Molecular Turing structures in the biochemistry of the cell

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Reactive lattice gas automata simulations show that Turing structure can form on a mesoscopic scale and are stable to molecular fluctuations in this domain. Calculations on the Sel'kov model suggest that Turing instabilities can give rise to global spatial symmetry breaking in ATP concentration within the cell cytoplasm with a mesoscopic Turing scale well within typical cell dimensions. This leads to a new mechanism for the global breaking of energy distribution in the cell. It also leads to reappraisal of the importance of the Turing effect on extended biochemical spatial structures and energy transport available to cell morphogenesis.

I. INTRODUCTION

Turing structures are well-studied nonlinear diffusive instabilities that provide a mechanism for global spatial symmetry breaking in far-from-equilibrium coupled reaction–diffusion systems. They allow a global transition from a homogeneous spatial state to a spatial state with nontrivial symmetry. These structures are characterized by an intrinsic wavelength—the Turing scale—which is independent of the geometrical parameters of the reacting system. Using chemical systems to study Turing structures implies the dynamical variables are the concentrations of reacting components and the symmetry breaking parameters are macroscopic reaction rates and diffusion coefficients.

Since Turing's classic paper, Turing structures have been suggested as a possible basis for morphogenesis in large-scale biological systems. In the past few years Turing structures have been seen in convincing laboratory demonstrations in the chlorite–iodide–malonic acid–starch reaction within gel reactors and intensively studied by several groups. All these experiments show distinctly threedimensional structures, and can be fully studied only by computer simulation. The intrinsic Turing scale in this reaction is of order a few tenths of a millimeter. It is believed that gels have tunable effective diffusion constants—starch acts as a delay mechanism within the gel allowing the separation of diffusion constants necessary for a true Turing effect without exotic mathematical mechanisms. Turing structures on much smaller length scales have been observed in catalytic oxidation of CO on Pt(110) surfaces but here the mechanism involves reconstruction of the Pt surface.

It is likely, as mentioned in Ref. 8, that Turing instabilities are a key to pattern formation in cell membranes, since substrate inhibition and membrane bound biochemical components are available. But autocatalytic processes can also generate molecular Turing instabilities and within the cell cytoplasm provide a mechanism for spatial symmetry breaking on a global scale.

Investigating this problem entails studying small systems on short time scales where the foundations of macroscopic reaction–diffusion descriptions are questionable. Full molecular dynamics simulations can be used to explore this regime and account for molecular fluctuations but are difficult to implement for multispecies, far-from-equilibrium reacting systems with large populations. Master equation methods and simulations based on Bird's method are other possible schemes for extending calculations beyond the reaction–diffusion regime. No studies of Turing structures using these methods have appeared in the literature.

We have used lattice-gas methods to study the dynamics of such systems on the relevant length and time scales since these methods provide a mesoscopic description of the reacting system. In Sec. II we describe the reactive lattice-gas method and discuss the space and time scales probed by this method. Section III presents simulation results for the Sel'kov model, a reaction scheme that models an important part of the glycolytic pathway. Finally in Sec. IV we argue on the basis of these results for the possibility that Turing bifurcations may occur in the glycolytic pathway within the single cell cytoplasm and thus provide a general mechanism for symmetry breaking within the cell.

II. REACTIVE LATTICE-GAS DYNAMICS

Lattice-gas cellular automaton (LGCA) models use a simplified molecular dynamics that embodies many features of real collision and propagation processes. The dynamics is fully discretized: space, time, and molecular velocities are discrete variables. The real collision processes in the system are modeled by collision events at lattice
nodes \( r \) and are embodied in collision rules. For applications to hydrodynamics it is essential that the automaton dynamics incorporate the correct collision invariants and symmetries; it has been demonstrated that properly constructed models can be used to simulate the Navier–Stokes equation. For reactive systems with no bulk fluid flows the appropriate macroscopic description is the reaction–diffusion equation and lattice-gas rules must incorporate the elastic and reactive collision events that give rise to this macroscopic equation.

The diffusion coefficient ratio is an important parameter in the study of Turing bifurcations and it is necessary to be able to construct automaton rules that allow the diffusion coefficients of the chemical species to be manipulated. In the reactive LGCA model this is achieved by using a multilattice version of the automaton dynamics where molecules of each species are confined to their own species lattice \( \mathcal{L}_\sigma \), \( \tau = 1, \ldots, n \) on which they move and undergo elastic collisions. The nonreactive dynamics can be carried out independently on each species lattice and this flexibility allows one to vary the diffusion coefficients of the different species.

