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On the self-poisoning of small particles upon island formation of the reactants in a model for a heterogeneously catalyzed reaction

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Assuming the Langmuir–Hinshelwood mechanism, the reaction $A + (1/2)\ B_2 \Rightarrow AB$ is studied on both small homogeneous and disordered surfaces by means of computer simulations. The occurrence of a new self-poisoning regime, where for a determined initial condition the substrata could be completely covered for each of the reactants with a certain probability, is observed and discussed. Large fluctuations in the size of adsorbed islands formed by A and B particles would cause the observed behavior. A crossover from the new self-poisoning regime to a steady state with AB production occurs at $L \simeq 30 \pm 5$ and $p_A \simeq 0.36 \pm 0.02$ for incipient percolation clusters and at $L \simeq 12 \pm 2$ and $p_A \simeq 0.51 \pm 0.02$ on the square lattice, where L and p_A are the lattice size and the mole fraction of the specie A in the gas phase, respectively. It is shown that surface diffusion of A particle does not influence the location of the self-poisoning regime.

I. INTRODUCTION

It is well known that adsorbed atoms and molecules of one or more species may form islands during heterogeneously catalyzed reactions.[1–8] These islands are regions of the substratum covered by a single type of the adsorbed species. The influence of island formation on the reaction kinetics can be understood with the aid of several mean-field models formulated for infinitely large homogeneous surfaces. Essentially, the growth of dense islands causes increased local concentrations of the reactants which may influence adsorption and desorption rates while the reaction takes place mainly along the perimeter of the islands. If the partial pressure of one of the reactants is high enough the growth of large islands of this specie is favored. Occasionally, self-poisoning of the catalyst may occur when the surface becomes fully covered by one of the reactants, as it has experimentally found (see Refs. 5, 9, and references therein) and observed in Monte Carlo simulations.[3–6]

The aim of this work is to study the self-poisoning of small disordered clusters and homogeneous substrata using a model recently proposed by Ziff, Gulari, and Barshad[3,5,6] (ZGB) for a surface reaction of the type $A + (1/2)B_2 \rightarrow AB$. For this purpose, it is assumed that the kinetics follows the Langmuir–Hinshelwood mechanism, that is

$$A(g) + * \rightarrow A(\text{ad}), \tag{1a}$$

$$B_2(g) + 2* \rightarrow 2B(\text{ad}), \tag{1b}$$

$$A(\text{ad}) + B(\text{ad}) \rightarrow AB(g) + 2*, \tag{1c}$$

where (ad) and (g) refer to adsorbed and gas phase, respectively, and (*) denotes a surface site. Equations (1) correspond, for example, to the thoroughly studied oxidation of carbon monoxide, i.e., $A = \text{CO}$ and $B_2 = \text{O}_2$.[10] The study of the reaction described by Eq. (1) on small disordered and homogeneous substrata is useful because most catalysts are constituted by small clusters (i.e., the active sites where the reaction takes place) dispersed on inert supports. Typical sizes of these clusters are between 10 and 100 Å. Also, there are experimental evidences showing that the surfaces of both the small metallic clusters[11] and the supports[12] are, in most cases, examples of microscopic fractals. Furthermore, thin metal films usually employed in adsorption–reaction experiments have percolation cluster like structures.[13] In a related context, the study of the elementary binary reaction $A + A \Rightarrow$ Products reveals that the largest deviations from classical kinetics are found working with small clusters and islands as substrata.[14]

In this work the model reaction is studied by means of the Monte Carlo simulation technique which can successfully be applied to finite samples (disordered and homogeneous), in contrast with mean-field approximations which are more appropriated for the study of reactions on infinitely large homogeneous samples. Also, the simulation method will allow us to understand the role played by concentration fluctuations of the reactants on the self-poisoning process, which is an important advantage since it is known that such fluctuations may, in some cases, significantly affect the reaction kinetics.[15]

