $S_i = 1$, for all $i$. From a spectral decomposition of the evolution operator associated with (10), together with appropriate treatment of the unnormalized initial conditions, we obtain the solution

$$S_i(\tau') = \pi^{-1} \int_0^\pi d\phi \exp[-4\sin(\frac{\phi}{2})^2 \tau'] \frac{\sin(i\phi + \eta) \cos(\frac{\phi}{2} + \eta)}{\sin(\frac{\phi}{2})}. \quad (11)$$

Here the real-valued "phase shift" $\eta = \eta(\phi)$ satisfies

$$e^{2i\eta} = \frac{[1 + (\varepsilon - 1)e^{-i\phi}]/[1 + (\varepsilon - 1)e^{i\phi}].}$$

Note that the diffusionless voter model corresponds to the particularly simple case where $\varepsilon = 1$, so $\eta = 0$. An asymptotic analysis of (11) yields

$$S_i \sim \pi^{-1/2}[i + (1 - \varepsilon)e^{-1}]/(\tau')^{1/2}, \text{ as } \tau' \to \infty. \quad (12)$$

Thus the reaction rate, which is determined by $[AB] = [A](1-[A])S_1$, always vanishes like $(\tau')^{-1/2}$, and like $\varepsilon^{-1}(\tau')^{-1/2}$ in the regime of large diffusion rates, $h$, or small $\varepsilon = \chi/(1 + 2h)$.
V. CONNECTION WITH THE MONOMER-DIMER REACTION

We now briefly comment on certain aspects of the behavior of the monomer-dimer surface reaction

\[ A(g) + * \rightarrow A(ad), \ B_1(g) + 2* \rightarrow 2B(ad), \ A(ad) + B(ad) \rightarrow AB(g) + 2* \]  

on a square lattice. Again, adsorption rates, \( p_A \) and \( p_B \), for \( A \) and \( B \), satisfy \( p_A + p_B = 1 \), and \( k \) denotes the reaction rate. Adsorbed species are immobile. Thus one obtains the rate equations

\[
\frac{d[A]}{dt} = p_A[E] - 4k[AB], \\
\frac{d[B]}{dt} = 2p_B[EE] - 4k[AB].
\]

For \( k > 0 \), the distribution of adsorbed species can potentially adjust to achieve a non-trivial steady state for a finite range of \( p_1 \leq p_A \leq p_2 \). In this steady state, one has \( p_A[E] = 2p_B[EE] \). On the other hand, for \( k = 0^+ \), this process reduces to the voter model for \( p_A = 1/5 \): once an \( AB \)-pair reacts, the empty pair formed is immediately filled by two \( B \)'s via \( B_2 \)-deposition, or sequentially by two \( A \)'s. (This is precisely the same behavior as in the above mentioned dimer-dimer reaction.) One can show that equal effective adsorption rates for \( A \) and \( B \) corresponds to \( p_A = 1/5 \); for \( p_A > 1/5 \) (\( p_A < 1/5 \)), the system "quickly" poisons with \( A \) (\( B \)). Thus the range, \( \delta p_A = p_2 - p_1 \), supporting a reactive steady state, must clearly shrink to zero as \( k \rightarrow 0^+ \) (\( p_1 = p_2 = 1/5 \) and \( \delta p_A = 0 \) at \( k = 0^+ \), compared with \( p_1 = 0.390, p_2 = 0.525 \) and \( \delta p_A = 0.135 \) at \( k = \infty \)). An interesting conjecture has been made suggesting that \( \delta p_A = 0 \) for \( 0 \leq k \leq k_c \), where the tricritical point is located at \( k_c = 0.08 \). This claim, which has been disputed recently, is reconsidered here.

Suppose that \( k_c > 0 \). One might expect that for \( k < k_c \) when \( p_A = p_1 = p_2 \), since any slight perturbation of the relative adsorption rates would cause rapid poisoning, the
system should exhibit voter or monomer-monomer reaction model type behavior. If true, then an epidemic analysis for an empty patch in an $A$-poisoned background at $P_A = P_2$ should yield the voter model value of $\delta = 1$, in contrast to the much higher values found previously\cite{5} for the monomer-dimer reaction with $k \geq 1/10$. We have performed such an analysis for $k = 1/20$ (well below the predicted value of $k^c$ above) at the appropriate $P_2 = 0.2576$. (This $P_2$-value was determined by varying $P_A$ until $\log_{10}(P_s)$ was found to decay asymptotically linearly with $\log_{10}(t)$; $\log_{10}(P_s)$ saturates for $P_A < P_2$, and decreases faster than linearly for $P_A > P_2$.) One finds a distinct crossover from voter model behavior with $\delta = 1.0$ for shorter times, to "finite-\(k\)" behavior with $\delta = 1.6$ for larger times (Fig.6). This suggests that there is no tricritical point $k^c > 0$ (or at least that $k_c < 1/20$). This analysis relies on the surprising observation that for the monomer-dimer model with $k > 0$, $\lim_{k \to 0} \delta(k) = 1.5$ differs from the voter model value of $\delta = 1$. 
Fig. 6 Simulation results for the decay of the survival probability, $P_s$, starting with a 2x1 empty patch in an A-poisoned background for the monomer-dimer ($AB_2$) reaction model with $k=1/20$ and $p_A=p_2=0.2576$. The slope of $\log_{10}(P_s)$ versus $\log_{10}(t)$ gives $-\delta$. The inset shows the variation of $\delta$ with $k$ for the monomer-dimer model, as compared with $\delta$ for the voter model.
VI. CONCLUSIONS

In this study, we have shown that the behavior of the diffusionless monomer-
monomer surface reaction model, with equal reactant adsorption rates and various
reaction rates, is fundamentally the same as that of the voter model. We also
conclude from analytic calculations that the slow poisoning kinetics exhibited by the
\( k=0+ \) monomer-monomer reaction and the voter model is most appropriately
described by the form \([AA'] \rightarrow \log(\tau)^{-\sigma}\), with \( \sigma \) close to (and possibly equal to) unity.
We emphasize that it is practically impossible to determine this true asymptotic
behavior from simulations. Introduction of place-exchange diffusion to the \( k=0+ \)
monomer-monomer reaction and voter models does not change their basic poisoning
behavior. It does however elucidate the relationship between them. In this
contribution, it is also shown that the survival probability in an “epidemic analysis”
of the diffusionless monomer-monomer reaction model decays like \( P_\sigma \sim \tau^{-1} \), with
possible logarithmic corrections, again consistent with voter model behavior. Our
understanding of the monomer-monomer reaction and voter models is useful for
elucidating the behavior of the monomer-dimer reaction model with low reaction
rate, and can also be applied to elucidate the behavior of a simplistic \( A+BC \) model
for the CO+NO reaction\(^{[28]}\).
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REFERENCES

[25] A reaction probability, \( r = 2k(1+2k)^{-1} \), is introduced in Ref.s 14 and 15.
NUCLEATION THEORY
FOR FIRST-ORDER POISONING TRANSITIONS
NUCLEATION THEORY FOR FIRST-ORDER POISONING TRANSITIONS

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ABSTRACT

We develop a nucleation theory to describe some aspects of non-equilibrium first-order transitions from "reactive" steady states to trivial adsorbing states. Specifically, we consider the CO-poisoning transition in the ZGB-model for CO-oxidation, and in generalizations of this model which include adspecies diffusion. The behavior of the "epidemic" survival or growth probability, $P_g$, for a non-poisoned patch embedded in a poisoned background is determined below the poisoning transition. We show how the characteristic or "critical" patch size increases, and the smooth increase of $P_g$ with size sharpens, as the transition is approached, or with the increase in diffusion. This behavior is elucidated through analysis of the propagation velocity of an interface separating reactive and poisoned phases, and of appropriate reaction-diffusion equations for the regime of high diffusion.
I. INTRODUCTION

The monomer-dimer surface reaction model involves adsorption of a monomer species (A) on single empty sites (E), adsorption of a dimer species (B₂) on adjacent pairs of empty sites, and reaction of different species adsorbed on adjacent sites [1,2]. The impingement rates for A and B₂ are denoted by \( y_A \) and \( y_B \), respectively, and are normalized so that \( y_A + y_B = 1 \). Below \( \theta_A \) and \( \theta_B \) will denote coverages of adsorbed species. The special case of instantaneous reaction in the absence of surface diffusion is referred to as the ZGB model [1]. Here we consider this model on a square lattice, and its generalization [3,4,5] to include adspecies diffusion. Specifically we allow hops to nearest-neighbor unoccupied sites at rate \( h \) for each possible direction, and for both A and B. Finally we note that these models mimic CO-oxidation, A corresponding to CO, and B₂ to O₂.

Of primary interest here is the feature that these models exhibit a first-order transition from a reactive steady state to an A-poisoned state at \( y_A = y_2 \), say [1-5]. See the schematic in Fig.1. The location of this transition shifts monotonically from \( y_2 = 0.5256 \) for no diffusion [6], to \( y_2 = 0.5951 \) in the limit of very rapid diffusion [5]. Our goal in this contribution is to elucidate the "nucleation and growth" of the reactive steady-state from a near A-poisoned state, for \( y_A \) "slightly below" the transition point, \( y_2 \). Traditionally such a description would be applied only for a metastable range \( y_s < y_A < y_2 \), where \( y_s \) denotes a lower spinodal. We just note here that the determination of \( y_s \) is problematic, and we defer further discussion to Sec.II below.

