Self-repairing and Mobility of a Simple Cell Model

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Abstract

The evolution of a mobile cell system is studied here. While a primary definition of life is self-reproduction, mobility is also a major characteristic. Typically, most alife models assume cell mobility a priori. Here, using a development of a cell model due to Varela and McMullin, we show the emergence of cell mobility. A balance between cell repair and mobility is demonstrated. The introduction of a new functional particle is a critical part of the present model.

Introduction

Self-reproduction, one of the primary characteristics of living systems, has been studied both experimentally and theoretically for more than 40 years. Initially, von Neumann showed the existence of a universal constructor in a 2-dimensional cellular automaton with 29 cell states (Neumann, 1966). More recently, Langton, Tempesti, Morita and Imai, Sayama, Suzuki and Ikegami have demonstrated the dynamics of self-replicators with special rule sets (Langton, 1984; Tempesti, 1995; Morita and Imai, 1997; Sayama, 1999; Suzuki and Ikegami, 2003). Although in these models self-replication occurs successfully, the question of how the underlying physical chemistry could support such specific symbolic rule sets has not received much attention. Recently, a series of studies by Rasmussen, McCaskill, Ono and Ikegami and others have indeed shown that simple catalytic networks can develop cellular membranes and self-reproduction processes(Mayer et al., 1997; Breyer et al., 1998; Ono and Ikegami, 1999; Ono and Ikegami, 2000). Self-reproduction in 3-dimensional space has also been demonstrated by extending Ono's model(Madina et al., 2003).

What has not been achieved so far is self-mobility; a unit that acquires mobility in some physical space. In this paper, we will study the origin of mobility in a cell system based on a simple discrete cell model, the so-called SCL model which was introduced by Varela(Varela et al., 1974) and reformulated by McMullin(McMullin and Varela, 1997). Varela used the model as a practical example of autopoiesis, a principle of biological autonomy common to all living things. An autopoietic system is organized as a network of processes that composes the system. Each process recreates the other processes which together comprise a coherent unit in some physical domain. Indeed, Varela claims that autopoiesis is a necessary and sufficient condition for what we call life (Varela, 1979). A cell that generates its own boundary to enclose a catalyst which in turn sustains the boundary is a simple example of autopoietic organization.

However, we believe that a new principle is required to understand the dynamics and evolution of living systems. Previous cellular models of autopoiesis stressed the notion of autonomous self-maintenance. Here, we stress the notion of motility by extending the SCL model. In the original model, components of the cell are used for maintaining the cell boundary. Here we also employ them for cell movement. We introduce a new kind of membrane component, a functional LINK particle. We use the term functional as these particles work both to repair membranes and move cells.

The Model

We use a modification of the "Substrate-Catalyst-Link" (SCL) model originally introduced by Valera to explain the idea of autopoiesis. Before introducing our modifications, we first explain how the original model works.

The SCL model consists of three kinds of particles which reside on a square lattice. They are called "SUBSTRATE" "CATALYST" and "LINK" particles. They all diffuse freely in the space at given constant rates. If two SUBSTRATE particles are in the Moore neighborhood of a CATALYST particle, they disappear and a single LINK particle is formed in place of one of them. LINK particles neighboring each other are bonded. Each LINK particle is allowed to bond with at most two other LINK particles, so that the bonding tends to form a linear chain. A quasi-stable structure is a closed loop of bonds. SUBSTRATE particles can diffuse freely through the loop, while CATALYST particles cannot.

Each LINK particle, whether bonded or not, decays into two SUBSTRATE particles at a certain rate. A loop of LINK particles may be regarded as a proto-membrane, as the loop

is maintained by the constant production of LINK particles via a CATALYST. Thus, we may refer to such a loop enclosing a catalyst as a cell unit.

In order to add mobil

ity to the model, first we introduce some restrictions in the bonding of free LINKs. In the original bonding rule, the catalyst's movement leaves some fragments of bonded LINKs, which disturb the maintenance of valid membrane structure.

One rule already introduced in the original model was that a free or single bonded LINK particle cannot bond with other LINKs when double bonded LINK exists in the vicinity. New restrictions we introduce here are:

RULE A Bonded Links may decay to two Substrate as long as there does not exist free LINK particles in either of the bonded LINK particles's Moore neightborhoods.

