Simulating Self-Organization for Multi-Robot Systems

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Abstract

How do multiple robots self-organize into global patterns based on local communications and interactions? This paper describes a theoretical and simulation model called "Digital Hormone Model" (DHM) for such a self-organization task. The model is inspired by two facts: complex biological patterns are results of self-organization of homogenous cells regulated by hormone-like chemical signals [6], and distributed controls can enable self-reconfigurable robots to performance locomotion and reconfiguration [1-3]. The DHM is an integration and generalization of reaction-diffusion model [4] and stochastic cellular automata [19]. The movements of robots (or cells) in DHM are computed not by the Turing's differential equations, nor the Metropolis rule [5], but by stochastic rules that are based on the concentration of hormones in the neighboring space. Experimental results have shown that this model can produce results that match and predict the actual findings in the biological experiments of feather bud formation among uniform skin cells [6]. Furthermore, an extension of this model may be directly applicable to self-organization in multi-robot systems using simulated hormone-like signals.

1. Introduction

This paper[1] is to develop a general computational model for self-organization in multi-robot systems. In particular, we describe the Digital Hormone Model (DHM) that is generalized from an existing distributed control system for self-reconfigurable robots [1-3]. The model is inspired by the fact that many complex patterns in biological systems appear to be the results of self-organization among homogenous cells regulated by hormones, and self-organization is based on local interactions among cells rather than super-imposed and pre-determined global structures [6, 7]. The paper describes the model in detail, reports the experimental results in simulating feather buds formation among homogeneous skin cells, and finds a number of correlations between individual hormone diffusion profiles and the features of final patterns. These results match the findings in the actual biological experiments and predict cases that have yet been observed in biological experiments but consist with the expected behaviors of hormone-regulated self-organization.

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2. Self-Organization in Nature

Self-organization is ubiquitous in nature. It appears in physics, chemistry, materials sciences, and others [12]. But perhaps the richest source for self-organizational phenomena is biological systems. Here, we will describe two of the most fascinating phenomena that are related to self-organization in multi-robot systems: morphallaxis and feather formation.

Morphallaxis is a process by which an organism can regenerate a part or the whole from a fragment by self-reorganization of cells without cell proliferation. This is a process of tissue reorganization observed in many lower animals following severe injury, such as bisection of the animal, and involves the breakdown and reformation of cells, movement of organs, and re-differentiation of tissues. The result is usually a smaller but complete individual, derived entirely from the tissues of part of the original animal. It is believed that such a reorganization process is the most efficient way for simple organisms to self-heal and self-regenerate. This is also extremely important for self-reconfigurable robots to perform self-repair functions. One of the most remarkable examples of morphallaxis is a type of invertebrate freshwater animal called a hydra. If a hydra is cut in half, the head end reconstitutes a new foot, while the basal portion regenerates a new hydranth with mouth and tentacles. Even if a hydra is minced and the pieces scrambled, the fragments grow together and reorganize themselves into a complete whole. How this dramatic self-healing and self-adaptation process takes place is still a mystery.

Another interesting self-organization phenomenon in biological systems is the formation of feathers. In chickens, for example, feathers are developed from skin cells during an early development stage before they hatch from the eggs. Homogeneous skin cells first aggregate and form feather buds that have approximately the same size and space distribution. The feather buds then grow into different types of feathers depending on the region of the skin. Many earlier theories believed that the periodic patterns of the feather buds are formed by sequential propagation and orchestrated by some "key" skin cells. These key cells occupy strategically critical positions on the skin. They first command their neighboring cells to form one sequence of feather buds, and then this sequence propagates to form other sequences of periodic patterns. However, recent findings in biological experiments, as

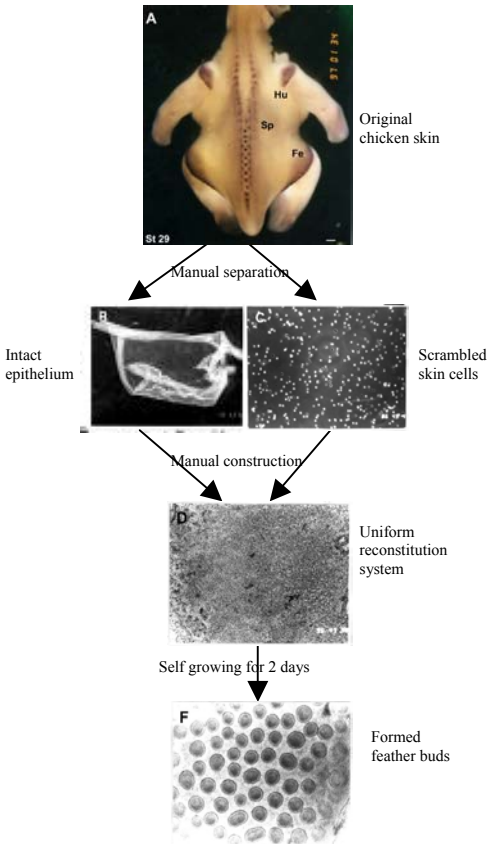

Figure 1: Self-organization in feather formation.