One is naturally lead to consider the "epidemic problem [7-9]" of determining the survival probability, \( P_s(t) \), of a patch of \( N \) empty sites embedded in an A-poisoned background [7]. Since a reactive state, rather than the A-poisoned state, is the stable steady state for \( y_A < y_2 \), it follows that there is a non-zero probability that the non-poisoned patch will survive indefinitely spreading the reactive steady state across the surface. From the theory of epidemics, one expects that \( P_s(t) \) has the scaling form, \( P_s(t) \sim t^{-2\phi(\Delta t^{1/\nu})} \), where \( \Delta = y_2 - y_A \) denotes the distance from the
Fig. 1 Schematic diagram of coverage of A ($\theta_A$) for a-typical $AB_2$-surface reaction. For $y_{s^-} \leq y_A \leq y_2$ and for $y_2 \leq y_A \leq y_{s^+}$ nucleation and growth are possible. The solid line corresponds to the reactive stable state.
transition, and $\phi(x) - x^{v\delta}$, as $x \to \infty$. Here $\delta$ and $v$ are non-trivial scaling exponents [7-9]. Consequently, the asymptotic survival probability has the form $P_s(\infty) \sim A^{v\delta}$, as $A \to 0$. Henceforth, we shall consider only this asymptotic probability which will be denoted by $P_s$ for brevity. One could also consider the asymptotic expansion velocity of a surviving patch, $V_p \sim A^{\alpha}$, where $\alpha = v(2-\eta-\delta)/2$ and $\eta$ is another epidemic exponent. Note that $V_p$ also corresponds to the velocity of a planar interface separating the reactive state from the $A$-poisoned state which it displaces.

In the context of the nucleation theory [10], one is particularly interested in the dependence of $P_s$ on the initial empty patch size, $N$. Clearly $P_s$ will increase monotonically with $N$, approaching unity as $N \to \infty$. Thus, for any $A$, we can define the characteristic size, $N^*$, of the initial empty patch as that for which there is an equal chance of growth or extinction, i.e., $P_s = 1/2$. One might also interpret $N^*$ as a critical size, at least in situations where $P_s(N)$ increases suddenly from near zero to near unity as $N$ increases through $N^*$. Of basic interest is the dependence of $N^*$ on model parameters such as $A$ and $b$. We characterize this scaling, together with the "sharpening" of $P_s$ as a function of $N/N^*$, as $A \to 0$ and as $b \to \infty$.

To elucidate the above behavior in the presence of surface diffusion, $b > 0$, it is especially useful to consider directly the regime of rapid diffusion via the appropriate "mean-field" reaction-diffusion equations [5,11,12]. The effective mixing caused by rapid diffusion validates these equations. Importantly, the lifetime of homogeneous metastable states (for fixed $A$) should diverge as $h \to \infty$, in order to recover the bistability of the mean-field theory. Correspondingly, $N^*$ should also diverge as $h \to \infty$ (for fixed $A$). This result can be extracted directly from the reaction-diffusion equations. It is also clear that fluctuations become less significant as one approaches the $h \to \infty$ deterministic limit, which explains the sharpening of $P_s$ mentioned above.

For these models, one could also consider "nucleation and growth" of the $A$-poisoned state from the metastable reactive state for $y_A$ "slightly" above the

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1The exponent $\eta$ characterizes the evolution of the average number of empty sites, $N_e(t) \sim t^\eta$, in the epidemic patch at the transition $\Delta = 0$. 
transition point. Specifically, this metastable state exists for $y_2 < y_A < y_{s+}$, where $y_{s+}$ denotes the upper spinodal (see Sec.II). Here, however, it is not possible to simply and precisely define the survival probability for an A-poisoned patch embedded in a metastable reactive background. [Can one be sure it is the embedded patch that is growing, as opposed to some nearby spontaneously nucleated A-poisoned patch? How does one determine extinction of the patch given that it can be spontaneously born?] These ambiguities also occur for nucleation of the reactive phase from a near A-poisoned metastable state when $y_A < y_2$ in models with non-reactive A-desorption (see below). They are also present in any conventional nucleation theory for non-zero temperatures. On the other hand, identification of the large critical sized clusters will be straightforward in practice since the process behaves semi-deterministically [6]. This is certainly true for rapid diffusion, where again a mean-field reaction-diffusion analysis elucidates nucleation behavior. We do not discuss this case further here.
II. SPINODAL POINTS

We begin with a brief discussion of the upper spinodal, $y_{s+}$. It may be defined via analytic extension of the reactive steady state above $y_2$ as the point where slopes of coverages with respect to $y_A$ become infinite \[^{[7]}\]. See Fig.1. As a practical matter, $y_{s+}$ can be determined from analysis of scaling of the poisoning kinetics for $y_A$ somewhat above $y_{s+}$. Specifically, one fits the kinetics to the form $\theta_A-K_+[(y_A-y_{s+})t]$, regarding $y_{s+}$ as a free parameter. (The quality of the fit is very sensitive to the choice of this parameter.) Alternatively $y_{s+}$ can be determined from a “constant-coverage (CC)” ensemble analysis \[^{[6]}\] (see Appendix). We find that: $y_{s+}=0.527-0.529$ compared to $y_2=0.5256$ for $b=0$ (Refs. \[^{[5]}\]); $y_{s+}=0.564-0.565$ compared to $y_2=0.5522$ for $b=3/8$; and $y_{s+}=2/3$ compared to $y_2=0.5951$ for $b=\infty$ (Ref. \[^{[5]}\]). Thus the range of metastability, $y_{s+}-y_2$, increases dramatically from 0.002-0.004 when $b=0$, to 0.012 when $b=3/8$, to 0.072 when $b=\infty$.

As indicated above, determination of the location of the lower spinodal\(^2\), $y_{s-}$, is more problematic since even its definition is unclear in the above models for $h<\infty$. One approach is to introduce into the model non-reactive desorption of species A with (small) rate $d>0$, then the stable steady state for high $y_A$ becomes non-trivial \[^{[2,6,7b,13]}\]. Again, see Fig.1. This would allow precise definition of $y_{s-}(d>0)$ via analytic extension analogous to that of $y_{s+}$ above. In principal, the value of $y_{s-}$ for the original desorptionless model could then be obtained from $y_{s-}(d)$, as $d\rightarrow 0$, although one would not expect this procedure to be practical. Thus as an alternative, we explore whether the kinetics of evolution from a near-A-poisoned state, for $y_A$ somewhat below $y_{s-}$, can be described by a scaled form $\theta_A-K_+[(y_A-y_{s-})t]$, regarding $y_{s-}$ as a free parameter. Indeed we find that this is the case and,

\(^2\)These models also exhibit a continuous transition from a B-poisoned state to a reactive steady state at $y_A=y_1$, say; $y_1$ has a value of 0.391 when $h=0$, and decreases quickly to zero as $h$ increases to about 3. Presumably $y_{s-}$ must be above $y_1$. 
importantly, that the quality of the fit is very sensitive to the choice of the parameter.

In these studies of kinetics, an 800x800 lattice is initially randomly filled with A to $\theta_A = 0.97$. The objective here is to create a near-A-poisoned surface, with the constraint that the initial state must contain a substantial number of empty pairs to avoid large fluctuations in the kinetics. (Most isolated empty sites will be "quickly" filled with A, causing $\theta_A$ to initially increase much closer to unity.) This leads to the following results: $y_{sc} = 0.495 - 0.50$ for $h = 0$ (see Fig. 2); $y_{sc} = 0.44$ for $h = 3/8$ (see Fig. 3). These results reflect a general trend which fortunately ameliorates to some extent difficulties with the determination of $y_{sc}$: as $h$ increases, $y_{sc}$ should decrease to zero (the value predicted by mean-field theory [5]), thus producing a broad well-defined range of metastability. One can also check the sensitivity of these estimates to the initial choice of $\theta_A$: for $h = 0$ with initial $\theta_A = 0.95$, one estimates $y_{sc} = 0.50$ consistent with the above (perhaps shifted slightly higher corresponding to the lower initial $\theta_A$).

As a final aside, it is appropriate to note that in an approximate "dynamic cluster" treatment of these models [2,14], $y_{sc}$ corresponds to a transcritical bifurcation [15], but its position depends on the order of the approximation. It should also be mentioned that in dynamic cluster treatments of the modified model with non-reactive A-desorption ($d > 0$), $y_{sc}$ is converted to a saddle-node bifurcation [15]. Of course, $y_{sc+}$ corresponds to a saddle-node bifurcation for all $d > 0$. 
Fig. 2. Diffusionless AB$_2$ lattice gas model. Top plot is the Coverage of A ($\theta_A$) vs $t$ for varying $y_A$. Bottom plot shows $\theta_A$ vs $(0.495-y_A)t$, giving $y_s=0.495$ due to the sensitivity of the relationship $\theta_A-K_s[(y_s-y_A)t]$. 
Fig. 3 AB₂ lattice gas model with \( h=3/8 \). Top plot is the Coverage of A (\( \theta_A \)) vs \( t \) for varying \( y_A \). Bottom plot shows \( \theta_A \) vs \( (0.44-y_A)t \), giving \( y_s=0.44 \) due to the sensitivity of the relationship \( \theta_A - K_B [y_s - y_A]t \).
III. SCALING THEORY FOR THE SURVIVAL PROBABILITY, $P_s$

One fundamental quantification of the nucleation process in these models is provided by the scaling of the characteristic size, $N^* \rightarrow \Delta^* \phi$, with the distance from the transition, $\Delta = y_A - y_A^*$. Here $\phi$ is a non-trivial scaling exponent (whose definition has been changed from Ref. [7a]). This behavior can be directly extracted from $P_s$ versus $N$ data, for various $\Delta$, from the relationship $P_s(N^*) = 1/2$. A more effective alternative determination of $\phi$ exploits a "constant-coverage (CC)" ensemble [6]. Here one runs a simulation on an $L \times L$ lattice attempting to deposit $A$'s or $B$'s as is appropriate to maintain some fixed high value of $\theta_A$. The stable steady state of this ensemble consists of a single "characteristic" reactive cluster of $N^* \approx L^2(1-\theta_A)$ non-$A$ sites, the impingement rate $y_A$ (or equivalently $\Delta$) being automatically selected to ensure equal probability of growth or extinction of this cluster (in the usual ensemble). See Appendix I.

