Structural Coupling of a Potts Model Cell

Eran Agmon\(^1\)*, James A. Glazier\(^2\), and Randall D. Beer\(^3\)

\(^1\) Department of Biological Sciences, Columbia University, New York City, NY 10027, USA
\(^2\) Biocomplexity Institute and Department of Physics, Indiana University, Bloomington, IN 47406, USA
\(^3\) Cognitive Science Program and School of Informatics and Computing, Indiana University, Bloomington, IN 47406, USA
* corresponding author: agmon.eron@gmail.com

Abstract

Organisms are embedded in environments, with which they engage in an ongoing two-way interaction called structural coupling. It is in this context that an organism develops, behaves, thrives, and ultimately dies. This paper introduces a network-based methodology for analyzing how an organism and environment unfold together through structural coupling, and demonstrates this methodology in a cellular Potts model. A morphology-environment transition network consists of all reachable combinations of morphological and environmental states as its nodes, and the transitions between these morphology/environment states as its edges. In a given simulation, the model cell and its environment move through this network as both dynamically unfold. Analysis of such a network reveals several interesting properties, including attractor states, divergence of network structure when the cell is placed in different environments, and niche construction in which the cell’s influence over its environment increases its own viability.

Introduction

Organism and environment are an inseparable pair, bound together by an ongoing two-way interaction called structural coupling [9]. As a consequence of their contact, the organism in part determines the state of the environment, and the environment in part determines the state of the organism. This exchange has the potential to disrupt the organism’s function, and send it on a trajectory towards disintegration. Despite this, adapted organisms maintain their viability, in part through readjustment of their own internal structures, and in part through modifications to their environments.

Structural coupling has implications for both behavior and development. Organisms often rely on feedback between themselves and their environment to guide coordinated behavior [4]. Development is also modulated by environmental factors such as temperature, nutrients, and social contact [3; 15]. Finally, the organism shapes its environment through its activities and byproducts, and in doing so it influence its behavioral and developmental circumstances [13].

In a previous set of papers, we developed a network-based methodology for analyzing how stationary model organisms respond to fixed environments by undergoing sequences of morphological change [2; 1]. A morphology transition network is the set of reachable morphologies and the transitions between them. Analysis of such networks can tell us each morphology’s local robustness (probability of maintaining the given morphology in a single time step), local plasticity (probability of changing to a different viable morphology in a time step), and local fragility (probability of transitioning to death within one time step). They can also demonstrate global properties of how organisms move through the space of possible morphologies throughout their development. These properties include irreversibility, branching, and attractors. Crucially, the morphology transition networks can also reveal each morphologies’ viability: the average expected survival time in the given environment starting from that morphology.

In this paper, we extend the network-based methodology to address structural coupling. To do so, we adopt the cellular Potts model (CPM) framework. CPM has been developed to simulate many cells and tissue-level behaviors, including cell sorting [7], tumor growth [12], and the aggregation, crawling behavior, and fruiting body of *Dictyostelium discoideum* [11]. It is ideal for investigating structural coupling because of its behavioral richness, its ability to capture biophysical properties of real cells [8], and the ease of incorporating environmental features such as chemical fields. In this paper, CPMs are subjected to theoretical study rather than comparison with empirical observations, adding to short list of other related theoretical developments [14; 6]. Rather than modeling many cells and their interactions, as is typical with CPM, this paper focuses on the micro-level dynamics of a single Potts model cell. This analysis will focus on how the one cell moves through a space of possible morphologies, how the environment influences dynamics in this space, and how the cell’s influence over the environment in turn shapes its own possibilities for morphological change.

First, we introduce the cellular Potts model framework, and the specific instantiation that is analyzed in the remainder of the paper. In the second section, we analyze its instantaneous transitions in a fixed chemical field environment that the cell cannot influence. In the third section, we de-
derive its morphology transition network in the fixed environment, and demonstrate some of this network’s properties. In the fourth section we introduce structural coupling into the model by adding a simple feeding behavior that removes chemicals from the environments and uses it to maintain cellular growth. An analysis of the resulting behavior warrants an extension of the network methodology, which incorporates the cell’s local environmental state into the transition network. We demonstrate several unique properties of structural coupling, which include diverging paths through the set of morphologies, an organism’s ability to influence its own energy landscape, and global properties of the morphological states such as viability.