II. BRIEF DESCRIPTION OF THE SUBSTRATA AND THE ZGB MODEL

The reaction is simulated on homogeneous samples and on incipient percolation clusters (IPCs), in both cases on the square lattice of size $L \times L$ and assuming periodic boundary conditions. The percolation theory is a rather well established field in physics, so for details on the properties of IPCs we address unfamiliar readers to a number of reviews such as, for example, Stauffer,[16] and references therein. Let us only recall that due to their geometrical heterogeneities, such as branches, loops, dangling ends, etc., IPCs are usually employed for simulations of reactions on rough surfaces and disordered media.[14,17]

The ZGB model used for the simulation of the reaction described by Eqs. (1) on homogeneous substrata has already been studied by means of mean-field approaches,[18] methods

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of equilibrium statistical mechanics,[19] and Monte Carlo simulations.[3,5,6,20,21] For clusters, neither adsorption nor reaction on empty sites of the lattice, i.e., sites which do not belong to the IPC, are considered. Let p_A be the mole fraction of A in the gas phase. Then, a particle striking the surface could be an A with probability p_A or a B_2 with probability $(1-p_A)$. The sticking coefficient is assumed to be 1. To simulate the adsorption process one has to scan the substratum in random order and the Monte Carlo time unit (t) is defined such as each site of the IPC (or the homogeneous surface) is visited once, in the average. Note that A adsorption requires a single adsorption site [Eq. 1(a)] while B_2 adsorption requires two adjacent sites [Eq. 1(b)]. For large L, the last condition is essential for the existence of a reaction window with steady-state production of AB.[3,4] After an adsorption event, all nearest-neighbor (N-N) sites have to be checked in random order for the presence of other adsorbed particles. If A and B are found occupying N-N sites one has a successful reaction event and an AB specie is desorbed leaving two sites of the substratum free [Eq. 1(c)].

Shortcomings of the ZGB model, such as, for example, the irreversibility of the reaction, the absence of surface diffusion and desorption of the reactants, etc. have been already recognized, nevertheless this simple model has concentrated considerable attention due to its interesting critical behavior.[3,5,6,18-21] In fact, two phase transitions from a reactive regime with AB production to off-equilibrium poisoned states with A *and* B species at the critical probabilities $p_{AA}^{\#} \cong 0.525$ and $p_{AB}^{\#} \cong 0.389$, respectively, have been reported on homogeneous media.[3,5,6] Let us emphasize that right at $p_{AA}^{\#}$ and $p_{AB}^{\#}$ the substrata become poisoned only with A and B particles respectively, so we call this kind of poisoning behavior "normal poisoning." In contrast to previous studies of the ZGB model aimed to understand its critical behavior, this work is devoted to the analysis of a regime, only observable with small samples, which has not been studied yet. Within this new regime the substrata become *always* self-poisoned by the reactants, but poisoning could be caused by each of the reactants, with a certain probability, for the same value of p_A. So, this new kind of poisoning behavior is called "anomalous poisoning." Furthermore, in order to relax some of the drastic assumptions of the ZGB model, the effect of surface diffusion of A particles is studied and the influence of reactants desorption is discussed.

III. RESULTS

Figure 1 shows the poisoning probability with B particles [PP(B)] vs p_A for IPCs on lattices of different size. Note that the poisoning probability with A particles [PP(A)] is simply given by PP(A) = 1 − PP(B), since in all these cases runs are performed until the samples become poisoned. A careful analysis of Fig. 1 shows that, on one hand for $0 \leqslant p_A \leqslant p_{AB}(L=15) \simeq 0.325$ the substrata become always poisoned by B particles while on the other hand for $1 \geqslant p_A \geqslant p_{AA}(L=15) \simeq 0.385$ the substrata is always poisoned by A particles (i.e., normal poisoning in both cases). Nevertheless, for $p_{AB}(L) < p_A < p_{AA}(L)$, the surface can be poisoned *either* by A or B particles with different probabilities. Therefore, for the same initial conditions (i.e., p_A and