RULE B A bond between two LINK particles break in the Moore neighborhood of a CATALYST.

RULE C A bond between two LINK particles break when free LINK particles exist on *both* halves of the Moor neighbors as divided by the bond.

Rule A, B are to assist cell movement as will be discussed below and Rule C is introduced to suppress entanglement of bonded loops.

The main feature of our new SCL model is the introduction of a new LINK particle, which we call "functional LINK" particles (Fig. 1). As this functional LINK particle can actively push a catalyst when it is in the catalyst's neighborhood, catalysts can move. Also, functional LINKs in cell membranes let substrate particles pass through the membrane. The other, normal LINK particles do not. These new functional LINK particles are produced when a catalyst is surrounded by a sufficient number of substrates. Namely, we have:

RULE D Two substrate particles in the Moore neighborhood of a catalyst may form a functional LINK particle if the total number of substrates in the region is above a given threshold V. Otherwise, normal LINK particles are produced.

Considering the Moore neighborhood of a catalyst particle, we compute the number functional LINKs in the North, South, East and West regions. Each side contains at most 3 particles. With these numbers, we compute the following probability for the direction (North, South, East and West) in which the catalyst will move.

$$P_k(i) = \alpha \cdot \frac{\exp(\beta N_k(i))}{\sum_{all\ l} \exp(\beta N_l(i))} \qquad (1)$$

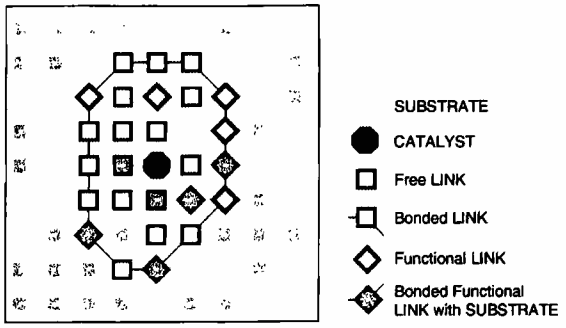

Figure 1: An example of cell formation in our model. A catalyst, surrounded by free LINK particles, is enclosed by the membrane (i.e. a closed loop of two kinds of bonded LINK particles). SUBSTRATE particles can only cross the membrane where functional LINK particles exist. Inside the membrane, SUBSTRATE particles can occupy the same sites as LINK particles.

This is the probability of the catalyst at the i-th site moving in the k-th direction. N_k is the number of the functional LINKs in the opposite side of the k-th direction. If another particle except the bonded LINK already exists in the catalyst's destination, we check to see if there is room for the other particle to move in the same direction, to make room for the catalyst. If there is room, both the catalyst and the other particle move. We say that the catalyst "pushes" the other particle. However, if there is not room for the other particle to move, the catalyst and the other particle exchange positions. This behavior is already a feature of Varela's original model. We take $\alpha = 0.05$, $\beta = 3.0$ in the following simulations.

In contrast to the original model, substrate particles can only cross a boundary of bonded LINK particles at the functional LINK particles. The more that a boundary is composed of functional LINK particles, the more substrates may cross the boundary. Therefore, where and how many functional LINK particles are embedded in the boundary determines where and how many substrate particles may enter or leave the cell.

Observation

There are some unique phenomena observed in our model. First, we describe these before analyzing them. In our simulations, we use the fixed parameters shown in Table 1. In addition, we allocate sufficient grid space for the movement of the cell.

Movement of the Cell

As Varela's model shows, simply establishing a self-sustaining unit just repairs itself, but does not change otherwise. However, in the original model, if any cell movement occurs, the boundary is easily damaged and as a result,