shown in Figure 1, have challenged these theories. Chuong and his colleagues [6, 7] first separated the embryonic chicken skin into a set of disassociated mesenchyme (i.e., skin cells before becoming feather buds) and an intact epithelium (a thin layer on which skin cells can move and aggregate). They then constructed a reconstitution system in which all mesenchymal cells are placed on the epithelium again, but scrambled and reset to an equivalent state so that they have the same probability to become primodia or interprimodia (the feather buds). Surprisingly, the cells in this reconstituted system still grow into patterns of feather buds, and such growth occurs almost simultaneously. These findings uncouple the feather bud pattern formation from the sequential propagations, and they suggest that there are no predetermined molecular addresses, and that the periodic patterning process of feather morphogenesis is likely a self-organizing process based on physical-chemical properties and reactions between homogeneous cells. For robotics research, these results indicate that it is possible for multiple robots to self-organize into interesting global patterns based on local communication and interactions without any pre-determined global patterns or structures.

During these experiments, the biologists also observed some interesting relations among the density of cell population, the individual hormone diffusion profiles, and the size and space distribution of the final patterns. In particular, they observed that while the number of formed feather buds is proportional to the cell population density, the size of the feather buds remains approximately the same regardless of different population densities. The size of the feather buds, however, is related to the diffusion profiles of the activator and inhibitor hormones secreted from the cells. If the concentration ratio of the activator to the inhibitor is high, then the final size of the feather buds will be larger than usual. If the ratio is balanced, then the size of the formed feather buds will be normal. If the ratio is low, then the size of the formed pattern will be smaller than usual. These observations are most interesting to us because they can be used as the basic criteria for evaluating computational models of self-organization in multi-robot systems.

3. Computational Models for Self-Organization

Throughout the history of science, there have been many computational models for self-organization. Perhaps one of the earliest is Turing's reaction-diffusion model [4], in which he analyzed the interplay between the diffusions of reacting species and concluded that their nonlinear interactions could lead to the formation of spatial patterns in their concentrations. Turing's model uses a set of differential equations to model the periodic pattern formation in a ring of discrete cells or continuous tissues that interact with each other through a set of chemicals he called "morphogens." Assuming that there are r = (1,…N) cells in the ring, and two morphogens X and Y among these cells, and letting the concentration of X and Y in cell r be X_r and Y_r, the cell-to-cell diffusion rate of X and Y be u and v, and the increasing rate of X and Y caused by chemical reactions be $f(X,Y)$ and $g(X, Y)$, respectively, Turing modeled the dynamics of this ring as the following set of 2N differential equations:

$$dX_r/dt = f(X_r,Y_r) + u(X_{r+1} - 2X_r + X_{r-1}),$$
$$dY_r/dt = g(X_r,Y_r) + v(Y_{r+1} - 2Y_r + Y_{r-1}).$$

By analyzing the solutions of these equations, Turing illustrated that a given ring of cells, which initially has the uniform concentration of Y and X, can self-organize through random fluctuations, chemical reactions, and diffusion, into a ring of periodic patterns in the concentration of Y. Two important conditions for Turing stability are: (1) between X and Y, one must be the inhibitor and the other activator, and (2) the inhibitor must have a greater diffusion rate than the actuator.

Turing's reaction-diffusion model was startlingly novel, and it has been supported both mathematically [13] and experimentally [14], and many applications are described in [15]. Interestingly, Witkin and Kass [16] extended the traditional reaction-diffusion systems by allowing anisotropic and spatially non-uniform diffusion, as well as multiple competing directions of diffusion. They use these models to synthesize textures with different patterns.

