

## SPIRAL WAVE STRUCTURE IN PRE-BIOTIC EVOLUTION: HYPERCYCLES STABLE AGAINST PARASITES\*

M.C. BOERLIJST and P. HOGEWEG

*Bioinformatica, Padualaan 8, 3584 CH Utrecht, The Netherlands*

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In the study of pre-biotic evolution self-structuring and evolutionary processes are themes that are usually studied separately. In this paper we demonstrate that spatial self-structuring in incompletely mixed media can profoundly change the outcome of evolutionary processes; for instance, positive selection for “altruistic” features becomes feasible.

A spatial cellular automaton model of self-replicative molecules that are linked cyclically by catalysis (i.e. a hypercycle) was studied. Because of the spiral structures that emerge in such a system, the hypercycle becomes resistant to a large class of “parasites”, i.e. molecules that give no catalytic support but receive increased catalytic support.

### 1. Introduction

Accumulation of information is a central issue in pre-biotic evolution. Eigen and Schuster [1] were the first to stress the existence of the information threshold; in a system containing self-replicative molecules the length of the molecules is restricted by the accuracy of replication. In their hypercycle theory Eigen and Schuster state that if the information threshold is to be crossed a number of molecules have to catalyse the replication of each other in a cyclic way (see appendix A, fig. 3). This so-called hypercycle has great selective advantages. Each molecule in the hypercycle is still bound to the maximum string-length, but the molecules can combine their information and thus cross the information threshold.

An important objection to the hypercycle theory has been raised by Maynard Smith [2]: because there is no selection for the giving of catalytic support to the replication of another molecule, this property cannot be maintained.

Giving catalytic support is in fact an “altruistic” property, i.e. it does not raise the number of copies of the molecule itself, but it does increase the number of copies of another (competing) species.

As a result a hypercycle is vulnerable to so-called “parasites”. Fig. 4 (see appendix A) shows a hypercycle with a parasite. The parasite is capable of self-replication; it gets catalytic support from species 2 but does not give catalytic support to any other molecule. If the support the parasite gets from species 2 is greater than the support species 3 gets from 2 (i.e.  $k_{\text{par}} > k_3$ ), the parasite will be selected in favour of species 3 and the entire hypercycle will be lost. There seems to be a large class of parasites that are fatal to hypercycles, i.e. hypercycles are evolutionarily unstable.

Up to now the hypercycle theory was formulated and studied in terms of ordinary differential equations. This model formalism implies an ideally mixed medium. Of course it is more realistic to assume incomplete mixing of the medium. In this study we will formulate a model in which molecules with hypercyclic interactions are embedded in an incompletely mixed medium. Such a spatial hypercycle system generates large scale

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spiral structures. We will show that this spatial self-structuring yields the hypercycle resistant to (many) parasites.

## 2. The model

In order to simulate incomplete mixing we use the model formalism of the cellular automata [3]. A cellular automaton is defined as a large tessellation of identical finite state automata (cells). Each automaton is defined as a triplet:  $\langle I, S, W \rangle$ , where  $I$  is the set of inputs,  $S$  is the set of states (both sets being finite and usually small), and  $W$  is the next-state function, defined on input-state pairs. The inputs are the states of "neighbour" cells, i.e. the adjacent cells in the tessellation. Cellular automata have proved to be a powerful tool in the study of spatial processes, for instance fluid dynamics [3–5].

In our cellular automaton the total space consists of  $300 \times 300$  cells in a square toroidal tessellation. The state of a cell refers either to its occupation by a molecule of a certain species or to its emptiness, i.e. a cell can contain one

molecule or it can be empty. In the next-state function of the cells (see table 1) we implement a representation of three processes, namely decay, replication and catalysis of molecules:

(1) *Decay* (see fig. 1A) can occur when a cell is occupied; after decay the cell becomes empty. The probability of decay is species (i.e. state) dependent.

(2) *Replication* (see fig. 4B) is only possible in empty cells; a molecule in one of the four direct neighbour cells can replicate into the empty cell. The probability of replication is species dependent. There is also a probability that the cell will remain empty.