For a more complete analysis, one is naturally interested in elucidating the dependence of $P_s$ on $N/N^*$, for various $\Delta$, including the $\Delta \rightarrow 0$ limit. Two regimes apparently need to be considered separately: (i) the "asymptotic regime" $N/N^* \ll 1$ typically analyzed from the perspective of epidemic theory; (ii) the "nucleation regime" $N/N^* = O(1)$ as is appropriate for elucidating the sharpness of a critical size.

For regime (i), we propose that

$$P_s \rightarrow F(N/N^*) \quad \text{where} \quad F(x) \approx x^{\phi/\delta}, \quad \text{as} \quad x \rightarrow 0.$$  \hspace{1cm} (1)

This small-$x$ behavior of $F(x)$ is required for consistency with the $P_s$-scaling relations for small $\Delta$, described in the Introduction. As a consequence, one has that $P_s \approx N^\delta/\phi \Delta^{-\delta}$ for $N \ll N^*$. This allows independent estimation of the exponent $\phi$, assuming that $\delta$ is known from the small $\Delta$ dependence of $P_s$, for fixed $N$ (see Ref. [7a]). Our main interest is in the nucleation regime (ii), where we propose that

$$P_s \approx G[(N/N^* - 1)/\sigma(N^*)],$$  \hspace{1cm} (2)
where G[0]=1/2, G[x]→0 as x→∞, and G[x]→1 as x→∞. Here σ measures the range of N/N* values over which P_s makes the transition from zero to unity, i.e., δN*−σN* measures the uncertainty in the critical size. Clearly, the smaller σ, the more N* reflects a sharp critical size above (below) which survival (extinction) is almost certain.

A primary goal is to understand the behavior of σ. Writing R* = \sqrt{N*} for the critical radius, then one has σ = δN*/N* − 2δR*/R*. Now if the fluctuations in the radial dimension of epidemic clusters are of order ξ lattice spacings, then the uncertainty, δR*, in the “critical radius” should be of the order of the minimum of ξ and R*, so σ = min[ξ, ξ/R*]. Thus the challenge is to characterize \( \xi \). We propose that \( \xi^2 = \xi_i^2 + \xi_{KPZ}^2 \), where \( \xi_i \) denotes the “intrinsic fluctuation” independent of R*, and \( \xi_{KPZ} \) corresponds to long-wavelength fluctuations. In Sec. V, we argue that the latter are described by the KPZ-equation which implies that \( \xi_{KPZ} (R^*)^{1/3} \), as \( R^* → ∞ \). Consequently one expects the asymptotic behavior, \( \sigma (R^*)^{-2/3} (N^*)^{-1/3} → 0 \), as \( N^* → ∞ \), producing a sharpening of P_s.

A note of caution is required in applying the above results, especially in the absence of diffusion. For \( b = 0 \) one finds large intrinsic fluctuations, \( \xi_i \), of at least 20 lattice spacings. This implies that \( σ \) is effectively fixed at least for \( Δ ≥ 0.005 \). Indeed it has been noted previously [7] that there is no sharp critical size in the diffusionless ZGB model, and that the above scaling relation with constant \( σ \) reasonably describes the model behavior for \( Δ ≥ 0.005 \). Ultimately one has \( σ → 0 \), as \( Δ → 0 \) and \( N^* → ∞ \), which implies a “sharpening” of P_s. This becomes visible for \( Δ = 0.003 \).

We are also interested in the behavior of P_s versus N for fixed Δ, as \( h → ∞ \). We shall see below that R* ∼ h^{1/2} and N* ∼ h, as \( h → ∞ \), reflecting an increase in metastable lifetime as one approaches mean-field bistability. Thus in applying (2), it remains to characterize the dependence of \( σ \) on \( h \). Studies below indicate that \( \xi_i \) decreases quickly with increasing \( h \), making it easier to see the sharpening of P_s with increasing \( h \). This is limited by the long-wavelength fluctuations and quantified by \( σ (R^*)^{-2/3} h^{-1/3} \) (which is substantiated by the simulations below).
IV. SIMULATION RESULTS FOR ZERO AND FINITE DIFFUSION RATES

The first step in analysis of nucleation behavior in these models is to determine the dependence of $y_2$ on the diffusion rate $b$. However direct simulation of the poisoning kinetics can lead to overestimation of $y_2$ since the process tends to "get stuck" in the reactive metastable state for $y_A$ slightly above $y_2$. (See Ref. [5] for a discussion of the results of Refs. [3,4].) These difficulties can be circumvented using either an epidemic analysis (Ref. [11]) or a "constant-coverage (CC) ensemble" analysis (see Ref. [6] and the Appendix) to determine $y_2$. Our results from the CC analysis are presented in Table I. The simulations were performed involving about 200 trials on a $100\times100$ lattice up to $\tau=O(10^3)$. Typical run times per 10 trials varied from 1 to 3 hours on a Silicon Graphics computer as we went from low to high diffusion. The results were verified on an 8-CPU Cray Y-MP C-90 computer using a $1024\times1024$ lattice with about $10^3$ trials up to $\tau=O(10^4)$ attempts (which took 5+ hours using 11.6% of the computer).

Next, using an epidemic analysis, values were found for $\delta$ and $\eta$, as previously defined. In our simulations, we start with an A-covered lattice except for an empty pair of sites. We set the A impingement rate $y_A$ at the transition rate $y_2$ and measure the asymptotic slope of the survival probability (for $\delta$) and of the number of empty sites (for $\eta$). Survival probability (and separately, number of empty sites) is plotted as a function of time in Fig. 4. Results use $O(10^7)$ trials. Computed "effective" values of $\delta$ and $\eta$ for $\tau=20$ are displayed in Table I, and are shown with best fit curves in Fig. 5. Effective values appear to be asymptotically approaching $\delta-\eta\approx 19.3-20.0$ as $h\to\infty$ but true asymptotic values are difficult to ascertain. This gives $v$ the asymptotic value of $\alpha$ in the extreme high diffusion case (since $\alpha=v(1-\delta-\eta)/2$). Gaining additional data to determine more accurate asymptotic behavior is not currently possible, requiring at least $O(10^8)$ trials.

Using the CC approach, we analyze the relationship between critical sizes and
Fig. 4 Epidemic simulations for the calculation of $\delta$ and $\eta$ for $h>0$. The top plot shows $P_s$ vs. $t$, which is used to calculate $\delta$. The bottom plot is of $N_e$ vs. $t$, which is used to calculate $\eta$. More precision is currently impractical due to the large number of trials needed to handle the small $P_s$ and $N_e$ values.

$^3$The values of $\delta$ and $\eta$ for $h=0$ were previously calculated$^{[7b]}$. 
Table I. Dependence of $y_2$ on diffusion $h$ for the $AB_2$ reaction model for infinite reaction rate. Also in the table is the probability of diffusion used in previous results [9] (given by $P_d = \frac{4h}{1+4h}$), and $\delta$ and $\eta$ estimates. Error for $y_2$ values is $\pm 10^{-4}$.

<table>
<thead>
<tr>
<th>$h$</th>
<th>$P_d$</th>
<th>$y_2$</th>
<th>$\delta$</th>
<th>$\eta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>0.0</td>
<td>0.5256</td>
<td>3.7</td>
<td>-2.4</td>
</tr>
<tr>
<td>0.375</td>
<td>0.6</td>
<td>0.5522</td>
<td>11.8</td>
<td>-14.3</td>
</tr>
<tr>
<td>1.0</td>
<td>0.8</td>
<td>0.5645</td>
<td>16.2</td>
<td>-16.2</td>
</tr>
<tr>
<td>2.25</td>
<td>0.9</td>
<td>0.5739</td>
<td>17.9</td>
<td>-17.9</td>
</tr>
<tr>
<td>4.0</td>
<td>0.941</td>
<td>0.5794</td>
<td>18.9</td>
<td>-18.9</td>
</tr>
</tbody>
</table>
Fig. 5 "Effective" $\delta$ and $\eta$ simulation results for various diffusion probabilities $P_d$ (related to $h$ as shown above). Values were calculated from epidemic analyses at the transition point $y_2$ and for $t=20$. Solid lines show best linear fit for all points and dashed lines show best linear fit for the final three points, both converging to $-20$ ($-20$ for $\eta$) in the extreme high diffusion case.
the pressure or impingement rate of \( A \) for different diffusion rates \( h \). Using \( L \times L \) lattices, we fix the coverage \( \theta_A \) (thus fix the average number of \( A \)' sites \( \bar{n}_A \)) and measure the average \( A \)-impingement rate \( \bar{y}_A \). Results are shown in Table II. Fig. 6 indicates that \( \bar{n}_A \sim \Delta^{-\phi} \) as \( \Delta \to 0 \). The corresponding values of \( \phi \) are also in Table II and are plotted as a function of both \( h \) and \( P_d \) in Fig. 7. While there is no clear trend, the \( \phi \) vs. \( P_d \) plot appears consistent with the fact that \( \phi \to 2 \) as \( P_d \to 1 \) (\( h \to \infty \)).