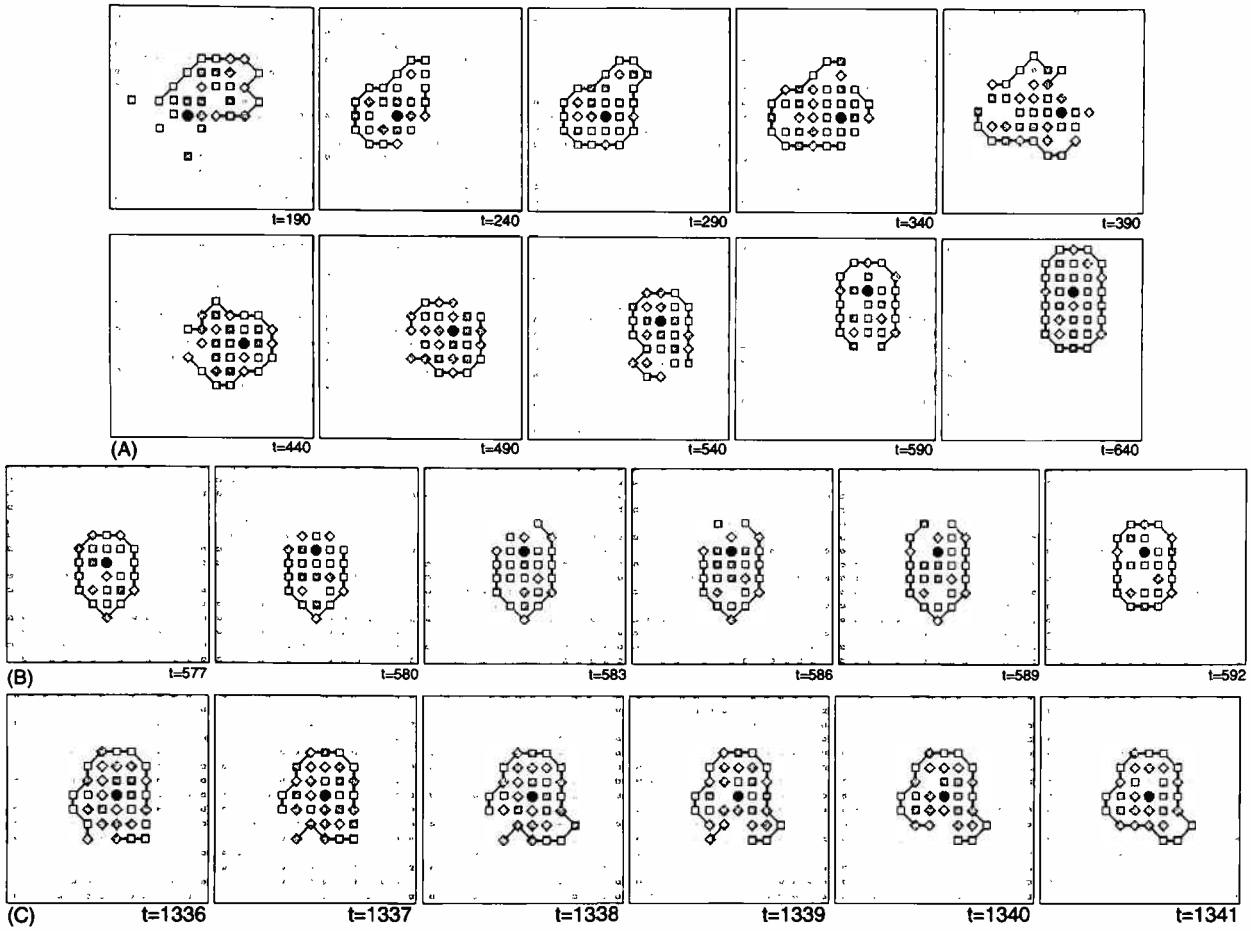

Figure 2: Examples of proto-cell behavior: (a) movement of a cell over a long period of time. The cell moves to the upper right corner while repairing its boundary. (b) The cell motion process in detail. Within 15 steps, the cell as a whole is moved in the upwards direction. (c) Repairing of the cell boundary with free LINK particles. Due to Rule A, bonded-links release LINK particles frequently and holes are created in the membrane. These holes are rapidly restored by the neighboring free LINK particles.

whole-cell movement cannot be observed. But in our model, the stability of the boundary seems much stronger because Rule C suppresses a formation of irregular structures of bonded LINKs such as a spiral structure, with which bonded LINKs does not enclose CATALYST. The cell is able to move around, continuously repairing its boundary(Fig. 2a). Indeed, the motion of a cell requires continuous repairing of the boundary. This happens as follows:

1. A catalyst moves into the vicinity of a boundary.
2. Due to the Rule B, a boundary particle in the vicinity of a catalyst disintegrates with a relatively high rate.
3. A substrate (often a functional LINK) will be inserted as a new component of the boundary.
4. Other parts of the boundary will be naturally replaced by LINK particles due to Rule A.