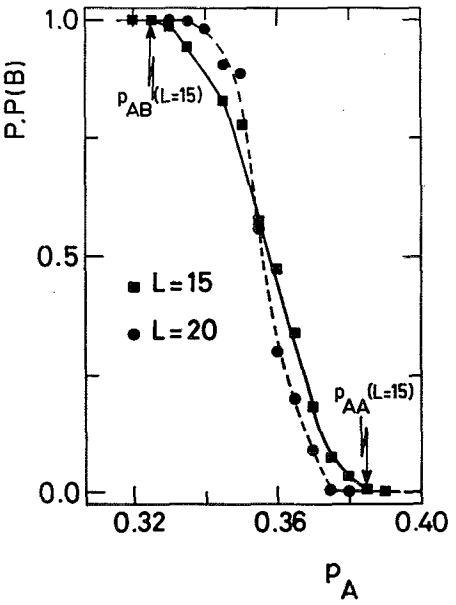

FIG. 1. Plots of the poisoning probability with B particles [PP(B)] vs p_A for IPCs with $L = 15$ (■, full line) and $L = 20$ (●, dashed line). Averages are taken over 150 different IPCs. The arrows show the position of $p_{AB}(L)$ and $p_{AA}(L)$ for $L = 15$, more details in the text.

L) the system could evolve towards two different poisoned states, i.e., this is the new anomalous poisoning behavior already mentioned. Particularly, close to $p_A^* \simeq 0.355$–0.360, one has that PP(A) ≃ PP(B). The same reasoning just applied to lattices of size $L = 15$ also holds for $L < 30$ (see also Fig. 1 for $L = 20$) but remembering that both $p_{AA}(L)$ and

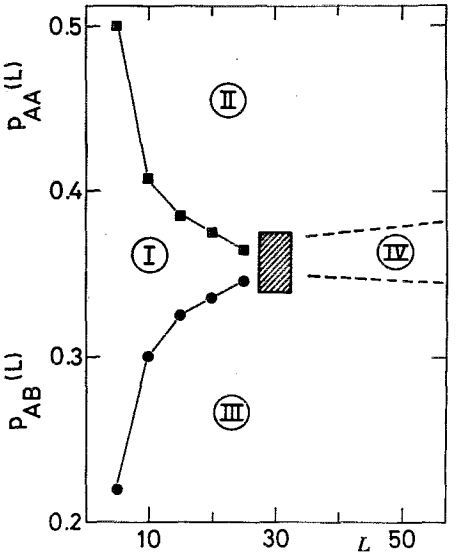

FIG. 2. Plots of (●) $p_{AB}(L)$ and (■) $p_{AA}(L)$ for IPCs vs L ($L < 30$). Results obtained from a set of figures like Fig. 1. Within region I the surface becomes anomalously poisoned either with A or B particles. Within regions II and III the samples are poisoned by A and B particles, respectively. Within region IV (shown schematically) the reaction reaches a reactive steady state with production of AB species (for details on the phenomena occurring in the borders between regions II–IV and III–IV, see Refs. 20 and 21. Within the dashed rectangle, centered at $p_c \cong 0.36$ and $L_c \cong 30$, one observes the crossover from the anomalous poisoning regime to the reactive steady state. More details in the text.

$p_{AB}(L)$ are L dependent values of the mole fraction of A in the gas phase. Also, from Fig. 1 and similar plots obtained for $L<30$ (not shown here), it follows that p_A^* does not appreciably depend on L.

Figure 2 shows plots of both $p_{AA}(L)$ and $p_{AB}(L)$, obtained as in Fig. 1, vs L for $L<30$. Within region I the samples become anomalously poisoned as discussed above, while within regions II and III the samples are normally poisoned by A and B particles, respectively. For $L \geqslant 30 \pm 5$, anomalous poisoning is not observed any more and within region IV the reaction reaches a reactive steady state with AB production. This reactive regime has been already analyzed in detail for both homogeneous[3,5] and disordered[20,21] samples.