Using an epidemic analysis we obtain survival probabilities as a function of the initial patch size for different values of \( y_A \) below the A-poisoning transition \( y_2 \), and for various values of \( h \geq 0 \). Results of \( P_s \) vs. \( N \) for \( h=0 \) are displayed in Fig. 8. In Fig. 9 we scale the patch size by \( 1/N^* \) (where \( N^* \) corresponds to \( P_s(N)=1/2 \)). As expected, we observe weak sharpening when this diffusionless epidemic data is scaled. We adopt the same scaling scheme for \( h=3/8 \) and for \( h=4 \) (Figs. 10 and 11)\(^5\). Again as expected, we observe sharpening as \( y_A \to y_2 \) (as \( \Delta \to 0 \)), and the sharpening becomes more pronounced as \( h \) increases. We can observe the sharpening behavior as a function of \( h \) by holding \( y_A/y_2 \) fixed and increasing \( h \). Fig. 12 shows this for \( y_A/y_2=0.951 \) and in Fig. 13 we see even more pronounced sharpening with \( y_A/y_2=0.98 \).

As an additional calculation, values of \( \sigma \) were computed for the two highest diffusion rates on the constant \( y_A/y_2 \) data using the horizontal distance between \( N \) when \( P_s(N)=.5 \) (our \( N^* \) value) and when \( P_s(N)=.2 \). For \( y_A/y_2=.951 \), we found that \( \sigma \sim h^{-31} \), and for \( y_A/y_2=.98 \), we found that \( \sigma \sim h^{-38} \), both consistent with the theoretical limit \( \sigma \sim h^{-1/3} \).

Using the \( N^* \) values for the epidemic model, we can come up with another estimate of \( \phi \). Again, using the three \( N^* \) values with lowest \( \Delta \), we obtain values for \( \phi \) and post them in Table III.

\(^4\) \( A' \) is used to denote 'not \( A \)' sites, i.e. sites which either contain a \( B \) or are empty.

\(^5\) Tables of \( N \) and \( N^* \) values for the epidemic model are in Appendix II.
Table II. ZGB model, CC approach used to measure average values of $y_A$ and $n_A$. In the case of high $n_A$, $n_A$ $\Delta \phi$ (see Fig. 6).

<table>
<thead>
<tr>
<th>$h$</th>
<th>$L^2 \times \theta_{A'} = \bar{y}_A$</th>
<th>$\bar{n}_A$</th>
<th>$\phi$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>128$^2 \times 0.2 = 3276.8$</td>
<td>128$^2 \times 0.1 = 1638.4$</td>
<td>$64^2 \times 0.1 = 409.6$</td>
</tr>
<tr>
<td></td>
<td>.5242</td>
<td>.5232</td>
<td>.5191</td>
</tr>
<tr>
<td>0.375</td>
<td>$100^2 \times 0.2 = 2000$</td>
<td>$64^2 \times 0.2 = 819.2$</td>
<td>$64^2 \times 0.08 = 327.7$</td>
</tr>
<tr>
<td></td>
<td>.5483</td>
<td>.5455</td>
<td>.5395</td>
</tr>
<tr>
<td></td>
<td>$32^2 \times 0.049 = 50.2$</td>
<td>$32^2 \times 0.03 = 30.7$</td>
<td>.4893</td>
</tr>
<tr>
<td>1.0</td>
<td>$164^2 \times 0.15 = 4034.4$</td>
<td>$100^2 \times 0.25 = 2500$</td>
<td>$64^2 \times 0.1 = 409.6$</td>
</tr>
<tr>
<td></td>
<td>.5606</td>
<td>.5592</td>
<td>.5470</td>
</tr>
<tr>
<td>2.25</td>
<td>$164^2 \times 0.15 = 4034.4$</td>
<td>$100^2 \times 0.25 = 2500$</td>
<td>$64^2 \times 0.108 = 442.4$</td>
</tr>
<tr>
<td></td>
<td>.5672</td>
<td>.5652</td>
<td>.5467</td>
</tr>
<tr>
<td>4.0</td>
<td>$164^2 \times 0.26 = 6993.0$</td>
<td>$100^2 \times 0.255 = 2550$</td>
<td>$100^2 \times 0.2 = 2000$</td>
</tr>
<tr>
<td></td>
<td>.5728</td>
<td>.5675</td>
<td>.5658</td>
</tr>
<tr>
<td></td>
<td>$64^2 \times 0.025 = 102.4$</td>
<td>$32^2 \times 0.03 = 30.7$</td>
<td>.3513</td>
</tr>
</tbody>
</table>
Fig. 6 Plot of $\bar{n}_A$ vs. $\Delta$ for the ZGB model using CC analysis (Log-Log plot). The asymptotic slope (generally – last 3 points) gives the value of $\phi$, shown in Table II.
Fig. 7 Dependence of $\phi$ on diffusion rate. Top plot is of $\phi$ vs $h$, and bottom plot is of $\phi$ vs $P_d$. The trend as $h \to \infty$ ($P_d \to 1$) is not obvious, but appears consistent with the theoretical limit of $\phi \to 2$ as $h \to \infty$. 
Fig. 8  Plot of $P_s$ vs. N for the diffusionless ZGB epidemic model.
Fig. 9 Plot of $P_s$ vs. $N/N^*$ for the diffusionless ZGB epidemic model, showing weak sharpening as $y_A \to y_2$. 
Fig. 10 Plot of $P_s$ vs. $N/N^*$ for the ZGB epidemic model with $h=3/8$ ($P_d=.6$). We observe sharpening as $y_A \rightarrow y_2$. 
Fig. 11 Plot of $P_s$ vs. $N/N^*$ for the ZGB epidemic model with $h=4$ ($P_d=.941$). We observe sharpening as $y_A \to y_2$, more pronounced than with $h=3/8$. 
Fig. 12 Plot of $P_s$ vs. $N/N^*$ for the ZGB epidemic model with $y_A/y_C = 0.951$. We observe sharpening as $h$ increases.
Fig. 13 Plot of $P_s$ vs. $N/N^*$ for the ZGB epidemic model with $y_A/y_C = 0.98$. We observe sharpening with increasing $h$. The sharpening is more pronounced than when $y_A/y_C = 0.951$. 
Table III. ZGB model for diffusion rate $h \geq 0$, epidemic approach used to compute $\phi$ values for different values of $h$. The fourth column is from the CC analysis (previously shown in Table II).

<table>
<thead>
<tr>
<th>$h$</th>
<th>$P_d$</th>
<th>$\phi$</th>
<th>$\phi_{(CC)}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>0.0</td>
<td>1.3</td>
<td>1.33</td>
</tr>
<tr>
<td>0.375</td>
<td>0.6</td>
<td>1.6</td>
<td>1.46</td>
</tr>
<tr>
<td>1.0</td>
<td>0.8</td>
<td>1.7</td>
<td>1.56</td>
</tr>
<tr>
<td>4.0</td>
<td>0.941</td>
<td>1.9</td>
<td>1.72</td>
</tr>
</tbody>
</table>
Finally, we use the CC data to calculate the value of $N^*$ for varying $h$ at fixed $y_A/y_2$ (see Table IV), and plot the $N^*$ values as a function of $h$ in Fig. 14. This shows that $N^* \sim h$, and that as $y_A \to y_2$ ($y_A/y_2 \to 1$) the slope of this linear relationship appears to grow without bound, agreeing with the theory. The asymptotic slopes are also listed in Table IV.

Table IV. ZGB model for diffusion rate $h \geq 0$, CC approach used to compute $N^*$ values for different values of $h$, and for different values of $y_A/y_2$. Data is plotted in Fig. 14 showing $N^* \sim h$. *Note: When $h=0$, $y_A/y_2=0.90$ corresponds to $y_A=0.90 \times 0.5256 = 0.4730$ which is below the lower spinodal $y_s=0.495$.

<table>
<thead>
<tr>
<th>$y_A/y_2$</th>
<th>0</th>
<th>3/8</th>
<th>1</th>
<th>2 1/4</th>
<th>4</th>
<th>Slopes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.90</td>
<td>*</td>
<td>57.9</td>
<td>101.6</td>
<td>187.8</td>
<td>309.1</td>
<td>69.3</td>
</tr>
<tr>
<td>0.95</td>
<td>68.5</td>
<td>124.7</td>
<td>198.6</td>
<td>416.0</td>
<td>688.1</td>
<td>155.5</td>
</tr>
<tr>
<td>0.97</td>
<td>131.8</td>
<td>237.1</td>
<td>430.5</td>
<td>886.2</td>
<td>1395.</td>
<td>290.8</td>
</tr>
<tr>
<td>0.98</td>
<td>221.4</td>
<td>400.3</td>
<td>795.4</td>
<td>1641.</td>
<td>2669.</td>
<td>587.3</td>
</tr>
</tbody>
</table>
Fig. 14 Plot of $N^*$ vs. $h$ for the ZGB CC model with $y_A/y_2 = .90, .95, .97$ and .98. This shows that asymptotically $N^* \approx h$. 
V. INTERFACE PROPAGATION AND NUCLEATION BEHAVIOR

Planar Interfaces

To elucidate the nucleation behavior described above in Sec. IV, it is appropriate to first consider the propagation of a planar interface separating the reactive and A-poisoned phases for $\gamma_A$ below the transition $\gamma_2$. Since the reactive phase is the stable phase, it will displace the A-poisoned phase corresponding to $V_p > 0$. We wish to determine the dependence of the planar interface velocity, $V_p$, on the model parameters. This can in part be achieved by identifying for our model the characteristic time scales and length scales (here measured in units of the lattice spacing), and then applying a dimensional analysis.