By both continually recreating the part of the boundary close to the catalyst and repairing the part of the boundary further away, a cell can gradually move as a whole(Fig. 2b). The addition of the new functional LINK particle is crucial to this ability. Fig. 3 shows the spatial trail of a cell's movement by tracing the position of its CATALYST for 10,000 time steps.

Such a stable dynamics is sustained by the result of a autopoietic organization. Bonded LINKs are frequently disconnected by Rule A when no free LINK particles exist in the vicinity. This event leads to the formation of holes in the middle of a membrane. However, these holes are immediately repaired by free LINKs near the hole. This suppresses undesirable irregular formation of bonded-LINKs. This repairing process is illustrated in Fig. 2c. Generally, a cell maitains the number of each LINK particles for hundreds of time steps due to the rapid repairing processes. But sometimes, a cell decays and reforms with very different compo-

Parameter	Values
the initial substrate density	0.6
the decay rate of LINK particles	0.003
the diffusion rate of CATALYST	0.01
the diffusion rate of LINK	0.3
the diffusion rate of SUBSTRATE	1.0
free-link-bond-restriction (Rule A)	0.5
opposite-side-link-bond-restriction (Rule B)	1.0
decay-with-catalyst (Rule C)	1.0
The threshold of functional LINK production V	5

Table 1: The parameters used in our simulations

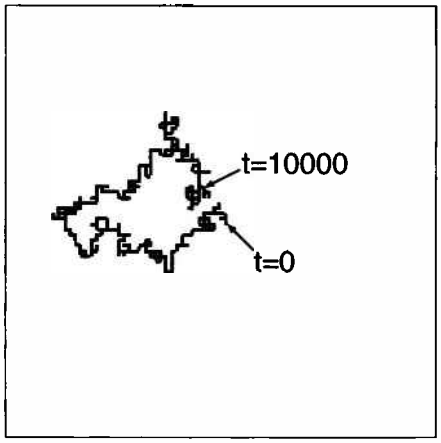

Figure 3: A spatial trail of a cell's catalyst after 10,000 steps on a 72x72 cell space.

nents.

Dynamic Processes

A functional LINK particle is generated from two substrates by Rule D only when a catalyst is surrounded by enough substrates. Since the substrates can only cross the cell boundary where the functional LINK particles are present, a cell in a poor environment (i.e. one with a low density of substrate particles) can only generate normal LINK particles. Since normal LINK particles cannot penetrate the boundary, the production of LINK particles is significantly suppressed. As a result, few repairing processes occur and the cell unit diminishes. Therefore, there exists a critical density of substrates to create a mobile cell structure (see Fig.4). We found that the critical density is around 0.5, where a membrane begins to last for significantly longer periods of time than with a substrate density of 0.4. It is worth noting that the mobility of a catalyst is accelerated by the formation of a membrane. The maximum speed of catalyst mobility is found when the substrate density is around 0.7. This shows that mobility requires a synchronous repairing process. As repairing and mobility processes are complementary to each other, a perfect repairing process suppresses mobility. Therefore in rich environments above density 0.7, the mobility is again significantly suppressed.

When a cell has a sufficient number of functional LINKs in its boundary, more substrates are accumulated inside the cell. When enough substrates are found around a catalyst, more functional LINK particles are generated. Indeed, we observe that the fraction of functional LINK particles in a cell increases as time proceeds(see Fig. 5). If a cell has a boundary composed only of functional LINK particles, the catalyst constantly generates excess functional LINK particles. Therefore any breakage of the boundary is immediately repaired by the functional LINK particles. However, if there are many ppfunctional LINK particles surrounding a catalyst, there is no free space and so the cell can only move when these particles decay. Such a cell unit is the most stable one. However, a cell typically breaks up before reaching this state.