All the species lattices \( \mathcal{L}_\sigma \) have identical node labels \( r \) and reactive collisions at lattice sites with same node label are determined by the numbers of molecules of the various species at the node. Thus, reactive collisions give rise to a coupling among the species lattices. From a physical point of view one may regard the reactive species as being dispersed in some background medium, solvent, gel, etc. Provided the solute species are dilutely dispersed in this background medium the overwhelming majority of nonreactive collisions will occur between the solute species, whose dynamics is of interest, and the molecules of the background medium; hence, it is reasonable to assume that the elastic collisions occur independently for each chemical species. This provides some rationale for the independent elastic collision dynamics on the species lattices. Reactive collisions typically occur with a much smaller frequency than the elastic collisions and to a first approximation it is plausible to suppose that the molecules are thermalized between reactive events. (This approximation neglects perturbations of the velocity distribution by the reaction but these effects are usually quite small and need only be considered in more refined models of the dynamics. The qualitative features of the time evolution and pattern formation processes will not change.) Thus, reactive collisions occur from a thermalized velocity distribution and are determined by the local species numbers.

An exclusion principle is assumed where only one particle of a given species \( \tau \) may reside at a node with a given velocity. If the coordination number of the lattice is \( m \) and there are \( n \) species then the maximum occupancy of a site with a given node label is \( mn \). The exclusion principle allows the dynamics to be simulated very efficiently since occupancy of a velocity cell on a species lattice is a Boolean random variable and a velocity configuration at a node \( r \) on a species lattice \( \mathcal{L}_\sigma \) is described by an \( m \)-bit binary word. Physically, the exclusion principle prevents particle density from building up at a local region of space and leads to some restrictions on the range of processes that can be simulated with the automaton.

Given this background the reactive lattice-gas automaton rules may be formulated as follows. The building blocks of the automaton rules are the three operators:

1. Propagation operator, \( P_\tau \), which moves particles of species \( \tau \) one lattice unit in the directions specified by their velocities to neighboring nodes of the lattice;
2. Velocity randomization, \( R_\tau \), which randomly rotates the particle velocity configuration on \( \mathcal{L}_\sigma \); and
3. Chemical transformation, \( C \), which effects local chemical reactions among the species. Given \( n \) chemical species \( \{ X_\tau ; \tau = 1, \ldots, n \} \) an elementary reaction among these species may be written as

\[
\alpha \cdot X \rightarrow \beta \cdot X.
\]

where \( X = (X_1, X_2, \ldots, X_n) \) is a vector of chemical species and \( \alpha = (\alpha_1, \alpha_2, \ldots, \alpha_n) \) and \( \beta = (\beta_1, \beta_2, \ldots, \beta_n) \) are vectors of stoichiometric coefficients, where \( 0 < \alpha_\tau, \beta_\tau \leq m \) for each \( \tau = 1, \ldots, n \). The reaction occurs with probability \( P(\alpha | \beta) \), which depends only on the occupancy of the nodes \( r \) on \( \mathcal{L}_\tau, \tau = 1, \ldots, n \) and not on the velocity configurations. The probability matrix \( P = [P(\alpha | \beta)] \) completely specifies the automaton reactive dynamics.

One step of the full automaton dynamics is determined by the operator \( M \) which is given by the following composition of these elementary operators:

\[
M = \prod_{r=1}^n (P_\tau \circ R_\tau)^{l_r} \circ C.
\]

Here \( (P_\tau \circ R_\tau)^{l_r} \) stands for \( l_r \) compositions of the operator \( P_\tau \circ R_\tau \). Since the product \( P_\tau \circ R_\tau \) generates a diffusion process and \( l_r \) may be different for each species it is not difficult to show that the diffusion coefficient for species \( \tau \) is \( D_\tau = l_r D \), where \( D \) is the diffusion coefficient when \( l_r = 1 \). This rule allows control over the magnitudes of the relaxation times of the non-reactive processes of each species individually relative to the chemical reaction time of the system.\(^{17}\)