It is instructive to couch our discussion in terms of a more general model which includes reaction between nearby (rather than just nearest-neighbor) species according to some "contact distribution" the range of which has variance $\sigma^2$. Reaction rates need not be infinite. We also include adspecies diffusion at rate $b$, as in the specific model described above. Further, we assume that the influence of the surface reaction is described by some pseudo-first-order rate constant, $k$. This would just correspond to the adsorption rate in the case of instantaneous reaction, or more generally be determined by the rate limiting step. Thus the characteristic time scale is given by $\tau_c = k^{-1}$. Both the square of the diffusion length, $\xi_{\text{diff}}^2 = b\tau_c$, and the variance of the contact or reaction range, $\sigma^2$, provide contributions to the square of the characteristic length scale, $\xi_c^2$. Then combining dimensional arguments with the general form of the dependence of $V_p$ on $\Delta$ mentioned in Sec.I, one writes

$$V_p \sim (\xi_c / \tau_c) \Delta^\alpha.$$  \hspace{1cm} (3)

Thus in the absence of diffusion one has $V_p \sim k \sigma \Delta^\alpha$, and in the regime of high diffusion one obtains $V_p \sim (b \cdot k)^{1/2} \Delta^\alpha$, with $\alpha = 1$ (see below). In the specific model analyzed in the above sections, one has $k = 1$ and $\sigma = 1$.

A more detailed foundation for these results might be provided as follows. A
coarse-grained description of models without diffusion maps onto "spatial contact models" traditionally used to describe ecological and epidemic spread [16]: the reaction terms are described by spatial convolution sums or integrals. Indeed the corresponding expression given above for $V_p$ is familiar in that context. The regime of high diffusion rates is even simpler to analyze as it can be described by mean-field reaction-diffusion equations [11,12]. The stated result for $V_p$ with $\alpha=1$ follows directly from an analysis of such equations. In an intermediate regime where $\xi_{\text{diff}}$ and $\sigma$ are comparable, the coarse-grained equations would include both contact (spatial convolution) and diffusion terms. Applying a so-called "diffusion approximation" to the contact terms reduces them to the same form as the conventional Laplacian diffusion terms with $k\sigma^2$ playing the role of the diffusion constant $h$. This observation motivates the above statement that $\xi_{c,2}$ includes diffusive and contact contributions, although the relationship is more complicated than a simple sum. Furthermore, one cannot conclude that $V_p$ should increase monotonically with some linear combination of these contributions.

From simulation studies of interface propagation in the principal axis direction [10], we find that $V_p \rightarrow \Delta \alpha$ where $\alpha=0.85$ for $h=0^6$ (See Fig. 15), and $\alpha=0.90$ for $h=3/8$, showing the trend towards $\alpha=1$, as $h \rightarrow \infty$. We note that for fixed $\Delta$, $V_p$ decreases in magnitude as $h$ first increases from zero. This decrease may be associated with the initial decrease in "intrinsic fluctuations" and thus in the roughness and reactivity of the interface. It should however be emphasized that eventually $V_p$ must increase like $h^{1/2}$. It is also instructive to determine whether $V_p$ depends on the mean orientation of the propagating interface. Any anisotropy, which is associated with the underlying square lattice, should be strongest in the absence of diffusion. Thus, for $h=0$, we examine propagation in the [11] direction (at 45° to the above direction): we find no detectable anisotropy to within the statistical uncertainty (1/2 %) of our measurement. We expect that, just as in the simple Eden

6This $\alpha$-estimate for $h=0$ differs from our previous less accurate estimate (footnote 1) which was extracted from epidemic exponents.
**Fig. 15** Interface propagation velocity, $V_p$ vs. $\Delta$ for $h=0$ (Log-Log plot). Slope of best fit line gives $\alpha=0.85$. 
growth model [17], anisotropy exists, but is very weak.

Further insight into this process comes from consideration of the stochastic evolution equation describing the propagation of an on-average planar interface normal to a principal axis, $i$, say, and with location $i=h(j)$ (reactive phase to the left, and $A$-poisoned phase to the right). In a coarse-grained description, $V=\partial h/\partial t$ satisfies a KPZ-type equation [18] of the form $V=V_p +\lambda <\nabla_j h^2> + \nabla_j^2 h + ...$, so $V_p=<V>=V_p +\lambda <\nabla_j h^2> + ...=V_p$. Here odd powers of gradients are absent by symmetry. Above we have shown above that the propagation velocity normal to the interface is independent of orientation. Thus it follows that for small $\nabla_j h$ and neglecting curvature, the propagation velocity projected in the $i$-direction satisfies $V=V_p [1+(\nabla_j h)^2]^{1/2}=V_p +\lambda /2 V_p (\nabla_j h)^2$, corresponding to $\lambda >0$. A similar analysis applies to the much simpler Eden growth model [17,19]. Finally, the Laplacian term with "kinetic surface tension" $\nu>0$ reflects the slower propagation of concave indentations into the $A$-poisoned phase (edge $A$'s are less exposed) and faster propagation of convex protrusions of $A$-poisoned phase into the reactive phase. For $\lambda >0$, this Laplacian term also provides needed regularity to the solutions of the evolution equation [20].

We conclude from this analysis that the propagating interface will "quickly" equilibrate on a local length scale. In fact $V_p =<V>$ should approach its asymptotic value like $t^{-2/3}$ (see Ref. [20]). However it will roughen on a global length scale as described by the KPZ-universality class. Thus for system of linear size $L$, the fluctuations in the interface width at time $t$ scale like $[18] \xi_{KPZ} = L^{1/2} f(t/L^{3/2})$, where $f(x) \sim x^{1/2}$, as $x \rightarrow 0$. For a circular geometry, one sets $t=L-R$ and obtains $\xi_{KPZ} = R^{1/3}$, the result mentioned in Sec.III. Finally, we note that right at the transition, $y_A = y_2$, the $A$-poisoned and reactive phases are in equilibrium so $V_p =<V>=0$ for all orientations, i.e., there is no dependence on $\nabla_j h$ in the evolution equation. Thus this interface, as for any non-driven interface, should exhibit conventional Edwards-Wilkinson-type "equilibrium" roughening [18], as is shown in Fig. 16.
Fig. 16 Interface profiles between equistable reactive and poisoned states for different diffusion rates $h$. Fluctuations in the interface are seen to decrease with increasing $h$, while spatial coarsening increases with increasing $h$. 
Curved Interfaces

In order to connect these ideas with nucleation theory, it is necessary to extend our consideration to the growth velocity of curved interfaces, \( V = V(R) \), where \( R \) denotes the local mean radius of curvature. We shall always select the locally convex (concave) region as the stable reactive (A-poisoned) phase, and displacement of the A-poisoned phase by the reactive phase corresponds to \( <V> > 0 \). Clearly one has \( <V(R)> \rightarrow V_p \), as \( R \rightarrow \infty \). One also expects that the introduction of curvature \( R < \infty \) will retard the propagation of the interface: microscopically, A-species in the poisoned region are less exposed to reaction than for a planar interface. This is consistent with the presence of the Laplacian kinetic surface tension term in the evolution equation described above. Thus we propose

\[
V_p <V(R)> - (\xi_c^2/\tau_c) R^{-1}, \text{ for } R > \xi_c.
\]

In the regime of high diffusion, this expression reduces to \( V_p <V(R)> \approx h/R \), which can be confirmed directly from an analysis of the appropriate reaction-diffusion equations in a cylindrical coordinate system.

Given (4), the behavior of the characteristic radius, \( R^* \), is clearly determined by the condition, \( <V(R^*)> = 0 \), which implies that \( R^* \approx \xi_c^2/(\tau_c V_p) \). Then using (3) for \( V_p \) and the basic relation, \( N^* = R^{*2} \), one obtains

\[
N^* \approx \xi_c^2 \Delta^{-2\alpha}.
\]

We thus relate the exponents for the \( \Delta \)-dependence of the critical size and of the planar interface velocity, \( \phi = 2\alpha \). This in turn relates \( \phi \) to standard epidemic exponents (see Ref. 7a). From above we see that: \( 2\alpha = 1.7 \) versus \( \phi = 1.3 \) for \( h = 0 \); \( 2\alpha = 1.8 \) versus \( \phi = 1.5 \) for \( h = 3/2; \ldots; 2\alpha = \phi = 2 \) for \( h \rightarrow \infty \). Thus the trend is certainly consistent, although numerical agreement is poor (perhaps due to imprecision in (5) and in numerical \( \alpha \) estimates). In the regime of high diffusion, one obtains the reaction-

\footnote{This revises the previous asymptotic limit for \( \delta \) to 1.}
diffusion result, $N^* - h\Delta^{-2}$, determining the scaling of $N^*$ with both $h$ and $\Delta$ ($\alpha=1$ so $\phi=2$).
VI. CONCLUSIONS

Here we have provided the first detailed analysis of nucleation phenomena for non-equilibrium first-order transitions to adsorbing states. Since the steady states of the reaction models considered here cannot be characterized in terms of free energy minimization, the standard framework of analysis is inapplicable. As an alternative we combine ideas from the critical theory of epidemics and from kinetic roughening to interpret the behavior seen in simulations. Our analysis applies both to cases where interface dynamics driven by local reaction rules (in the absence of diffusion), and where dynamics driven by mean-field reaction-diffusion equations (the scaling of which is independent of the details of the reaction mechanism).