Extensive simulation results show that there is a crossover from the anomalous poisoning regime (region I) to the reactive regime (region IV) close to $L_c \simeq 30 \pm 5$ and $p_{AC} \simeq 0.36 \pm 0.02$ [where L_c and p_{AC} are the lattice size and the value of p_A at the crossover, respectively (see, also, the dashed rectangle in Fig. 2)]. The behavior of the system close to the crossover region can also be understood by comparing the histograms of Fig. 3. These histograms show the poisoning probabilities with A and B particles, after 3×10^4 Monte Carlo time steps, vs the lattice size for different values of p_A. Note that in contrast with the results shown in Fig. 1, some samples are not poisoned during the evolution of the simulation because the final Monte Carlo time step is fixed in advance. In fact, it has to be recognized that both $p_{AA}(L)$ and $p_{AB}(L)$ should slightly depend on a certain reasonable compromise between the number of averaged samples (N_A) and the maximum time (t_M) that one can wait for poisoning. That is, for $N_A \to \infty$, $t_M \to \infty$, and L finite, it should be possible to find samples poisoned with A (B) particles even for very small (large) values of p_A, respectively. This fact resembles the percolation problem where for L finite one can find percolation clusters for very small values of the probability. Also for this reason a more detailed determination of the crossover boundary is not possible. Pointing again our attention in Fig. 3, the histogram [Fig. 3(a)] corresponds to $p_A = 0.375$ and consequently one observes anomalous poisoning with A and B particles for $L \leqslant 20$. Note that the poisoning probability with A particles is always greater than that with B particles, while for $L \geqslant 25$ the system becomes normally poisoned by A particles only. So, the histogram [Fig. 3(a)] shows the crossover from region I to region II close to $L \simeq 20$ (see, also, Fig. 2). The histogram [Fig. 3(b)] corresponds to $p_A = 0.355$, i.e., a value very close to $p_{AC} \simeq p_A^* \simeq 0.36$. In this case one has anomalous poisoning with A and B particles even for $L = 35$, while for $L \geqslant 40$ poisoning is not observed at all. This result reflects the crossover from region I to the reactive regime of region IV. Finally, the histogram [Fig. 3(c)] corresponds to $p_A = 0.335$ and it shows the crossover from region I to region II close to $L \simeq 20$.

Since the behavior of the reaction of IPCs is quite different than on homogeneous media,[3,5,20] it is interesting to investigate if the regime of region I is also possible on the latter. For this purpose the reaction described by Eqs. (1) has been simulated on the square lattice for $L<30$. Simulations on bigger lattices have already been published but anomalous poisoning has not been reported yet.[3,5,6] Figure 4 shows the