**Cellular Potts Model**

The cellular Potts model (CPM) is a well-developed framework for simulating whole cells as deformable spatial objects that can move through environments made of chemical fields and other cells [7]. A basic CPM consists of a spatial lattice, in which individual cells are defined as regions of multiple lattice sites that share an index value, $\sigma$ (Figure 1). CPM cells behave by free energy minimization, in which internal and external forces acting on its outer membrane are resolved. This procedure can reproduce many biophysical properties of cells, such as cell sorting, adhesion, cohesion, and compressibility [8].

The effective energy, $\mathcal{H}$, is defined by a Hamiltonian equation that includes all of the forces acting upon the cell:

$$\mathcal{H} = \sum_{ij} \sum_{i'j'} J(\sigma_{ij},\sigma_{i'j'}) \left(1 - \delta_{ij,i'j'}\right) + \sum_{\sigma} \lambda (a_{\sigma} - A(\sigma))^2$$

These sums are computed across a lattice of size $N_x \times N_y$. The cell’s sites are identified by a shared index value, $\sigma$. The first sum of the Hamiltonian is the adhesion constraint between neighboring sites. Interaction values, $J$, define the adhesion between sites with different index values: positive interaction values assure that large bending fluctuations of the cell membrane are avoided, and that sites with the same index value remain close to each other. The Kronecker delta term $(1 - \delta_{ij,i'j'})$ keeps neighboring sites with the same index value from contributing to the effective energy. The second sum of the Hamiltonian is the area constraint: $a$ is the current cell area, $A(\sigma)$ is the cell’s target area, and $\lambda$ is a constant that specifies the strength of the area constraint. This can be interpreted as the cell’s internal pressure; if the current area is less than the target area, the cell will tend to grow; if the current area is more than the target area, the cell will tend to shrink.

Typically, a CPM is run with the Metropolis Monte Carlo method [10], for which there are as many updates as there are sites in the lattice. In a single update, a site $(ij)$ is chosen from the lattice at random, and a copy attempt is made from one of its von Neumann neighbors $(i'j')$ to that site. If different index values occupy these sites, the algorithm changes the site’s index value to its neighbor’s index value $(\sigma_{ij} \rightarrow \sigma_{i'j'})$ with a probability given by the Boltzmann acceptance function:

$$p(\sigma_{ij} \rightarrow \sigma_{i'j'}) = \left\{ \begin{array}{ll} 1 & \text{if } \Delta \mathcal{H} < 0 \\ e^{-\Delta \mathcal{H}} & \text{if } \Delta \mathcal{H} \geq 0 \end{array} \right.$$  

$\Delta \mathcal{H}$ is the change in effective energy if the copy occurs. $T$ is the simulation temperature, which determines the fluctuations with which a cell’s effective energy can increase. This function always accepts changes that move the cell towards lower effective energy, and changes that increase its effective energy are accepted with the Boltzmann probability ($e^{-\Delta \mathcal{H}}$).

Chemotaxis is introduced by adding a chemical field to the environment, and an additional term to $\Delta \mathcal{H}$ [11]. This new force causes the cell to move up chemical gradients by reducing the effective energy of configurations that move towards increased chemical concentrations.

$$\Delta \mathcal{H} = \mathcal{H}_{\text{after}} - \mathcal{H}_{\text{before}} - \mu (c_{ij} - c_{i'j'})$$

Here, $c$ represents the local chemical concentrations at each of the two sites, the one being copied $(i'j')$ and the one that is being updated $(ij)$. The membrane experiences forces in the directions of increased chemical concentrations, with a strength of $\mu$.

In this paper, we consider a model on a $N_x = N_y = 20$ square lattice, with toroidal boundary conditions. The model has a temperature $T = 1$, interaction values $J = (60 \ 60 \ 1)$, volume constant $\lambda = 40$, chemotaxis constant $\mu = 800$. These parameters were selected to balance the different forces, so that none of them dominates the cell dynamics. Different parameters are expected to yield different results.
Chemotaxis is sensitive only to the relative differences in concentration rather than absolute concentrations, and in an environment with a linear slope all locations have the same concentration. This makes the location of the cell irrelevant in the analysis of transitions, because it will behave the same at any location (with the exception of when it is on the boundary that wraps from the top of the lattice, at $y = 20$, to the bottom of the lattice, $y = 1$. This region will not be included in this section’s analysis).