We find non-trivial model-parameter-dependent exponent characterizing the divergence of the critical size, $N^*$, approaching the transition. Also the survival probability, $P_s$, as a function of $N/N^*$, sharpens to a step-function approaching the transition. This is understood comparing the length scale for fluctuations with the increasing critical radius, $R^* = \sqrt{N^*}$. Of particular interest is the effect of increasing diffusion rate, $h$, on nucleation behavior, and specifically the approach to the deterministic mean-field limit. We characterize the increase in $N^*$ with $h$, as well as the sharpening of $P_s$.

The model described here mimics CO-oxidation and poisoning on surfaces. In this respect the model is somewhat unrealistic, for example exhibiting an oxygen poisoning transition for low CO-pressures which is not observed in experiment [21]. However natural refinements to this model to make it more realistic preserve the discontinuous transition, so results of this study still relevant.
APPENDIX I: CRITICAL SIZES FROM THE CC-ENSEMBLE

Simulation of the reaction model in the CC-ensemble [6] involves attempting to deposit A's or B2's, not at fixed relative rates, but rather so as to maintain some fixed value, $\theta_A^*$, of $\theta_A$. Thus one attempts to deposit A if $\theta_A < \theta_A^*$ and B if $\theta_A > \theta_A^*$, and measures the asymptotic value, $y_A^*$, of the fraction of attempts to deposit A. For an arbitrarily large system, $(y_A^*,\theta_A^*)$ should correspond to steady-state values in the conventional (constant pressure) ensemble. Thus one has $y_A^* = y_2$ for the entire coexistence line $\theta_m < \theta_A^* < 1$, where $\theta_m$ is the limiting steady-state value of $\theta_A$, as $y_A \rightarrow y_2^*$.

Behavior is modified by finite-size effects in ways that can facilitate the understanding of nucleation in these models. For a finite LxL system, suppose that $\theta_A^*$ is chosen sufficiently high that the $N^* \sim L^2(1-\theta_A^*)$ non-A sites cannot form a stable percolating region. (A very narrow non-A-poisoned band stretching across the system will not be stable.) Then the one or more non-percolating non-poisoned blobs will be formed. If more than one exists, then $y_A^*$ will be selected to ensure that the mean sized blob has an equal chance of growth or shrinkage, thus maintaining the number of non-A sites. Thus as the system evolves, smaller blobs will occasionally shrink and disappear and the current $y_A$ will increase to accommodate the larger mean blob size. This process will continue until just one blob remains and $y_A^* < y_2$ will correspond to an equal chance of growth or shrinkage for that blob of $N^* \sim L^2(1-\theta_A^*)$ non-A sites. Provided that $N^*$ is sufficiently large, this blob will remain stable as soon as it reaches a circular or near-circular shape (due to surface tension between the A-poisoned and blob states). From mean-field theory, we can apply an argument from synergetics to show that the blob is in the stable steady state while the A-poisoned sea is in a metastable steady state [13].

One thus obtains a relationship, $N^*(y_A^*) = L^2(1-\theta_A^*)$, which appears to map out a middle unstable branch of the phase diagram (see Fig. 1). However it is clear that this branch depends on $L$, i.e., $1-\theta_A^* \sim O(L^{-2})$, as $L \rightarrow \infty$, for fixed $y_A^*$. 
Finite size effects are also seen for $\theta_A^*$ somewhat above $\theta_m$. In this case, $fL^2\theta_m$ A's should be associated with the reactive phase and $(1-f)L^2$ "excess" A's with the A-poisoned phase. Here $f=(1-\theta_A^*)/(1-\theta_m)$ is the fraction of the system covered with the reactive phase. However for $\theta_A^*$ only slightly above $\theta_m$, it is difficult to nucleate the A-poisoned phase and instead a spatially uniform metastable reactive phase is formed with $y_A^*>y_2$. The maximum value of $y_A^*$ thus achieved might be associated with the upper spinodal, $y_{s+}$, however this value may depend on simulation time and system size. For larger $\theta_A^*-\theta_m$, clusters of the A-poisoned phase can nucleate, but there are not sufficiently many excess A's to form a stable percolating region. Instead a critical A-poisoned cluster for $y_A^*>y_2$ is formed (see Ref. [6]).
APPENDIX II: TABLES OF SURVIVAL PROBABILITIES AND N* VALUES FOR
THE EPIDEMIC AB₂ MODEL

Epidemic ZGB simulations were run on an A-poisoned 100x100 lattice with an N=L² empty patch in the center, and the survival probability was measured by running several trials. The number of simulations was determined for each patch size/diffusion rate to assure 1/2% error.

---

8 A 164×164 lattice was used if the patch was larger than N=42².
Table A.1 Survival probabilities for AB$_2$ epidemic model with diffusion rate $h=0$. Interpolated value of $N^*$ (corresponding to $P_s(N)=.5$) are also listed.

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<td></td>
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</tr>
<tr>
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<td>.14</td>
<td>.353</td>
<td>.77</td>
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$N^*$: 842 309 137 79 42
Table A.II Survival probabilities for $\text{AB}_2$ epidemic model with diffusion rate $h=3/8$. Interpolated value of $N^*$ (corresponding to $P_s(N)=.5$) are also listed.

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Table A.III Survival probabilities for AB$_2$ epidemic model with diffusion rate $h=1$. Interpolated value of N* (corresponding to $P_s(N)=.5$) are also listed.

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Table A.IV Survival probabilities for AB$_2$ epidemic model with diffusion rate $h = 4$. Interpolated value of N* (corresponding to $P_s(N) = .5$) are also listed.

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<td>23$^2$=529</td>
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</tr>
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<td>26$^2$=676</td>
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<td>.967</td>
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<td>28$^2$=784</td>
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<td>36$^2$=1296</td>
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<td>38$^2$=1444</td>
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<td>40$^2$=1600</td>
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<td>42$^2$=1764</td>
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<td>71$^2$=5041</td>
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<td>N*</td>
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ACKNOWLEDGMENTS

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REFERENCES


MEAN-FIELD TREATMENT OF NUCLEATION THEORY
FOR FIRST-ORDER POISONING TRANSITIONS
Mean-Field Treatment of Nucleation Theory for
First-Order Poisoning Transitions
J.W. EVANS AND T.R. RAY
AMES LABORATORY AND DEPARTMENT OF MATHEMATICS,
IOWA STATE UNIVERSITY, AMES IOWA 50011
I. INTRODUCTION

The monomer-dimer, or \( AB_2 \) surface reaction model involves adsorption of a monomer species \( A \) and of a dimer species \( B_2 \), and reaction of adspecies \( A \) and \( B \). Schematically, this is described by

\[
A + E \xrightarrow{y_A} A(ads), \quad B_2 + 2E \xrightarrow{y_B} 2B(ads),
\]

\[
A(ads) + B(ads) \xrightarrow{k} AB + 2E.
\]

\( y_A \) and \( y_B \) are the impingement rates for \( A \) and \( B_2 \), respectively\(^1\), and \( k \) is the reaction rate. We consider mean-field treatment of this reaction, where we also allow for spatial inhomogeneity in coverages of adspecies via reaction-diffusion equations.

In the case of the monomer-dimer model, both the \( A \)-poisoned steady state (for simplicity called the \( \alpha \) state), and the reactive steady state (called the \( \beta \) state) are stable in the region \( 0 < y_A < y_2 \). With a planar interface of the \( \beta \) state on one side and the \( \alpha \) state on the other, the \( \beta \) state will displace the \( \alpha \) state if \( y_2 < y_A < y_3 \). If \( 0 < y_A < y_2 \), the \( \alpha \) state will displace the \( \beta \) state. Here \( y_2 \) denotes the equistability point. The idea of nucleation is summarized in Fig. 1. For \( 0 < y_A < y_2 \), if a nucleus of the \( \beta \) state is placed in an (infinite) sea of the \( \alpha \) state, then the size (or radius \( R \)) of the nucleus will determine whether the nucleus grows or shrinks. If \( R < R^* \) it shrinks and if \( R > R^* \) it grows, where \( R^* \) is called the critical radius. At \( R = R^* \) a stable nucleus is formed. As \( y_A \rightarrow y_2 \), the critical nucleus necessarily becomes larger, asymptotically approaching \( \infty \).

We investigate the properties of nucleation for a 2-dimensional model near the \( A \)-poisoning transition with finite reaction rate \( k \), and then extrapolate the values

\(^1\)\( y_A \) and \( y_B \) are normalized so that \( y_A + y_B = 1 \).
to $k = \infty$. We use a circular nucleus, and rely on axial symmetry to allow us to use a cylindrical reaction-diffusion equation.