The final step is the construction of the reaction probability matrix \( P \). Two principles are used in its construction. First, the elements of \( P \) should describe the mechanism of the reaction under study. One may assume that only one elementary reaction may occur at a node at a given time step. Thus, the only nonzero elements of \( P \) are those that correspond to possible reaction steps in the mechanism. Second, the mean field rate equations can be used as guide in the selection of numerical values for the microscopic reaction probabilities. Let \( \rho_\tau \) be the average concentration of species \( \tau \) per node and \( c_\tau = \rho_\tau / m \) be the average concentration of species \( \tau \) per velocity cell. Ignoring correlations the probability of occupancy of a node is given by a binomial distribution and the rate of change of \( \rho_\tau \) is given by\(^{14,15}\)

\[
\frac{d\rho_\tau}{dt} = \sum_{\alpha, \beta} (\beta_\tau - \alpha_\tau) P(\alpha | \beta) \prod_{\tau=1}^{n} \left( \frac{m}{\alpha_\tau} \right) c_\tau^{\alpha_\tau} (1-c_\tau)^{m-\alpha_\tau}.
\]
Identification of the phenomenological mass action rate law with Eq. (3) yields conditions on the $P(\alpha|\beta)$ and guarantees that the automaton dynamics will reduce to the chemical rate law if correlations and fluctuations can be neglected. The exclusion principle and the condition $0 < P(\alpha|\beta) < 1$ provide additional constraints on the allowed values of $P(\alpha|\beta)$. Other ways may be devised for the construction of $P$ but the above scheme has proved to be effective.

Space and time scales. The automaton provides a mesoscopic description of the reactive dynamics; it involves coarse graining over short distance and time scales. Any complete picture of the coarse-grained dynamics entails a consideration of the relation between the model space and time scales and those of the true molecular dynamics underlying the process of interest. In order to establish this relation for the reactive lattice-gas automaton it is necessary to consider the manner in which molecules and their collisions are represented in the automaton.

Since the solvent is not explicitly treated in the automaton the nature of the medium in which the solute species are dispersed enters only indirectly through the nonreactive collision rates which set the time scale for the dynamics. This naturally implies that the automaton time scale must be interpreted differently for different media; it will not be the same, for example, in dense gases, liquids, gels, or porous media. (In refined automaton models it is possible to build the locally inhomogeneous structure of a gel or porous medium into the dynamical model but we restrict our considerations to the simpler case where this local inhomogeneous “solvent” structure is treated in a mean-field sense.)

The automaton particles reside at the nodes of the lattice where they undergo collisions. For the automaton dynamics described above there is a maximum of $nm$ particles of all species per node but in actual simulations the average number of particles per node is far smaller, usually a few particles, since node concentrations rarely achieve their maximum values. In this description solute molecules are treated as point particles and the length scales must be large enough for this approximation to be reasonable. From a physical point of view each cell of the automaton corresponds to a volume of solution large enough to contain, on average, a few solute molecules and the associated solvent molecules for the given solute concentration of the solution. The solution must be sufficiently dilute that the solute species are small compared to the cell volume.

As an example, consider a solution of small molecules and suppose the linear dimension of a volume of solution containing on average a few solute molecules is $10^{-5}$ Å. In a dense liquid of small molecules the diffusion coefficient is typically of order $10^{-10}$ cm$^2$/s; thus, on average, it will take a molecule 1 ns to traverse the cell length. Consequently, for such a system, we may say that the automaton length scale is tens of angstroms and the time step is nanoseconds. Of course, the interpretation of the length and time scales in the automaton simulations depends on the system being modeled and the nature of the collision processes that are involved in the motion of a molecule over a distance corresponding to one coarse-grained cell length. The cell sizes may be somewhat larger and the diffusion coefficients somewhat smaller in gel media but the order of magnitude estimate given above suffices for illustration. Thus, a mesoscopic treatment of the reacting system is possible.

Due to the introduction of an artificial dynamics which is intended to reproduce only gross features of the real collision processes only order of magnitude estimates of the space and time scales can be given. One cannot literally associate an automaton cell with a specific solution volume nor can one associate real solute particles with the fictitious automaton particles. Nevertheless, the automaton is constructed to describe the major features of the dynamics on mesoscopic scales. This coarse-grained treatment of the dynamics is the key to success of the automaton: It treats the inessential aspects of the dynamics in a simple fashion that leads to orders of magnitude savings in computer time in comparison with molecular dynamics and focuses on the processes and time scales of interest. Because of this reactive lattice-gas automata can explore phenomena that cannot be studied by full molecular dynamics simulations and since the automaton incorporates fluctuations that arise from the interaction of reaction and diffusion processes it can be used to study regimes that are inaccessible by reaction-diffusion simulations.