Cellular Automata (CA) [8, 18], especially those that have stochastic characteristics [19], are another important modeling technique for self-organization. Perhaps the most famous illustration of self-organization using CA is the game of Life, where randomly distributed cells on a space

of grids will live or die based on a set of very simple and deterministic rules. Life is a deterministic CA, but when rules of a CA have stochastic characteristics, then they could also be capable of modeling random fluctuations in the environment, and that may be a critical element in simulating interactions among many autonomous elements that perceive and react to local information in the environment. In fact, the Digital Hormone Model to be proposed here is essentially an integration of stochastic CA, reaction-diffusion models, and network-like diffusion space with dynamic topology.

Amorphous computing is another interesting technique that can be potentially useful for modeling self-organization. In an amorphous system, a large number of irregularly placed asynchronous, locally interacting computing elements are coordinated by diffusion-like messages and behave by rules and state markers. These systems have already been applied to building engineered systems for elements to organize and behave in predefined trend [9, 10]. Similar ideas may be directly applicable to self-organization [20], provided that they do not rely solely on the positional information of the self-organizing elements.

Pheromone-based multi-agent systems are also of interest as a tool for studying self-organization. There are already some experimental results [11], showing that a set of autonomous agents can use pheromones to form interesting and complex global behaviors. Such an idea shares many common design principles described here except that pheromones emphasize more diffusion than reaction.

4. The Digital Hormone Model

The Digital Hormone Model (DHM) is designed for simulating, understanding, and controlling self-organization in large-scale multi-robot systems. In this model, robots are simulated as cells that secrete hormones, and hormones diffuse and influence the behaviors of other cells. The Digital Hormone Model consists of a space (we use grids in this paper) and a set of moving cells. The term "cell" here can stand for any type of autonomous and intelligent elements, such as robots, agents, unmanned vehicles, mobile sensors, network nodes, or weapons. Among the grids, cells can live, evolve, migrate, or die as time passes. Each living cell occupies one grid at a time and a cell can secrete chemical hormones (or communication signals in general), which diffuse into its neighboring grids to influence other cells' behaviors. Hormones may have different types and diffusion functions. Two types of hormones are most common: an *activator* hormone that will encourage certain cell actions, while an *inhibitor* hormone will prohibit certain cell actions. We assume that hormones may react to each other (summation, subtraction, or modification), and may diffuse to the neighboring grids according to certain functions. Similar to the extensions used in [16], we allow anisotropic and spatially non-uniform diffusion. Cells are autonomous and intelligent robots that can react to hormones and perform actions such as *migration*, *secretion,* *differentiation*, *proliferation*, *death*, or *adhesion*.

At any given time, a cell selects and executes one or more actions according to a set of internal behavior rules. These rules can be deterministic or probabilistic. We assume that the rules are given and will not cause a cell to select conflicting actions. Given the grids, cells, hormones, actions, and rules, the DHM works as follows:

1. All cells select actions by their behavior rules;
2. All cells execute their selected actions;
3. All grids update the concentration of hormones;
4. Go to Step 1.

To illustrate the above definitions, let us consider a simple DHM_0 in Figure 2, where cells (shown as black dots in the grids) migrate on a space of N^2 grids. The space is a torus in the sense that the leftmost and rightmost columns are neighbors, and the topmost and bottommost rows are neighbors. Cells in DHM_0 have only two actions: secretion and migration, and the former is a constant action that always produces two hormones: the activator A and the inhibitor I. The diffusion rates for A and I secreted from a cell at the grid (a,b) to its surrounding grids (x,y) are characterized by Guassian distributions:

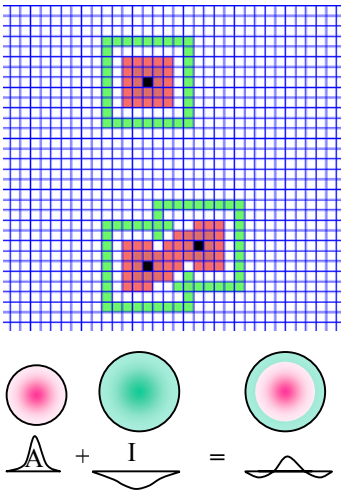

Figure 2: The simple DHM_0

$$f_A(x,y) = (2\pi\sigma^2)^{-1} \exp\{[(x-a)^2+(y-b)^2]/2\sigma^2\}$$

$$f_I(x,y) = -(2\pi\rho^2)^{-1} \exp\{[(x-a)^2+(y-b)^2]/2\rho^2\}$$

where $\sigma < \rho$ in order to satisfy Turing's stability condition. Notice that the activator A has the positive value and the inhibitor I has the negative value. Because $\sigma < \rho$, A has a sharper and narrower distribution then I. We assume that the two hormones react to each other so that the concentration of hormones in any given grid can be computed by summing up all present "A"s and "I"s in the grid. In Figure 2, we have illustrated in the grids the combined hormones around a single cell and around two nearby cells. Since the grids are discrete, the rings around the cells are shown as squares instead of circles.