In section 2, we describe the reaction-diffusion equations for a circular nucleus, and develop a discrete form of these equations that can to be solved numerically. The equations for the planar interface (rectangular coordinates) have been developed for a previous study [1]. We use these equations to determine $\gamma_2$ for the specific values of the reaction rate $k$ chosen. As an aid in finding $R^*$, at the chosen values of $\gamma_1 \gamma_2$, we measure the propagation velocity $c$ for a planar interface trigger wave where $\beta$ displaces $\alpha$. We then use ideas from synergetics [2] to relate the propagation velocity $c$ to $R^*$ by the relation

$$R^* = \frac{D}{c}$$

where $D$ is the diffusion rate. With these estimates as a guide, we finally use the cylindrical reaction-diffusion equations to exactly calculate the critical nucleus sizes $R^*$. The results of our calculations are presented in chapter 3, showing convergence of the $R^*$ for finite $k$ to $R^*$ for $k = \infty$. We conclude by discussing the connection of the mean-field theory results to the lattice gas results previously obtained using the CC ensemble.
Figure 1. Diagram summarizing the idea behind nucleation.

If $R > R^*$ ($R < R^*$) the nucleus grows (shrinks).
II. REACTION-DIFFUSION EQUATION DEVELOPMENT

Our approach is to analyze the reaction-diffusion equations corresponding to the model with finite reaction rate, \( k \), and then extrapolate to \( k = \infty \). These equations are [3]

\[
\frac{\partial \theta_A}{\partial t} = R_A + D_A \nabla^2 \theta_A + (D_{AB} - D_A) (\theta_B \nabla^2 \theta_A - \theta_A \nabla^2 \theta_B), \tag{1}
\]

\[
\frac{\partial \theta_B}{\partial t} = R_B + D_B \nabla^2 \theta_B + (D_{AB} - D_B) (\theta_A \nabla^2 \theta_B - \theta_B \nabla^2 \theta_A)
\]

where

\[ R_A = y_A(1 - \theta_A - \theta_B) - 4k\theta_A\theta_B \]

and

\[ R_B = 2y_B(1 - \theta_A - \theta_B)^2 - 4k\theta_A\theta_B. \]

Here \( D_A \) (\( D_B \)) denotes the coefficient of diffusion for species \( A \) (\( B \)), associated microscopically with the place exchange of adspecies \( A \) (\( B \)) and an empty site; \( D_{AB} \) denotes the coefficient of diffusion associated microscopically with the place exchange of adspecies \( A \) and the adspecies \( B \).

For analysis of the evolution of the configuration shown in Fig. 1, one can, exploiting rotational symmetry in a cylindrical coordinate system with radial coordinate, \( r \), write

\[
\frac{\partial \theta_A}{\partial t} = R_A + D_A \left( \frac{\partial^2 \theta_A}{\partial r^2} + \frac{1}{r} \frac{\partial \theta_A}{\partial r} \right)
\]

\[
+ (D_{AB} - D_A) \left( \theta_B \left( \frac{\partial^2 \theta_A}{\partial r^2} + \frac{1}{r} \frac{\partial \theta_A}{\partial r} \right) - \theta_A \left( \frac{\partial^2 \theta_B}{\partial r^2} + \frac{1}{r} \frac{\partial \theta_B}{\partial r} \right) \right)
\]

and

\[
\frac{\partial \theta_B}{\partial t} = R_B + D_B \left( \frac{\partial^2 \theta_B}{\partial r^2} + \frac{1}{r} \frac{\partial \theta_B}{\partial r} \right)
\]

\[
+ (D_{AB} - D_B) \left( \theta_A \left( \frac{\partial^2 \theta_B}{\partial r^2} + \frac{1}{r} \frac{\partial \theta_B}{\partial r} \right) - \theta_B \left( \frac{\partial^2 \theta_A}{\partial r^2} + \frac{1}{r} \frac{\partial \theta_A}{\partial r} \right) \right).
\]
The existence of a unique solution to these types of equations has been rigorously verified \[4\]. For stable numerical integration of (2) at points near \( r = 0 \), it is helpful to perform the change of variables \( x = \frac{r^2}{4} \) \[5\]. This gives

\[
\frac{\partial}{\partial r} = \frac{r}{2} \frac{\partial}{\partial x} \quad \text{and} \quad \frac{\partial^2}{\partial r^2} = \frac{1}{2} \frac{\partial}{\partial x} + x \frac{\partial^2}{\partial x^2},
\]

so

\[
\frac{\partial \theta_A}{\partial t} = R_A + D_A \left( x \frac{\partial^2 \theta_A}{\partial x^2} + \frac{\partial \theta_A}{\partial x} \right) + (D_{AB} - D_A) \left( \theta_B \left( x \frac{\partial^2 \theta_A}{\partial x^2} + \frac{\partial \theta_A}{\partial x} \right) - \theta_A \left( x \frac{\partial^2 \theta_B}{\partial x^2} + \frac{\partial \theta_B}{\partial x} \right) \right)
\]

and

\[
\frac{\partial \theta_B}{\partial t} = R_B + D_B \left( x \frac{\partial^2 \theta_B}{\partial x^2} + \frac{\partial \theta_B}{\partial x} \right) + (D_{AB} - D_B) \left( \theta_A \left( x \frac{\partial^2 \theta_B}{\partial x^2} + \frac{\partial \theta_B}{\partial x} \right) - \theta_B \left( x \frac{\partial^2 \theta_A}{\partial x^2} + \frac{\partial \theta_A}{\partial x} \right) \right).
\]

We now perform a spatial discretization and assume the existence of an \( \mathcal{X} > 0 \) such that

\[
\theta_A(x) = \theta_A(\mathcal{X}) \quad \forall x > \mathcal{X}
\]

and

\[
\theta_B(x) = \theta_B(\mathcal{X}) \quad \forall x > \mathcal{X}.
\]
This gives the finite coupled set of ordinary differential equations

\[
\dot{A}(1) = R_A(1) + \frac{D_A}{\Delta x} \left( \theta_A(2) - \theta_A(1) \right) + \left( \frac{D_{AB} - D_A}{\Delta x} \right) \left( \theta_B(1)\theta_A(2) - \theta_A(1)\theta_B(2) \right)
\]

\[
\dot{A}(i) = R_A(i) + \frac{D_A}{\Delta x} \left( \frac{x(i)}{\Delta x} \left( \theta_A(i + 1) - 2\theta_A(i) + \theta_A(i - 1) \right) + \frac{1}{2} \left( \theta_A(i + 1) - \theta_A(i - 1) \right) \right)
\]

\[
+ \left( \frac{D_{AB} - D_A}{\Delta x} \right) \left( \frac{x(i)}{\Delta x} \left( \theta_B(i)\left( \theta_A(i + 1) + \theta_A(i - 1) \right) - \theta_A(i)\left( \theta_B(i + 1) + \theta_B(i - 1) \right) \right) \right)
\]

\[
\dot{A}(n) = R_A(n) + \frac{D_A}{\Delta x} \left( \frac{x(n)}{\Delta x} - \frac{1}{2} \right) \left( \theta_A(n - 1) - \theta_A(n) \right)
\]

\[
+ \left( \frac{D_{AB} - D_A}{\Delta x} \right) \left( \frac{x(n)}{\Delta x} - \frac{1}{2} \right) \left( \theta_A(n - 1)\theta_B(n) - \theta_A(n)\theta_B(n - 1) \right)
\]

and

\[
\dot{B}(1) = R_B(1) + \frac{D_B}{\Delta x} \left( \theta_B(2) - \theta_B(1) \right) + \left( \frac{D_{AB} - D_B}{\Delta x} \right) \left( \theta_A(1)\theta_B(2) - \theta_B(1)\theta_A(2) \right)
\]

\[
\dot{B}(i) = R_B(i) + \frac{D_B}{\Delta x} \left( \frac{x(i)}{\Delta x} \left( \theta_B(i + 1) - 2\theta_B(i) + \theta_B(i - 1) \right) + \frac{1}{2} \left( \theta_B(i + 1) - \theta_B(i - 1) \right) \right)
\]

\[
+ \left( \frac{D_{AB} - D_B}{\Delta x} \right) \left( \frac{x(i)}{\Delta x} \left( \theta_A(i)\left( \theta_B(i + 1) + \theta_B(i - 1) \right) - \theta_B(i)\left( \theta_A(i + 1) + \theta_A(i - 1) \right) \right) \right)
\]

\[
\dot{B}(n) = R_B(n) + \frac{D_B}{\Delta x} \left( \frac{x(n)}{\Delta x} - \frac{1}{2} \right) \left( \theta_B(n - 1) - \theta_B(n) \right)
\]

\[
+ \left( \frac{D_{AB} - D_B}{\Delta x} \right) \left( \frac{x(n)}{\Delta x} - \frac{1}{2} \right) \left( \theta_B(n - 1)\theta_A(n) - \theta_B(n)\theta_A(n - 1) \right)
\].
Here " \dot{\cdot} " is the time derivative, and \( x(i) = i \Delta x \) is the discrete space variable\(^2\). \( R_A(i) \) & \( R_B(i) \) are discrete analogs of \( R_A \) & \( R_B \) defined with equation (1). These equations are integrated numerically using the IMSL mathematical software library routine DGEAR.