A cell’s transition probabilities depend on its particular environment, as is demonstrated in Figure 2. Figure 2A shows the transition probabilities of a morphology in a chemical field with no gradient ($\alpha = 0.0$), and Figure 2B shows the transition probabilities of the same morphology in an environment with a positive gradient ($\alpha = 0.4$). $\alpha$ determines the gradient as shown in Figure 2C. The highlighted sites of Figure 2A and 2B can become part of the cell if they are presently part of the environment, or they can become a part of the environment if they are presently part of the cell. The probabilities associated with these changes are calculated by identifying the update probabilities of each site according to equation 2, and then normalizing these probabilities. As can be seen, morphologies are accessible to the cell in different probabilities in these different environments. The environment influences the cell’s total plasticity (probability of morphological change) and robustness (probability of remaining at the same morphology): morphology 2A’s plasticity is 0.27 while 2B’s plasticity is 0.03, and reciprocally 2A’s robustness is 0.73 while 2B’s robustness is 0.03.

A cell’s behavior is a sequence of configurational transitions (Figure 3). When a transition occurs, the cell may or may not be in a new configuration. In the fixed environment, if the cell’s morphology remains the same, its transition probabilities also remain the same — this can be seen by comparing the transition probabilities at $t = 1$ with $t = 2$, and $t = 3$ with $t = 4$. If the cell transitions to a new configuration, a new set of transitions becomes available, and can reach configurations there were not previously directly accessible.

**One-Step Transitions**

Given a cell’s morphology (the spatial arrangement of its constituent sites) and its local environment (the adjacent set of lattice sites that can influence a cell’s instantaneous behavior), there are a finite number of possible transitions that could occur within one time step. In order to simplify the analysis of this transition structure, the chemical field is in this section held at a fixed gradient with a linear slope. Chemotaxis is sensitive only to the relative differences in concentration rather than absolute concentrations, and in an environment with a linear slope all locations have the same relative difference between adjacent sites. This makes the location of the cell irrelevant in the analysis of transitions, because it will behave the same at any location (with the exception of when it is on the boundary that wraps from the top of the lattice, at $y = 20$, to the bottom of the lattice, $y = 1$. This region will not be included in this section’s analysis).

A cell’s transition probabilities depend on its particular environment, as is demonstrated in Figure 2. Figure 2A shows the transition probabilities of a morphology in a chemical field with no gradient ($\alpha = 0.0$), and Figure 2B shows the transition probabilities of the same morphology in an environment with a positive gradient ($\alpha = 0.4$). $\alpha$ determines the gradient as shown in Figure 2C. The highlighted sites of Figure 2A and 2B can become part of the cell if they are presently part of the environment, or they can become a part of the environment if they are presently part of the cell. The probabilities associated with these changes are calculated by identifying the update probabilities of each site according to equation 2, and then normalizing these probabilities. As can be seen, morphologies are accessible to the cell in different probabilities in these different environments. The environment influences the cell’s total plasticity (probability of morphological change) and robustness (probability of remaining at the same morphology): morphology 2A’s plasticity is 0.27 while 2B’s plasticity is 0.03, and reciprocally 2A’s robustness is 0.73 while 2B’s robustness is 0.03.

A cell’s behavior is a sequence of configurational transitions (Figure 3). When a transition occurs, the cell may or may not be in a new configuration. In the fixed environment, if the cell’s morphology remains the same, its transition probabilities also remain the same — this can be seen by comparing the transition probabilities at $t = 1$ with $t = 2$, and $t = 3$ with $t = 4$. If the cell transitions to a new configuration, a new set of transitions becomes available, and can reach configurations there were not previously directly accessible.

**Morphology Transition Network**

In a given simulation, a cell can only experience one behavioral trajectory through the space of possible morphologies. But due to the model’s stochasticity, different simulations can be expected to generate different behaviors and reach different morphological states. In a morphology transition network the nodes are the set of reachable morphologies, and the edges between them are the transitions between these morphologies, weighted by their probability of occurring. A full morphology transition network is obtained by characterizing a configuration’s full set of possible one-step transitions, and then repeating the process for all uncovered morphologies until closure is achieved (i.e. every transition results in a previously characterized morphology) [1; 5].