In this simple model DHM_0, two simple rules govern the cell's actions. One rule states that "secrete A and I for every step", and this means that each cell secretes these hormones at every step. The second rule states that "migrate to an immediate neighbor grid based on the hormone distribution in these neighbors." More specifically, the probability for a cell to migrate to a particular neighboring grid (including the grid it is currently occupying) is proportional to the concentration of A and inversely proportional to the concentration of I in

that grid. This rule is fundamentally stochastic, so that the selection of migrating grid is non-deterministic. To implement this rule, let the hormone value in the occupying grid be h_0 and let the values in the eight immediate neighbors be h_1, h_2, h_3, h_4, h_5, h_6, h_7, and h_8, respectively. Based on their signs, these values are grouped into three groups: G1, G2, and G3, where the members in G1 all have positive values (say sum to P_{G1}), those in G2 have zero values, and those in G3 have negative values. To decide which group to migrate to, a random number x is generated in the range of (0, $100*P_{G1}+10*|G2|+|G3|$]. If $0<x<=100P_{G1}$, then the cell will migrate to G1. If $100P_{G1}<x<=100P_{G1}+10|G2|$, then the cell will migrate to G2. Otherwise, the cell will migrate to G3. The decision ensures that a cell will migrate to G1 with the highest probability, to G2 with lower probability, and to G3 with the lowest probability. After a group is selected, we then select a grid from the group with a similar procedure. For example, to select a grid from G1, a random number will be generated in the range of (0, $h_{i1}+ h_{i2}+ h_{i3}+\ldots+ h_{i|G1|}$], where h_{ij} are individual values in G1 ($h_{ij}>0$), and a grid will be selected depending on where the number falls in the range. This ensures that grids with higher concentrations of the activator hormone will be selected with higher probabilities. To select a grid from G2, we order the grids in the group g_1, g_2, …, $g_{|G2|}$ (note that all these grids have zero hormone values), and a random number y is generated in the range of (0, |G2|], and the grid of g_y is selected. To select a grid from G3, a random number will be generated in the range of (0, $(-h_{j1})^{-1} + (-h_{j2})^{-1} + (-h_{j3})^{-1}+\ldots+ (-h_{j|G3|})^{-1}$], where h_{ij} are individual values in G3 ($h_{ij}<0$), and a grid will be selected depending on where the number falls in this range. This ensures that grids with lower concentrations of the inhibitor hormone will be selected with higher probabilities.

Notice that the above rule for selecting migration direction is different from the Metropolis rules used in simulated annealing [5], which first randomly selects a neighbor without considering the concentration of hormones, and then makes a go or no-go decision based on the energy difference and the current temperature. In the Digital Hormone Model, the notion of temperature is embedded in the decision rules described above. Interestingly, our experiments show that the Metropolis rule does not allow cells to converge into patterns in this model no matter what temperature is set.

Since all movements are local and synchronized, there may be a chance where multiple cells "collide" in the same grid. The collision of cells is solved in a simple manner. All cells first "virtually" move to the grids they selected. If there are multiple cells in the same grid, then the extra cells will be randomly distributed to those immediate neighboring grids that are empty. This is an environmental function, not a cellular action. But this action will ensure that no grid is hosting more than one cell at any time.

5. The Experimental Results of the DHM

Using the digital hormone models, we hope to learn valuable detailed computational knowledge about how hormones and receptors affect the result of self-organization in a large system with many autonomous elements. In particular, the initial research issues we would like to investigate are as follows:

- Will the proposed Digital Hormone Model enable cells to self-organize into patterns at all? Although self-organization has been widely studied by many different models, this is perhaps the first attempt to model mobile intelligent elements that have dynamic structure topology.
- Will the size of final patterns be invariant to the cell population density? Assuming that the cells' hormone diffusion profiles are fixed, will the results match the observations made in the biological experiments?
- Will the hormone diffusion profiles affect the size and shape of the final patterns as shown in the biological experiments?
- Will an arbitrary hormone diffusion profile enable self-organization and pattern formation? In general, how do the profiles affect the results of self-organization process?