III. SEL’KOV MODEL AND GLYCOLYSIS

The full molecular mechanisms of cellular biochemical processes are incompletely understood. However, to study the problem of molecular-scale Turing structures it is sufficient to examine the dynamics of a highly stylized model for the product-activated conversion of substrate $S$ to product $P$, typical of many enzyme catalyzed reactions:

$$
\begin{array}{c}
A \rightleftharpoons S, \quad S + 2P \rightleftharpoons 3P, \quad P \rightleftharpoons B. \\
\end{array}
$$

If the concentrations of the $A$ and $B$ species are assumed to be fixed by external sources and sinks of reagents the rate laws are two coupled equations for the substrate and product concentrations:

$$
\frac{d[S]}{dt} = k_1[A] - k_{-1}[S] - k_2[S][P]^2 + k_{-2}[P]^3, \quad (5)
$$

$$
\frac{d[P]}{dt} = k_3[B] - k_3[P] + k_2[S][P]^2 - k_{-2}[P]^3. \quad (6)
$$

These rate equations are the Sel’kov model (including reverse reactions) which was derived from analyzing the glycolytic pathway. This model has a complicated bifurcation structure and contains steady states, oscillations and bistabilities.

In the lattice-gas model the dynamics of the constrained $[A]$ and $[B]$ species are not followed, instead it is assumed that these species are uniformly distributed in space and their concentrations always enter in combination with rate constants. Thus, only the motions of $S$ and $P$ species are followed and the $[A]$ and $[B]$ concentrations are
FIG. 1. Reactive lattice-gas automaton Turing structure. The Turing pattern evolved from a random initial condition. The microscopic automaton dynamics was constructed (Ref. 14) to correspond to the Sel'kov rate law with the following kinetic parameters: $k_{1}(A)=0.002 \; 565 \; 673$, $k_{2}=0.006 \; 65 = 10k_{1}$, $k_{3}=k_{-3}=0.015$, and $k_{-2}[B]=0.000 \; 531 \; 334$. The concentration of the product, $P$, is displayed. The diffusion coefficients are $D_{A}=50D$ and $D_{P}=2D$ with $D=0.25$ in units of the lattice spacing squared per time step. The simulation was carried out on square lattice (1024 $\times$ 1024) with periodic boundary conditions and one of the two realizations that result from the square lattice simulation is shown. The six panels (a)-(f) (left to right and top to bottom) are for times $t=0$, $t=1000$, $t=2000$, $t=3000$, $t=10000$, and $t=15000$.
used as bifurcation parameters. The calculations were carried out on a square lattice and the (25 × 25) reaction probability matrix \( P(\alpha_S P, \beta_S P) \) that specifies the transition probability for the reaction, \( \alpha_S S + \alpha_P P \rightarrow \beta_S S + \beta_P P \), was constructed to conform to the reaction mechanism, Eq. (4). In addition, the elements of the probability matrix satisfy conditions such that the mean field rate law derived from the automaton dynamics corresponds to Eqs. (5) and (6). For the reaction probability matrix used in our simulations the chemical relaxation time is of the order of \( 10^3-10^6 \) time steps.

The spatial symmetry-breaking instability in the automaton can be studied by choosing rate constants, \( [A] \) and \( [B] \) concentrations and diffusion coefficients to drive a Turing bifurcation in the Schrödinger–diffusion equation.\(^{14}\) The parameters were selected so that Eqs. (5) and (6) possess a stable fixed point (focus) which lies close to a Hopf bifurcation line. This solution is linearly stable to small inhomogeneous perturbations if the diffusion coefficients of \( S \) and \( P \) are equal. Given these fixed rate constants and \( [A] \) and \( [B] \) concentrations the diffusion coefficient ratio was varied until the homogeneous steady state became unstable and the system bifurcated to an inhomogeneous steady state. Let \( L \) be the matrix that determines the linearized evolution of Eqs. (5) and (6) about the homogeneous fixed point:

\[
\begin{align*}
L_{SS} &= -k_1 - k_3 \rho_S^2, \\
L_{SP} &= -k_1 + 2k_2 \rho_S^* \rho_P^* - 3k_2 \rho_P^2, \\
L_{PS} &= -2k_3 \rho_S^* \rho_P^* + 3k_2 \rho_P^2, \\
L_{PP} &= -k_3 \rho_P^2.
\end{align*}
\]

where \( \rho_S^* \) and \( \rho_P^* \) are the homogeneous steady state concentrations. A Turing bifurcation will occur when \( D_S/D_P \) exceeds the critical value.