It is straightforward to derive a full morphology transition network for the Potts model cell when there is no structural coupling. Each configuration has fixed transition probabilities no matter where it is placed, so each morphology will therefore have the same transition probabilities and will only

---

**Figure 2:** One-step transition probabilities for the same morphology in two different fixed environments. A) Transition probabilities in an environment with a flat chemical field ($\alpha = 0.0$). Darker colors indicate a higher probability of that site being updated. Sites within the cell’s boundaries would become part of the environment in the next time step. Sites within the environment would become part of the cell. B) Transition probabilities of the cell in an environment with a positive linear chemical gradient ($\alpha = 0.4$). The arrow points in the direction of increased chemical concentrations. C) Schematic showing how the environment’s linear chemical gradient is determined by $\alpha$.

**Figure 3:** A behavioral sequence in environment with $\alpha = 0.4$. Snapshots show how the cell’s morphology and transition probabilities unfold over time, as the configuration changes within its fixed environment.
Figure 4: A) A morphology network, showing all reachable morphologies in an environment with no chemical gradient ($\alpha = 0.0$). Node size represents robustness (probability of remaining in that configuration). Edge weight is directly proportional to probability of transition. Death (∅) and two morphologies are pointed to — the initial morphology and the single pixel morphology, which is the only one that connects to death. B) The histogram shows the distribution of $\Delta H$ (differences in effective energy between source and target morphologies) across all observed edges, with an arrow showing the cutoff made in the network derivation; $\Delta H$ values above this arrow have a low probability ($p \leq 3.0e^{-44}$) and were removed from the network, and the morphologies associated with them were not further searched. C) The same network shown in (A) is mapped according to volume and effective energy. Node size indicates that number of configurations found at the exact value of volume and effective energy. The gray bar along the y-axis represents the death state. D) A second network obtained in a graded chemical field ($\alpha = 0.4$) is shown in the same format as above.

have to be characterized once. We impose a maximum $\Delta H$ on the network (shown by the red arrow in Figure 4B) to rule out unlikely transitions with high energy states. After all transition probabilities for a given morphology are assessed (including robust transitions), they are normalized to determine the probability of any given transition occurring.

The resulting morphology transition network is shown in Figure 4. This network has 130 morphologies, including the death state. Statistical analysis reveals that they vary widely in their responses. The death state is the only absorbing state in this network, and has a robustness of 1. Robustness of morphologies occur with a probability from the range 0.0 to 0.875, and plasticity occurs with the inverse probability of 0.125 to 1.0. This means there are some nodes that have no robustness, and will undergo every site update available to them, and there are some highly robust morphologies, which maintain their state up to 87.5% of the time.

The analysis is further enriched by viewing the morphologies according to their volume and effective energy, shown in Figure 4C. Here, energy states are quantized into just a few states for each given volume, and the transitions between nodes are orderly, only connecting nodes that are within one volume unit of each other. The network in Figure 4D was obtained in a positive chemical gradient ($\alpha = 0.4$), previously shown in Figure 1. The increase in the number of nodes and their size in Figure 4D demonstrates a large increase in the amount of reachable morphologies when the cell is placed in a graded environment. Morphologies at higher effective energy come with more scattered configurations that have longer perimeters, and therefore higher adhesion with the environment. These are generally not favored, but in Figure 4D the chemical gradient increases the effects of chemotaxis, which overcomes the adhesion constraint and allow the cell to reach more scattered morphologies. These provide bridges to more morphologies, and so increase the size of the morphology transition network.

**Structural Coupling**

Up to this point, we have only studied one side of the organism-environment interaction — the cell had no influence over a static environment. For full structural coupling, the environment must have its own dynamics that are influenced by the organism’s state, hence it will be called a dynamic environment. To explore the consequences of this relationship, feeding is here introduced. Feeding is a behavior required by all lifeforms, which provides a strong example of structural coupling; the environment is modified as materials are removed from it, and the cell’s intrinsic state
depends on these materials for its maintenance, energy, and growth. This dependence makes a mere chemical field into a food field. The organism must continue to move in order to find food, using behaviors such as chemotaxis.

Feeding requires two additions to the model: 1) environmental dynamics that incorporate the cell’s state, and 2) food dependence that determines a cell’s growth based on its food availability.