To find solutions for these questions, we ran two sets of experiments using the simplified digital hormone model DHM_0 described above. In the first set of experiments, we set the hormone diffusion profile to approximate the standard distributions. For any single isolated cell, let the cell's n^{th} ring of neighbors be the neighboring grids at a distance of n grids away from the cell. Using this definition, we define the concentration level of the activator hormone at the cell's surrounding grids as follows: 0.16 for the 0^{th} ring (i.e., the occupying grid), 0.08 the 1^{st} ring, 0.04 the 2^{nd} ring, 0.02 the 3^{rd} ring, and 0 the 4^{th} and beyond. For the inhibitor hormone, the concentration levels for the 0^{th} through the 4^{th} rings of neighbors are: -0.05, -0.04, -0.03, -0.02, and –0.01, respectively, and 0.0 for the 5^{th} ring and beyond. Thus the combined concentration levels of hormones at the 0^{th} through 4^{th} rings are: 0.11, 0.04, 0.01, 0, and –0.01, respectively, and 0.0 for the 5^{th} ring and beyond. We assume that the concentrations of hormones secreted by a cell at grids beyond the 4^{th} ring are so insignificant that they can be practically ignored.

Given this fixed hormone diffusion profile, we have run a set of simulations on a space of 100x100 grids with different cell population densities ranging from 10% through 50%. Starting with cells randomly distributed on the grids, each simulation runs up to 1,000 action steps, and records the configuration snapshots at steps of 0, 50, 500, and 1,000. As we can see from the results in the upper part of Figure 3, cells in all simulations indeed form clusters with approximately the same size. These results demonstrate that the digital hormone model indeed enables cells to form patterns. Furthermore, the results match the

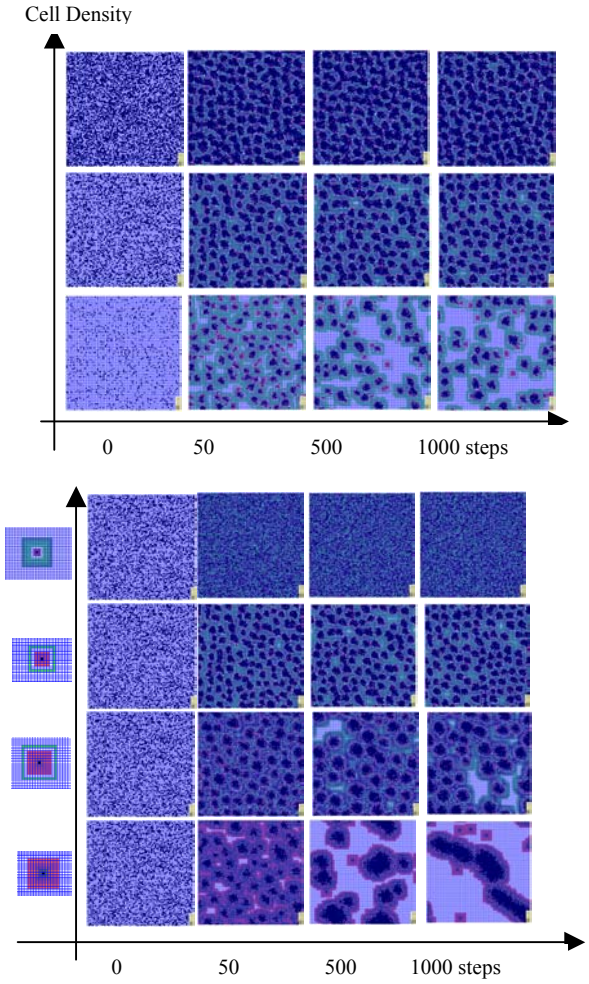

Figure 3: Two sets of experimental results on DHM_0

observations made in the biological experiments. The size of the final clusters does not change with cell population density, but the number of clusters does. Lower cell densities result in fewer final clusters, while higher densities form more clusters.

In the second set of experiments, we started with the same cell population density, but varied the hormone diffusion profiles. We wanted to observe the effects of different hormone profiles on the results of pattern formation. As we can see from the results shown in the lower part of Figure 3, when a balanced profile of activator and inhibitor is given (see the second row), the cells will form final patterns as in the first set of experiments. As the ratio of activator over inhibitor increases (see the third row), the size of final clusters also increases. These results are an exact match with the findings in the reported biological experiments [6].