\(^2\)The space variable is chosen linear in \( z \) to deemphasize the less interesting information at the origin [6].
III. RESULTS

The diffusion rates are set as follows:

\[ D_A = D_B = 1, \quad \text{and} \quad D_{AB} = 0 \]

according to choice (i) in Ref. [3]. The critical radii \( R^* \) are computed for various \( A \)-deposition rates \( y_A \) and for various reaction rates \( k \). We used 200 grid spacings with \( \Delta x \sim R^*/400 \) (corresponding to \( \Delta x \sim 0.1 \) to 4). Each calculation was run up to \( \sim 100 \) time units. Our results are reported in Table I (\( y_2 \) values were computed as in reference [3]). The initial discontinuous interface soon undergoes a smoothing to form a transition layer of constant finite width (see Fig. 2). As \( y_A \to y_2 \), we see that \( R^* \to \infty \) (see Fig. 3). We also confidently expect that \( R^* = R^*_k \) converges to a finite non-zero value \( R^*_\infty \) as \( k \to \infty \). This is shown in Fig. 4 with the extrapolated values listed in Table I.

Using the computed \( R^*_\infty \) values\(^3\) and the definition from Part 3, \( \phi \) is calculated to be \( \phi = 1.93 \). This is close to the theoretical limit of \( \phi = 2 \).

Using results from synergetics [2], one expects to see \( R^*_k(D) \sim \frac{D}{c_k} \), where \( c_k \) is the velocity of the trigger wave [3] corresponding to a planar interface, and \( D \) is the diffusion rate, set here to \( D = 1 \). These velocities were used to give an initial estimate for \( R^*_k(1) \) and are shown in Table II. along with the corresponding ratio \( \frac{R^*_k(1)}{D/c_k} \).

Finally, we note the consistency between the reaction-diffusion equation model and the lattice gas model discussed in Part 3. We have used \( D = 1 \) for all of our calculations. From the form of equations (2) it follows that \( R^*_k(D) = R^*_k(1)D^{1/2} \), so the area of the critical nucleus satisfies

\[ A^*_k(D) = \pi R^*_k(D)^2 = \pi R^*_k(1)^2 D. \]

\(^{3}\)We use the already computed value [1] of \( y_2 = 0.5951 \) for \( k \to \infty \).
Thus $A^*_k(D)$ is proportional to $D$ with the constant of proportionality determined by $R^*_k(1)$. For comparison with the simulations on a square lattice constant $a$, we note that $D_{SIM} = ha^2$ and $A^*_{SIM} = N^* a^2$. Therefore the relation $N^* \sim \alpha + \beta h$ described in part 3 becomes $A^*_{SIM} \sim \alpha a^2 + \beta D_{SIM}$. Our calculated values of $\pi R^*_\infty(1)$ and the calculated values$^4$ of the asymptotic $N^*$ vs. $h$ slopes $\beta$ are listed in Table III, along with the ratio $\frac{\beta}{\pi R^*_\infty(1)^2}$, indicating how close the agreement is.

Table I. Critical radius $R^*_k(1)$ for reaction-diffusion equation treatment with diffusion rates $D_A = D_B = 1$ and $D_{AB} = 0$.

<table>
<thead>
<tr>
<th>$k$</th>
<th>$y_2$</th>
<th>0.90</th>
<th>0.95</th>
<th>0.97</th>
<th>0.98</th>
<th>0.99</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.4435</td>
<td>4.1</td>
<td>8.6</td>
<td>14.1</td>
<td>21.7</td>
<td>43.2</td>
</tr>
<tr>
<td>4</td>
<td>.5193</td>
<td>4.0</td>
<td>6.8</td>
<td>11.6</td>
<td>17.1</td>
<td>33.1</td>
</tr>
<tr>
<td>10</td>
<td>.5498</td>
<td>3.7</td>
<td>6.4</td>
<td>10.4</td>
<td>15.2</td>
<td>29.4</td>
</tr>
<tr>
<td>196</td>
<td>.5878</td>
<td>3.3</td>
<td>5.5</td>
<td>9.0</td>
<td>13.0</td>
<td>25.4</td>
</tr>
<tr>
<td>$\infty$</td>
<td>.5951</td>
<td>3.3</td>
<td>5.5</td>
<td>9.0</td>
<td>13.0</td>
<td>25.4</td>
</tr>
</tbody>
</table>

$^4$See Table IV in part 3, along with figure 14. The slope value for $y_A/y_2 = 0.99$, omitted from the Table IV in part 3, was calculated in the same way as the other values.
Figure 2. Plot of stable nucleus profile for each reaction rate $k$ at $0.98y_2$ (chosen for example).
Figure 3. Plot of critical radius $R^*$ versus $y_A$ for $k = 1, 4, 10 & 196$.

(Vertical line designates location of transition $y_2$)
Figure 4. Plot of critical radius $R^*$ versus $\frac{k}{k+1}$ for $y_A/y_2 = 0.95, 0.97, 0.98 & 0.99$

(Best fit lines facilitate extrapolation to $R^*(\infty)$)
Table II. Use of the velocity of planar interface $c_k$ as a predictor of critical radius $R_k^*$ for reaction-diffusion equation treatment with diffusion rates $D_A = D_B = 1$ and $D_{AB} = 0$. Values shown are $D/c_k$ where $D = D_A(= D_B)$. Values in parentheses are ratio of observed $R_k^*$ to predicted $R_k^*$.

<table>
<thead>
<tr>
<th>$k$</th>
<th>0.90</th>
<th>0.95</th>
<th>0.97</th>
<th>0.98</th>
<th>0.99</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8.4(.49)</td>
<td>15.7(.55)</td>
<td>25.4(.56)</td>
<td>37.3(.58)</td>
<td>74(.58)</td>
</tr>
<tr>
<td>4</td>
<td>5.1(.78)</td>
<td>9.4(.72)</td>
<td>15.1(.77)</td>
<td>22.2(.77)</td>
<td>43.1(.77)</td>
</tr>
<tr>
<td>10</td>
<td>4.2(.88)</td>
<td>7.6(.84)</td>
<td>12.2(.85)</td>
<td>17.8(.85)</td>
<td>34.4(.86)</td>
</tr>
<tr>
<td>196</td>
<td>3.2(1.03)</td>
<td>5.8(.95)</td>
<td>9.3(.97)</td>
<td>13.5(.96)</td>
<td>26.2(.97)</td>
</tr>
</tbody>
</table>

Table III. Comparison of mean-field and lattice gas nucleation. Theory predicts that $A^* = \pi R_k^{*2} \sim \beta$ where $\beta$ is the asymptotic slope of the $N^* \text{ vs } h$ graph for the lattice gas model (from part 3). Included in the table is the ratio between $A_k^*$ and $\beta$, indicating better fit as $y_A \to y_2$.

<table>
<thead>
<tr>
<th>$y_A/y_2$</th>
<th>$\pi R_k^{*2}$</th>
<th>$\beta$</th>
<th>$\pi R_k^{*2}/\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.90</td>
<td>32.2</td>
<td>69.3</td>
<td>.465</td>
</tr>
<tr>
<td>0.95</td>
<td>95.0</td>
<td>155.5</td>
<td>.611</td>
</tr>
<tr>
<td>0.97</td>
<td>254.5</td>
<td>290.8</td>
<td>.875</td>
</tr>
<tr>
<td>0.98</td>
<td>530.9</td>
<td>587.3</td>
<td>.904</td>
</tr>
<tr>
<td>0.99</td>
<td>2026.8</td>
<td>1932.3</td>
<td>1.049</td>
</tr>
</tbody>
</table>
IV. CONCLUSIONS

We have analyzed the nucleation behavior of the $AB_2$ model near the $A$-poisoning transition. A mean-field reaction-diffusion equation approach was used, approximating the critical radius $R^*$ with the velocity of a planar interface at the same below the $A$-poisoning transition, then calculated $R^*$ directly using a numerical scheme to solve the nonlinear reaction-diffusion equation which describes the system.

Our attempts to match the reaction-diffusion theory to the lattice gas theory are successful. The value for $\phi$ agrees well with the theoretical mean-field value, and the matching between the lattice gas nucleation data and that using the reaction diffusion equations was successful.
ACKNOWLEDGMENTS

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REFERENCES

GENERAL CONCLUSIONS

In this thesis we have presented results concerning the monomer-monomer and monomer-dimer surface reaction models.

In the Introduction we presented the underlying theory behind surface reaction kinetics. Background into mathematical modeling, chemical kinetics and catalysis was provided. Examples were given for the monomer-monomer and monomer-dimer surface reactions. The exact rate equations were presented for these reactions, and in the limit of high surface diffusion a mean-field approach was used to investigate the steady states of these equations. Differences between this mean-field theory approach and the lattice gas approach (which is necessary for finite diffusion rates $h > 0$) were noted.

Paper 1 was published in Physical Review E describing the slow poisoning behavior of the monomer-monomer surface reaction for equal impingement rates for the two reactants. The main thrust of this work was in comparing the monomer-monomer model with the voter model, which has been shown rigorously to have only poisoned steady states.

Papers 2 and 3 studied the nucleation theory of the monomer-dimer model. In Paper 2 we studied nucleation in lattice gas models with infinite reaction rate (the ZGB model and a generalization which added surface diffusion). We consider the size dependence of the growth probability $P_s$ of a nucleus of stable phase embedded in a poisoned background. We observed a sharpening of $P_s$ both as $y_a \to y_2$ and as $h$ increased, and saw that the size of a critical radius grew proportionally with $h$. In Paper 3, we examined the mean-field theory of nucleation in the extreme high diffusion case. Using nonlinear reaction-diffusion equations in polar coordinates with finite reaction rates, we examined critical nuclei. We concluded the thesis with comments on the connection of this theory with that of the lattice gas model by extrapolating the mean-field results for finite $k$ to $k \to \infty$. 
REFERENCES


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