\[
\frac{D_S}{D_P} = \left( \frac{|L|}{-L_{SP}L_{PS} + 2(-L_{SP}L_{PS}|L|)^{1/2}} \right)/L_{PP}.
\]

The critical wavelength is

\[
\lambda_T = 2\pi \left( \frac{D_S D_P}{|L|} \right)^{1/4}.
\]

Figure 1 shows the results of an automaton simulation in a parameter region (given in the figure caption) where a Turing structure is expected to exist. For these kinetic parameters the critical diffusion coefficient ratio is \( D_S/D_P = 16.2 \). In the simulation the diffusion coefficients were selected to be \( D_S = 50 \) and \( D_P = 2 \) with \( D = 1/4 \) cells\(^2\)/time step. The random initial condition was chosen so that the average concentrations of the \( S \) and \( P \) species were equal to the homogeneous fixed point values. The system evolved from this random initial state to a spot state with disordered hexagonal symmetry whose wavelength compares well with the value predicted from the linear stability analysis \( \lambda_T \) (linear stability) = 173.4 cells, \( \lambda_T \) (simulation) = 179 ± 18 cells.\(^{23}\) The Turing pattern is stable for long time periods but is subject to the effects of fluctuations (cf. Fig. 1). Figure 2 shows the time evolution of the spatially averaged concentrations of \( S \) and \( P \) as a plot in the concentration plane. From this figure and Fig. 1 it is clear that the system has evolved from a random initial state to an inhomogeneous steady state confirming the fact that a Turing bifurcation has taken place.

An inhomogeneous state develops quickly from the random initial state, in a few thousand time steps (see the first few panels in Fig. 1). During this period the global concentration undergoes damped oscillations to the inhomogeneous steady state concentrations. These bulk oscillations are evident in Fig. 2 and in panels (b)–(d) of Fig. 1. For subsequent times the bulk concentration fluctuates slightly about its steady state value. In this longer time regime the Turing pattern undergoes a slow annealing process where defects in the hexagonal spot patterns move. Larger fluctuations can also lead to spot fusion followed by reorganization of the pattern. Apart from these dynamical effects the gross structure of the pattern does not change (cf. panels (e) and (f) of Fig. 1). If the simulation is repeated for the same system parameters and diffusion ratios but with \( D_S = 25 \) and \( D_P = 5 \), the Turing length is smaller by a factor of the \( 2 \) and the concentration fluctuations produced by the reaction are stronger (the time scale separation between the reactive and nonreactive events is smaller). In this case, once again an inhomogeneous state develops quickly from a random initial state but the resulting pattern has far less organized structure; it is difficult to discern regular hexagonal regions. From such simulations one can obtain information on the lower limits for regular Turing structure formation in the presence of fluctuations. We note that even the highly inhomogeneous but irregular spatial structure can give rise to important physical and biological effects.

**IV. DISCUSSION**

These results show that under suitable conditions for fast reactions Turing structures can exist on small meso-
scopic length scales and are robust under the rather strong molecular fluctuations that occur on these scales. The results further suggest that reactions that occur naturally and universally in cell cytoplasm may have Turing scales much below a typical cell size, perhaps on the molecular scale of typical biochemical reactions. A universal chemical system in cytoplasm is the glycolytic pathway, which is a well-studied and complex reaction. However, it is worth noting that the Sel'kov model describes qualitatively the basic dynamical properties of the phosphofructokinase reaction in glycolysis controlling the crucial fructose 1,6-diphosphate subsystem. It is a compact description of a vital piece of the highly complex nonlinear chemistry of the glycolytic reaction which models part of the ATP-ADP conversion process. (The ATP and ADP molecules are rather small chemical species and conform to the cell size restrictions discussed above.) The molecular reaction mechanism we investigated and the corresponding Sel'kov equations are skeletal models compared to the full glycolytic pathway in the cell. However, our lattice-gas simulations demonstrate that fast autocatalytic biochemical reactions in small gel-like or condensed phase systems can create Turing structures. Therefore we conjecture that mesoscopic Turing patterns exist in more complex biological contexts; for example, in the simplest case, in the glycolytic pathway of prokaryotic cells under anaerobic conditions.