When the ratio of A/I becomes so high that there are only activators and no inhibitors (see the fourth row), then the cells will form larger and larger clusters, and eventually become a single connected cluster. On the other hand, when the ratio is so low that there is only inhibitor and no activator, then the cells will never form any patterns (see the first row), regardless of how long the simulation runs. This shows that not all hormone profiles enable self-organization. These results are yet to be seen in biological experiments, but they are consistent with the principles of hormone-regulated self-organization and thus qualified as meaningful predictions of cell self-organization by hormones.

The results presented in Figure 3 not only demonstrate that the proposed digital hormone model is indeed an effective tool for simulating and analyzing self-organization phenomena, but that it is also capable of producing results that match the actual findings in the biological experiments and can predict the possible outcomes for new biological experiments. The results show that hormones play a critical role in self-organization, and they enable many autonomous elements to form globally interesting patterns based on only their local information and interactions. This provides a departure point for new hypotheses, theories, and experiments for self-organization. Since the model is mathematically adjustable, it is much more economic and efficient for scientists, including biologists, to design new experiments and to hypothesize new theories.

In addition to changing the ratio of activator and inhibitor hormones, we also have also varied the shape of hormone diffusion profiles and observe their effects on the features of the final patterns. For example, we have observed that if the profile is a narrow and long sandwich with the same orientation (the activator is in the middle and the inhibitors are on the outside), then cells will form striped patterns as shown in Figure 4. This shows that given the proper hormone diffusion profiles, the DHM will allow cells to form patterns with different shapes.

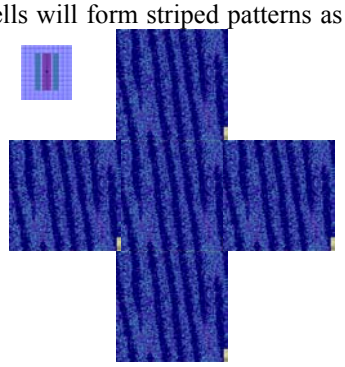

Figure 4: Diffusion profile for stripe patterns.

Furthermore, we have also experimented with different mechanisms for decision making when selecting the migration direction, including the random procedure and the Metropolis rule. Experimental results have shown that Metropolis rule does not enable cells to aggregate into groups no matter what temperature setting is used. This is a bit unexpected, but one possible reason is that Metropolis rule first randomly selects a neighbor without considering the concentration of hormones, and then makes a go or no-go decision based on probability. This does not reflect the true distribution of hormone concentration in the neighboring grids. Similarly, and as expected, the random procedure for selecting migrating directions does not produce any interesting results either.

6. Extend DHM for Multi-Robot Self-Organization

In the above discussion, we have used grids as an approximation for space in which robots can move around. Furthermore, we have assumed that grids can also perform computations to update their hormone concentration. To relax these assumptions and extend DHM for multi-robot self-organization, we must find a way to allow robots themselves to update the hormone-concentration in their surrounding space.

To simulate hormones in grids, we assume robots have wireless communications and can send each other "hormone signals" that carry the necessary information of hormone type and diffusion functions. The strengths and direction of the signal can also be used to calculate the distance and directions between robots, which are the sources of different hormones. Using the information, a multi-robot system can simulate a DHM as follows:

1. All robots select actions by their behavior rules;
2. All robots execute their selected actions;
3. All robots broadcast and receive hormone signals;
4. All robots update the concentration of hormones in their surrounding space;
5. Go to Step 1.

Furthermore, the discrete grids should be generalized into continuous space. Since we have used standard distributions for the hormone diffusion functions, this generalization is not difficult. Instead modeling the neighbors using grids, a robot's neighboring space will be modeled as a continuous circle, with directions ranging from 0.0 to 2π. Thus, robot's moving direction will also take a continuous value. To implement this extension, each robot only requires a compass to specify the continuous direction. The computation in step 3 may be expensive, but we believe a proper and inexpensive algorithm can be designed to estimate the distribution of hormones in a robot's surrounding space without demanding outrageous computational resources.

7. Conclusion

We have presented the Digital Hormone Model (DHM) as a new computational model for self-organization in multi-robot systems. As for future research directions, we will enrich the actions, rules, and hormones in this model, develop the proper algorithms for computing hormone diffusion in continuous space using robots' onboard resources, and simulate larger scale and more complex self-organization phenomena using real robots. We will also develop formal relationships between hormone diffusion profiles and the final global patterns, and investigate the strengths and limitations of DHM for self-organization in general.

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