The environmental dynamics are introduced in three parts: 1) diffusion, which makes chemical concentrations spread throughout the lattice, 2) logistic population growth, which slowly increases local chemical concentrations towards a carrying capacity, and 3) consumption, which removes chemical concentrations in the presence of the cell. These factors are implemented by the following partial differential equation:

\[
\frac{\partial f}{\partial t} = D_f \nabla^2 f + r_f f (1 - f/K) - u f_{\sigma=1}
\]

(4)

The first term is the diffusion component, with \( D_f \) as the diffusion rate. The second term is logistic population growth for the food: \( r_f \) is the growth rate towards a carrying capacity, \( K \). The third term is consumption by the cell: \( u \) is the rate by which food \( f \) is consumed at every site with a cell index value, \( \sigma = 1 \). In this paper we use \( D_f = 0.01 \), \( r_f = 0.01 \), \( K = 1 \), and \( u = 0.4 \).

The cell’s food dependence is introduced by adding dynamics to a cell’s target volume, in which food availability determines the target volume and therefore growth rate. The desired dynamics would give the cell a target volume to find food, using behaviors such as chemotaxis.

In order to obtain a full morphology-environment transition network, we used a sampling-based approach. We ran 8,000 simulations for 2,000 time steps each, and recorded their trajectories through the morphology-environment state space. Observed morphologies and their local environments were grouped according to equivalence classes that account for rotational symmetry (at 0°, 90°, 180°, and 270°), and reflection across horizontal and vertical axes. The spatial extent of a local environment is the set of lattice sites that can influence the cell within a single time step: all sites occupied by the cell, and all sites directly adjacent to it. Observed local environments were binned by discretizing the concentrations at each of the environment’s sites into one of
Figure 5: Structural coupling sequence shows a trajectory in which the configuration and environment unfold together. Morphologies are shown in white and their local environments are shown in green. Darker shades of green indicate higher concentrations of food. Morphology and local environment units such as the ones shown here make up the nodes of the next section’s morphology-environment transition networks.

Figure 6: Two superimposed morphology transition networks highlight their divergence: one was derived in the static environment (without structural coupling), and the other was derived in the dynamic environment (with structural coupling). Each node represents a morphology — morphologies shared by both networks are white, those resulting from the static environment alone are green, and those resulting from the dynamic environment alone are orange. The same color scheme pertains to the edges, except black (rather than white) edges are shared by both networks. Both networks have the same starting morphology (the node labeled A) and the same initial environment, they are also searched to an equal depth of four steps from the starting configuration. On the right, a subset of the combined network is shown in greater detail, illustrating divergence between the static environment (green edges) and the dynamic environment (orange edges).

4 possible concentrations, \([0, 0.25], (0.25, 0.5], (0.5, 0.75], \) and \((0.75, 1]\), and identifying environments with identical discretized values across the spatial grid. Given these conditions, the sampled morphology-environment transition network reached convergence by about 2,500 simulations in both the number of observed morphology/environment pairs and the number of observed edges between these pairs. The final network has a total of 626 unique morphologies, 38,571 unique environments, and 39,502 unique pairs of morphology and environment.

The full morphology-environment transition network contains one absorbing state corresponding to death, and one nearly-absorbing state corresponding to a stable block morphology with a symmetric environment. Figure 7 shows the nodes within five transitions of these two attractors. In the figure, node size depicts robustness, and the two attractors have the two highest robustness values: death is irreversible with a robustness of 1.0, and the stable block state has a high robustness of approximately 0.9997 (meaning it has only a probability of 0.0003 of transitioning to a different state in a given time step). There is some cross-over between the local basins of the attractors, meaning there are some morphologies that are in the basins of both attractors. Finally, each node has a viability value, which is the average expected time to death [1]. The stable block state has the highest viability, of approximately 150,000 time steps to death. The remainder of the nodes have varying viabilities, which appear to decrease as they get closer to the death state.

Structural coupling allows the cell to create an environment in which its viability is increased. One example of this behavior is shown in Figure 8A, with a sequence of transitions that end at the highly viable stable block morphology and its symmetric environment. Before reaching this state, the cell goes through some transient morphologies, it
Figure 7: A morphology-environment transition network, highlighting the attractor basins of the network’s two (near) attractors — death and the block morphology with a symmetric environment. Each node represents a unique pairing of cell and local environment, and only nodes within a depth of five edges from the basins are shown — 1,961 out of the total 39,502. Node size corresponds to robustness. Nodes are colored according to their viability (average time steps to death), with green being the most viable, and red being the least viable. Here, maximum viability is approximately 150,000 time steps, and the minimum viability is 0.