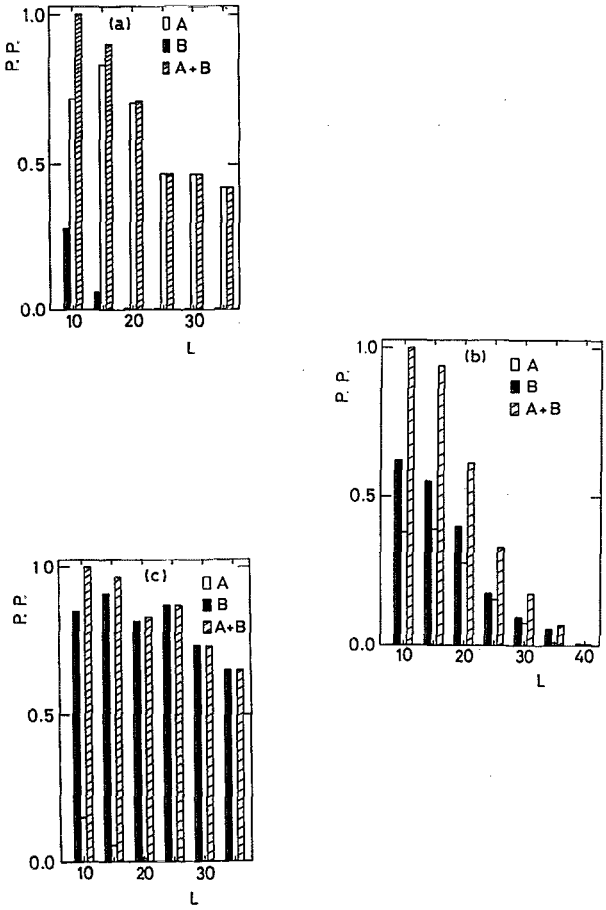

FIG. 3. Histograms showing the poisoning probability (PP) of IPCs vs the lattice size (L) for different values of p_A. Black, white and dashed columns correspond to the PP with A, B, and with both $A+B$ particles, respectively. Results averaged over 150 reactions on different IPCs. Runs are performed until a maximum of 3×10^4 Monte Carlo time steps. (a) $p_A = 0.375$, (b) $p_A = 0.355$, and (c) $p_A = 0.335$.

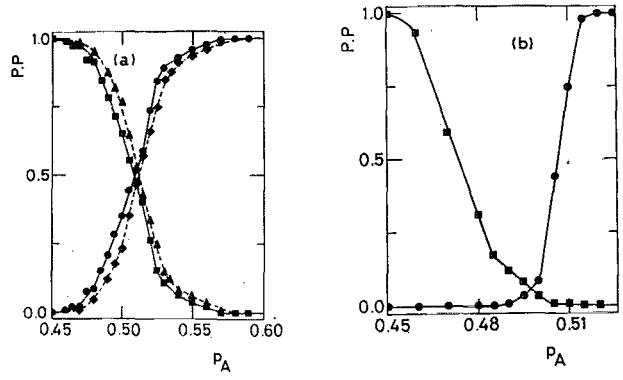

FIG. 4. Plots of the poisoning probability with (●) A and (■) B particles vs p_A for homogeneous substrata without diffusion of A particles. (a) $L = 5$ and (b) $L = 10$. The symbols (◆) and (▲) correspond to the poisoning probabilities with A and B particles, respectively, but considering surface diffusion of A particles (more details in the text). Averages are taken over 200 samples up to 3×10^4 Monte Carlo time steps.

poisoning probabilities with A and B particles against p_A for homogeneous samples. For $L=5$ [Fig. 4(a)], all samples (200 for each data point) become anomalously self-poisoned after few Monte Carlo time steps within a wide range of p_A values ($0.45<p_A<0.57$). For $L=10$ [Fig. 4(b)], all samples become poisoned only for $p_A \leqslant 0.45$ and $p_A \geqslant 0.52$ after 3×10^4 Monte Carlo time steps. With the available statistics anomalous poisoning with A and B particles is observed within $0.49 \leqslant p_A \leqslant 0.51$. Self-poisoning by the reactants has not been observed for $L=15$ and taking over 200 simulations for each value of p_A ($0.495 \leqslant p_A \leqslant 0.51$) until 3×10^4 Monte Carlo time steps. This result suggests that the crossover between regions I and IV on homogeneous substrata should lie close to $L'_c \simeq 12 \pm 2$ and $p'_{AC} \cong 0.51 \pm 0.02$.