Much experimental data exists on the oscillatory and limit cycle regimes of the glycolytic pathway, for very dilute enzyme concentrations. From the laboratory kinetic data on glycolysis the Turing length has been estimated to be $10^{-4}-10^{-2}$ cm, large compared to mesoscopic dimensions. The reactive events occurring in living cell cytoplasm are less well understood. Cell enzyme concentrations may be orders of magnitude greater than in laboratory experiments and dynamically controlled from within the cell. This allows fast reaction rates by concentration effects for the nonlinear kinetics or by reaction mechanism modification.

The existence of Turing patterns depends on the general kinetics of the reaction mechanism and reaction rate parameters (rate constants and feed concentrations), as well as the diffusion coefficient spread of the chemical species. Glycolysis, and other autocatalytic biochemical reactions, certainly can satisfy these kinetic criteria as shown by our Sel'kov model results. Furthermore, the complex, dense, membrane-filled structure of cell cytoplasm is an effective gel in our sense of the term. The general situation for many cell reactions is that they are enzyme catalyzed and that enzyme concentrations are very high. This suggests that mechanisms similar to those found in laboratory gel experiments may operate to create sufficient diffusion coefficient differences. From the analysis of laboratory experiments, it is likely that an effective renormalization of species diffusion constants is a generic effect in gels or in any system containing a structural matrix which suppresses hydrodynamic modes and can temporarily bind at least one chemical component. Because of these features we believe our results are robust and independent of the details of the model. The order of magnitude of the Turing scale should be given correctly in our simulations. We note that the Turing instability supplies not only the topological structure of Turing patterns but also metric information embodied in the intrinsic Turing scale, which could influence other scales in molecular biochemical pathways.

We have less to say about the importance of this Turing mechanism in eukaryotic cells both because of their more complex internal structure and because most ATP is produced by other mechanisms in the mitochondria or chloroplasts. Still, we speculate that the reaction and spatial scales in these organelles may support such patterns.

In summary, recent simulations with reactive lattice gas automata provide evidence that Turing structures can exist on mesoscopic scales in the presence of fluctuations. The Turing effect is a viable mechanism for global symmetry breaking of extended spatial concentrations of chemical species in biochemical pathways at mesoscopic scales. The simulations suggest that global symmetry breaking in the spatial concentration distribution can occur on scales as small as 0.1–1 μm for small molecules assuming the automaton length scale is 10–100 Å. For normal dense phase diffusion coefficient values ($D \sim 10^{-5}$ cm$^2$/s) this implies chemical relaxation times in the μs to ms range. Based on our simulations we predict that spatial inhomogeneities in the ATP concentration within the prokaryotic cell cytoplasm which arise from Turing bifurcations can occur on these mesoscopic scales.

Direct experiments at these scales in the cell, necessary to verify these predictions, are unavailable at present. The subject of spatial scale breaking in species concentration within the cell is likely to become more extensive and complex as our ability to simulate these processes grows in sophistication. In a sense we have a digital laboratory to test out ideas at a mesoscopic scale. Novel experimental methods will have to be developed if this class of ideas is to be tested in cell environments at molecular and mesoscopic scales.

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The automaton modeling of collision processes resembles that of the BGK kinetic equation [P. L. Bhatnagar, E. P. Gross, and M. Krook, Phys. Rev. 94, 511 (1954)] where collisions are treated as thermalizing events that occur with a given frequency and molecules free stream between such collisions. Thus each "collision" event is the result of a number of real collisions in the system.

For very large solute molecules the solution should be sufficiently dilute that their volume fraction is small compared to the cell volume. If the solvent is very large and the solute concentration is high the mean field description of the solvent may have to be replaced by a more detailed solvent model in order to describe the local solute dynamics accurately.

This corresponds to a few hundredths molar solution, which is sufficiently dilute so that a small fluid volume can easily contain the small solute molecules and many solvent molecules.

In dense gases both microscopic collisional and collective (Stokes-law-like) effects contribute to the diffusion process [J. T. Hynes, R. Kapral, and M. C. Weinberg, J. Chem. Phys. 70, 1456 (1978)]. In this regime the time scales may be somewhat shorter and the automaton collisional processes may mimic more closely those of the real system.


The Turing length was extracted from the simulation by measuring the distances between the density maxima on a 1024 × 1024 lattice.