reaches the block morphology and then escapes from it for a short period of time before returning, and then the environment slowly fills in around it, and it remains in the highly robust attractor for the remainder of the simulation (shown up to 1,000 time steps). We can see the reason for this in Figure 8B, which shows the $\Delta H$ values of all possible transitions at each time step. The earlier states have a broad range of available $\Delta H$ values, many of them below the $\Delta H = 0$ line at which the transition is always accepted. Meanwhile, the later states have a narrower range and all options are above the $\Delta H = 0$ line, this makes escape much less likely. We can also see that the earlier block morphology, reached at $t = 40$, had a much broader range of $\Delta H$ than the later block morphology, and this is why it was able to escape. The reason for the differences in the $\Delta H$ range between these two block morphologies is the differences in their environment; whereas the earlier block had a non symmetric environment, the later block had the environment fill in around it, with food diffusing in at a stable rate. This demonstrates how structural coupling allows the cell to change its environment in its favor, in a way that increases its viability.

Discussion

Organisms are embedded in environments with which they engage in ongoing structural coupling, and this in part determines their behavioral and developmental trajectories. Here, we investigated the structural coupling of a Potts model cell with a chemical environment, which it consumed and used to maintain a positive target volume. We extended a network-based methodology for analyzing morphological transitions to incorporate environmental state, and showed how this can uncover several features of the Potts cell’s dynamics.

This paper began by examining a Potts cell’s transition probabilities in different static environments, without structural coupling. We saw that the cell’s morphologies can be quantified according to their robustness (probability of remaining at the same morphology), plasticity (probability of transitioning to a different morphology), and death (probability of transitioning to death, for which no lattice site has the cell’s index value), and that these quantities depend on their environment’s state. We also saw that different morphologies are accessible in different environments. The one-step transitions were extended to longer timescales by considering full morphology transition networks. The number of morphologies and the accessible effective energies were...
again shown to depend on the environment.

Next, we included environmental dynamics by introducing diffusion, food growth, and feeding behavior, in which the cell removes chemicals from the environment and uses them to maintain its target volume. This created variation in the cell’s local environment, and since a given morphology’s transition structure depends on environmental state, it became necessary to incorporate the environmental state into the network framework. Thus, we created a morphology-environment transition network to analyze the structure of paired morphological and environmental states. We saw attractors within this network, one corresponding to death and one (near attractor) corresponding to a block morphology with a symmetric environment. We measured viability on this network, and showed that some morphology-environment pairs are much further from death than others, the most viable being the block morphology with a symmetric environment. We also saw how niche construction can increase a given morphology’s robustness and viability: as the cell removed food from its environment, the environment could fill in around it and continually resupply more food. This drove up the effective energies of all possible morphological transitions, and greatly decreased the probability of morphological change, thus stabilizing the morphology-environment state.

In the future, this network methodology for studying structural coupling can be further developed in several ways. One could investigate the network more thoroughly; for example, morphological dependence on the environment can be examined by looking at morphologies that appear many times in the full network, and analyzing how their different local environments shift their transition probabilities. The same can be done for the environment’s dependence on the morphological state. The environment’s influence could be studied beyond the immediate local environment, which only included chemical concentrations within one lattice site of the cell. Structural coupling could be investigated more broadly by running parameter scans of different environmental parameters. For example, the food’s diffusion rate, $D_f$, uptake, $u$, and growth rate, $r_f$, can be reduced to two parameters ($D_f/u$ and $r_f/u$), and a systematic search of the space defined by these parameters could uncover different patterns of structural coupling.

Cellular Potts models take the cell as pre-given, a simplification that allows them to focus on cell-level behaviors and the formation of higher-order structures such as tissues. However, for a complete organismic theory, future analyses of structural coupling will need to address both how cells emerge from a chemistry and how they produce behavior in their environments. Prior research by two of the authors has shown how a minimal protocol cell can emerge from simple chemical dynamics through metabolism-boundary co-construction [2]; this approach can be further extended from the bottom-up to produce behaviors similar to those seen in cellular Potts models. For example, the introduction of sensors and cilia could support the bottom-up generation of chemotaxis.

These analytical techniques provide a foundation for studying structural coupling in more biologically realistic models. Future research could incorporate more complex environments with several chemical species, which could include resources, noxians, and obstacles. Byproducts could be excreted from the cells, which influence the environmental structure. Other cells can be incorporated, which will engage in either cooperative or competitive interactions. Ultimately, multicellular organisms can be studied through the jointly coupled interactions of many specialized cells.

References