In order to analyze the effect of reactants surface diffusion on the predictions of the ZGB model, the diffusion of A particles is studied. This assumption is supported by the fact that adsorbed carbon monoxide [i.e., A(ad) particles in Eq. (1)] is much more mobile than adsorbed oxygen atoms [B(ad) particles in Eq. (1)]. During the simulation, A particles are allowed to diffuse with the same probability to each of the four N-N sites, provided that the selected N-N site is empty. Figure 4(a) also shows the poisoning probabilities with A and B particles vs p_A for homogeneous samples ($L=5$) taking diffusion into account. As it follows from Fig. 4(a), the relaxation of the diffusion restriction causes a little shift of the poisoning probabilities. It is found that samples become anomalously self-poisoned within the range $0.445<p_A<0.575$, that is nearly the same interval of p_A that without diffusion. For $L=10$ anomalous self-poisoning with A diffusion is found within $0.49<p_A<0.515$, i.e., the same range that without diffusion, so the results are not shown in Fig. 4(b) for the sake of clarity. Additional simulation studies of A diffusion show negligible effects on the critical behavior of the ZGB model.[22]

IV. DISCUSSION AND CONCLUSIONS

The anomalous poisoning regime observed within region I can be understood on the basis of general arguments. Let us first discuss the behavior of the reaction on IPCs. During the transient period, which runs between the beginning of the simulation with clean samples until the final poisoned (regions I, II, III) or reactive (region IV) states, one observes the growth of islands formed by A or B particles. Also, within this transient regime one has production of AB species and the reaction takes place mainly in the border between islands of A and B particles. Due to the reaction the typical size ξ_A (ξ_B) of A (B) islands and consequently the coverage ϑ_A (ϑ_B) of A(B) particles fluctuates, respectively. These fluctuations are shown in Fig. 5 where the growth of ϑ_A is accompanied by the diminution of ϑ_B and vice versa, until the sample suddenly becomes poisoned at $t\simeq3.9\times10^4$ Monte Carlo steps. Strong fluctuations of the coverages are characteristic of small samples but they become attenuated for large values of L.

A careful finite-size scaling analysis of region IV reveals that both ξ_A and ξ_B diverge like[20,21]

$$\xi_x \sim |p_A - p^*_{Ax}|^{-\nu_x}, \quad L=\infty, \quad \nu_x = 1, \tag{2}$$

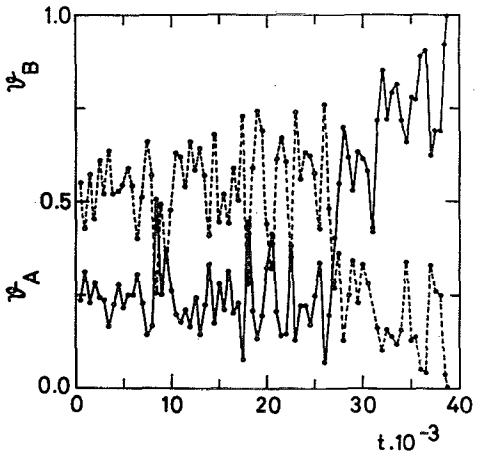

FIG. 5. Plots of ϑ_A (full line) and ϑ_B (dashed line) for an IPC vs the Monte Carlo time t during the transient period of the reaction. $L=20, p_A=0.375$.

for p_A close to p^*_{AX} ($X=A,B$) with $p^*_{AA}=0.408$ and $p^*_{AB}=0.314$, respectively. Both p^*_{AA} and p^*_{AB} are critical probabilities at which kinetic (irreversible) transitions between the steady-state regime and off equilibrium normally poisoned states with A and B particles take place, respectively. Within the interval $p^*_{AB}<p_A<p^*_{AA}$, both ζ_A and ξ_B remain finite but it is possible to distinguish the following regimes:

(i) for $L>35$ and close to p^*_{AA} one has that $\xi_A>\xi_B$ and if $\xi_A>L$ the sample becomes normally poisoned with A particles (region II). Otherwise ($\xi_A<L, \xi_B<L$) the system reaches a reactive steady state regime (region IV). (ii) For $L>35$ and close to p^*_{AB} one has that $\xi_B>\xi_A$ and the same argument used in case (i) holds but now for B particles. (iii) At the crossover from region I to region IV, i.e., close to $p_{AC}\simeq0.36\pm0.02$ and $L_c\simeq30\pm5$, one has that $\xi_A\simeq\xi_B\simeq L_C$. The last is precisely the necessary condition for the onset anomalous poisoning because both A and B islands could grow and match the lattice size causing poisoning. An estimation of p_{AC} can be obtained by demanding $\xi_A=\xi_B$ and using Eq. (2). Neglecting possible differences between the prefactors and using the values of p^*_{AA} and p^*_{AB} one gets $p_c\cong0.361$, i.e., an excellent agreement considering that Eq. (2) only holds close to p^*_{AA} and p^*_{AB} and in the limit $L\Rightarrow\infty$. (iv) Within region I, anomalous poisoning occurs because both $\xi_A>L$ and $\xi_B>L$. The poisoning probability with A or B particles is the same for $p^*_A\cong p_{AC}\cong0.36\pm0.02$ (see Fig. 1) because $\xi_A\cong\xi_B$, independent of L. On the other hand, for $p_A>p^*_A$ ($p_A<p^*_A$) one has $\xi_A>\xi_B$ ($\xi_A<\xi_B$) and the poisoning probability with A (B) particles increases, respectively (see Figs. 1 and 3). Samples become poisoned with A (B) particles and probabilities close to 1 for $p_A\geqslant p_{AA}(L)$ [$p_A\leqslant p_{AB}(L)$] because $L<\xi_A$ and $\xi_A\gg\xi_B$ ($L<\xi_B$ and $\xi_B\gg\xi_A$), respectively.

The discussion of the anomalous poisoning regime on homogeneous samples (square lattice) must be more qualitative due to the lack of a finite-size scaling analysis. In this case, kinetic transitions from the steady-state reactive regime with AB production to off equilibrium states poisoned

with A and B particles are observed at $p_{AA}^{\#} \simeq 0.525$ and $p_{AB}^{\#} \simeq 0.389$,[3,5,6] respectively.

Figure 4(a) shows that for $L = 5$ the anomalous poisoning window partially overlaps the steady-state reactive window and that the poisoning probability with A and B particles is roughly the same for $p_A \simeq 0.51$, i.e., very close to $p_{AA}^{\#}$. For $L = 10$ [Fig. 4(b)] the anomalous poisoning window lies within the reactive window but also very close to $p_{AA}^{\#}$. Therefore, the crossover between region I and region IV, in the square lattice, should be very close to $p_{AA}^{\#}$, probably because at this critical point the transition is of first order instead of second order transitions observed at $p_{AB}^{\#}$ (Refs. 3, 5, and 6) as well as at both p_{AB}^{*} and p_{AA}^{*} on IPCs.[20] In fact, at the first order phase transition, A islands of relevant size suddenly grow only close to $p_{AA}^{\#}$. Therefore, for the square lattice the condition $\xi_A \simeq \xi_B$ would hold close to $p_{AA}^{\#}$. Since the onset of anomalous poisoning requires that $\xi_A \simeq \xi_B \simeq L_c$ and the size of B islands is expected to be very small close to $p_{AA}^{\#}$ the fact that L_c' (square lattice $\simeq 12 \pm 2$) $< L_c$ (IPCs $\simeq 30 \pm 5$) is also reasonable.

The effect of A(ad) diffusion has also been studied in order to remove one of the most crude assumptions of the ZGB model. It is shown that diffusion causes almost negligible effects on the location of the anomalous self-poisoning regime in particular and on the overall predictions of the ZGB model in general.[22] In another context it should be mentioned that diffusion-limited reactions of the types (i) $A + A \rightarrow$ inert, (ii) $A + B \rightarrow$ inert, etc., are being studied with increasing interest (see, for example Refs. 14, 17, and 23–25 and references therein). Comparisons between results of the reaction (ii) and the ZGB model with diffusion are rather difficult because on the former both species require a single adsorption site and the interesting results; namely the so called fractal chemical kinetics,[14,17,25] segregation of the reactants,[23,24] etc., are obtained in the low concentration limit of the reactants; while on the latter the existence of a reactive window is due to the requirement of two adsorption sites for B_2 and the study of both, the critical behavior and the poisoning regime, are performed in the high concentration limit of one reactant.