In order to investigate the disorder of cell movement, we compared the deviation of the CATALYST movement in three cases: the model with only normal LINK particles (like the original model), the model with functional LINK particles but where SUBSTRATEs may pass through the nomal LINKs (that is, functional LINKs act only as "motors"), and the complete model with functional LINKs as already described above (Fig. 6). Cell units with fully functional LINK particles ("motor" and "sensor") show deviate significantly from random walking after around 100 time steps, while the lifetime of a cell is several hundred time steps. Therefore, it can be said that a cell can move directionally for a certain period of time until a reconfiguration of internal LINK particles occurs.

The Functional LINK Particle as a "Sensor"

Functional LINK particles on the membrane act as protosensors of a cell system, since substrates can across the membrane only via the functional LINKs. The substrates that come across the membrane will compose either normal or functional LINK particles. A part of functional LINK stays around the catalyst to push it. Therefore, functional LINK gives a way of coupling inputs and motor outputs. External substrates are detected by the functional LINK on the boundary. The absorbed substrate will compose LINK particle as described. Then the final displacement of a catalyst is determined by the cooperative effect of functional LINK particles.

This cooperative effect has some time delays. In Fig.7, we computed the time correlation between inputs of SUBSTRATE with displacement of CATALYST $< S(t)D(t+\tau) >$ and repairing a membrane $< S(t)R(t+\tau) >$. Here, $S(t)$ is the number of substrate through the membrane. $D(t)$ is a moving average with width 40 of the moving events of the CATALYST. Similarly, $R(t)$ is a width-40 moving average of the number of discrete double bonding events which only

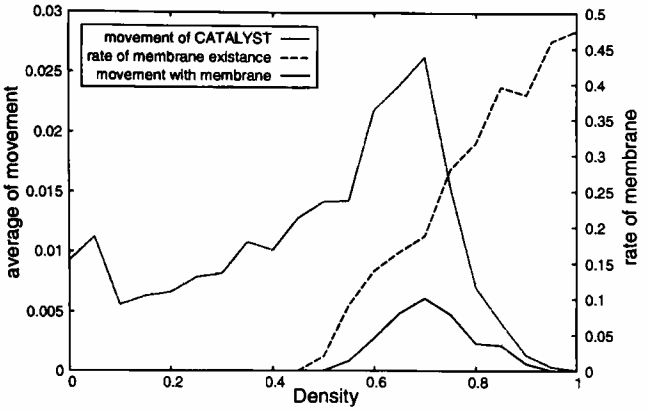

Figure 4: Average values of the displacement of the CATALYST, membrane existence time and displacement of the CATALYST when a membrane exists are computed for a range of initial substrate densities. The lower substrate densities cannot support membrane formation. A mobile cell only appears when the density is around 0.6-0.9. It is worth noting that the speed of movement is increased by membrane formation.

occur repairing a membrane.

Comparing with the two peaks, we estimate that the substrate input is used as a repairing output around 5 time steps and as a motor output around 10 time steps. As the latter correlation lasts longer, it suggests that some functional LINK particles are accumulated a few dozen steps in a cell being recruited for repairing or pushing. Combining the present result, we can determine some primitive form of diffusive coupling between the inputs and motor output.

Discussion

Varela's original cell model demonstrates cells that keep their identity whilst maintaining their own boundaries. Varela noted, however, that living autonomous systems must do more than this; they should not only keep their identity but at the same time couple with their environment(Varela, 1979). In his model, a coupling between the cell system and its environment was not shown explicitly. The present model is a first realization of such coupling, with the membrane as an input channel and movement of whole cell units.

Shifting our focus from self-reproduction to self-motility, we presented some necessary conditions for acquiring motility in a self-sustainable cell system. Such a system has two complementary aspects: movement and repair. By introducing a new type of LINK particle, the functional LINK particle, we can treat sensors and active movement of a cell system in a same level.

We consider it is very effective for discussing a origin of sensor-motor-coupling. Generally, sensory-motor coupling is discussed in artificial neural system with navigation.