Another drastic assumption of the ZGB model is the absence of reactants desorption. Mean-field and Bethe–Peirels approximations of the ZGB model[18] show that desorption of both reactants causes a little effect on the position of $p_{AA}^{\#}$ and $p_{AB}^{\#}$. The existence of a finite desorption probability of one or both reactants inhibits, in principle, the respective poisoned states and the anomalous poisoning regime. Nevertheless, preliminary results[22] obtained assuming only A desorption with probability p_D, show that the rate of AB production decreases abruptly few orders of magnitude close to $p_{AA}^{\#}$ ($p_A \gtrsim p_{AA}^{\#}$), for $p_D < 0.05$. Therefore, under these conditions one may consider the surface as "effectively" poisoned, while mathematically, with infinite reaction times, only poisoning by B should be possible.

The existence of the "anomalous self-poisoning" regime is a prediction of the ZGB model applied to the reaction described by Eq. (1) on both disordered and homogeneous media. Within this context, if an appropriate mixture of two gases (A and B_2) is flowing over a catalyst constituted by finely divided metal particles supported by an inert matrix, one could expect that particles with typical sizes smaller than L_c (or L_c') would become poisoned by A or B species, while only bigger particles would contribute to the reactive regime with AB production. It is interesting to note that the conditions for the existence of the anomalous self-poisoning regime are not confined to a narrow interval which would make unlikely its observation in real systems. In fact, assuming a N-N distance of 2.5 A between metal atoms, $L_c = 35$ and $L_c' = 12$ correspond to 90 and 30 A, respectively; i.e., within the typical size of metallic particles present in real catalysts.[11] On the other hand, the possible values of the mole fraction of A and B in the gas phase where the regime is expected to occur, account for between 30 and 1 % of the total ($L = 5$ and $L = 30$ in Fig. 2, respectively); i.e., a rather wide interval. Another remarkable feature of the anomalous self-poisoning regime is that its location in a pressure-lattice size diagram (Fig. 2) depends on the fractal dimension of the substrata (D_f). For $D_f = 1.90$ (i.e., IPCs) the range of favorable conditions is wider than for $D_f = 2$ (i.e., homogeneous media). A more detailed comparison with the experiments is not possible due to the crude assumptions involved, nevertheless we expect that the prediction of the ZGB model concerning the anomalous poisoning regime could be verified (particularly with small fractal particles) or ruled out using modern surface sensitive techniques.

It is interesting to note that simulations performed using 1D rings of 2^{18} sites suggest the absence of a steady-state regime with AB production.[5] That is, samples become always poisoned with A or B particles but, anomalous poisoning has not been reported.[5] We expect that the anomalous poisoning regime should also occur in 1D but, in rings smaller than those used in Ref. 5.

Considering that within region I, for the same initial conditions (p_A and the lattice size but for a different set of random numbers) the system could evolve towards two different final self-poisoned states, it could be conjectured that the "anomalous poisoning regime" is some sort of "chaotic regime." The differentiation between chaos and fluctuations of the stochastic system requires considerable effort[26] and it is not clear if the available methods would work using very small samples ($L \lesssim 30$ and $L \lesssim 12$ for IPCs and the square lattice, respectively) as in the present case. So, no attempts in this way were made.

Summing up, anomalous self-poisoning by the reactants during the reaction described by Eq. (1) has been observed on both the square lattice and IPCs. The anomalous (normal) poisoning regime occurs when adsorbed islands of *both* reactants (a single reactant) could grow and match the sample size, respectively.

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