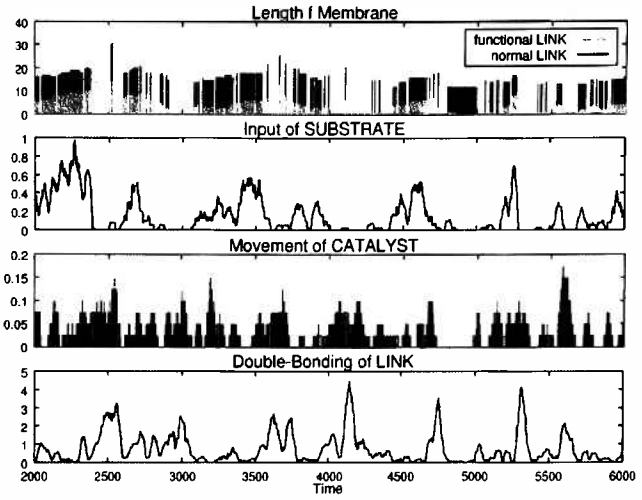

Figure 5: Time evolution of the length of membranes, the rate of substrate input, the catalyst displacement and the rate of double bonding events. The length of membranes is defined whenever the bonded LINK particles form a closed loop. The percentage of functional LINKs of the membrane increases during each cell lifetime. The substrate input is detected only when membrane is closed. The value increases when the membrane lives long enough. The movement of the catalyst may be computed regardless of the state of membrane formation. The double-bonding event corresponds to repairing of membrane formation. This occurs when the membrane diminishes.

There are sensory inputs and motor outputs connected to a separate controller. A primitive life system could not have such sophisticated neural connections. Indeed, insects and protozoa use internal chemical networks to make sensor-motor couplings. In our model, functional LINK particles bifurcate into sensors on a cell boundary and engines giving a catalyst motility. The coupling between these is still ambiguous, however some adequate evolutionary pressure may strengthen this coupling to make it more reliable. An extension of our model in this direction will be reported elsewhere.

Acknowledgments

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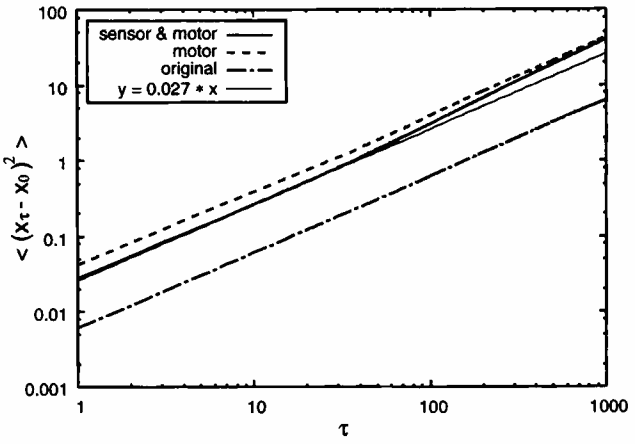

Figure 6: deviations of the displacement of the CATALYST of three cases: the original model with three bonding restriction(no functional LINK), the model with functinal LINK as only a "motor" not "sensor"(SUBSTRATE can penetrate normal LINK), and the model with functional LINK as both "motor" and "sensor"(full role of functional LINK). a thin line is a $y = 0.027x$. only the model with full role of functional LINK shows a declination from a random walk from 100 to 1000 time steps.

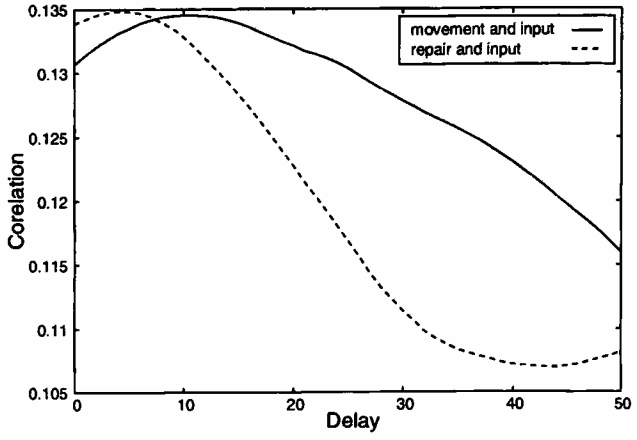

Figure 7: Two Time correlation functions are computed. The first one is between the displacement of catalyst and substrate flows through the membrane (solid line). The second one is between the repairing events and the substrate flows (segmented line).

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