(3) *Catalysis* (see fig. 4C) is related to replication; the probability that a molecule will replicate into an empty cell is increased when there are catalytic molecules in at least one of the four cells that lie adjacent to the direction of replication.

In addition to these three processes *diffusion* is included in our model as a separate process, operating in between "time steps". We use the diffusion algorithm of Toffoli and Margolus [3], which ensures particle conservation. In this algorithm the space is divided into subfields of  $2 \times 2$

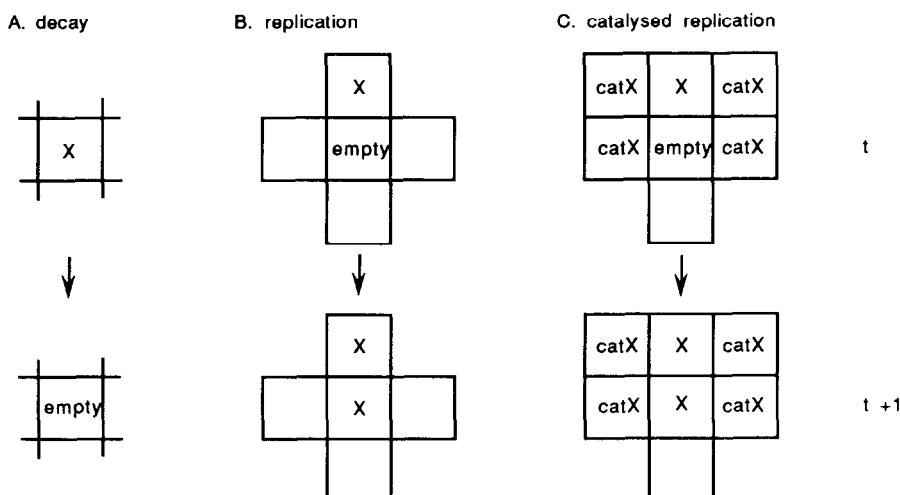


Fig. 1. Three state transitions of a cell in a cellular automaton in which hypercycles can be simulated. The next state  $t + 1$  is drawn below the present state  $t$ ; X is a molecule of a certain species; catX is a molecule that catalyses molecule X's replication. See text for further explanation.

Table 1

Next state probability function  $p(\cdot)$  of a cellular automaton in which hypercycles can be simulated. Explanation of some terms:  $\text{decay}[x]$  is the decay parameter of molecule  $x$ ;  $\text{self}[x]$  is the replication parameter of  $x$ ;  $c[x, y]$  is the catalytic support  $x$  gets from  $y$ ;  $\text{Claim}_{\text{empty}}$  is a constant, which is set to 11; N(orth), S, W, E, NW, NE, SW, SE are the states of the eight neighbour cells indicated by their compass direction.

If the cell is occupied by a molecule $x$ :
$p(\text{empty}) = \text{decay}[x];$
$p(x) = 1 - \text{decay}[x];$
If the cell is empty:
$p(\text{empty}) = \text{Claim}_{\text{empty}}/\sum \text{Claims}$ (no replication);
$p(N) = \text{Claim}_N/\sum \text{Claims};$
$p(S) = \text{Claim}_S/\sum \text{Claims};$
$p(W) = \text{Claim}_W/\sum \text{Claims};$
$p(E) = \text{Claim}_E/\sum \text{Claims};$
In which:
$\sum \text{Claims} = \text{Claim}_{\text{empty}} + \text{Claim}_N + \text{Claim}_S + \text{Claim}_W + \text{Claim}_E;$
$\text{Claim}_N = \text{self}[N] + c[N, NE] + c[N, NW] + c[N, E] + c[N, W];$
$\text{Claim}_S = \text{self}[S] + c[S, SE] + c[S, SW] + c[S, E] + c[S, W];$
$\text{Claim}_W = \text{self}[W] + c[W, NW] + c[W, SW] + c[W, N] + c[W, S];$
$\text{Claim}_E = \text{self}[E] + c[E, NE] + c[E, SE] + c[E, N] + c[E, S];$

cells. At each diffusion step the states of a sub-field are rotated 90° clockwise or anti-clockwise with equal probability. After a diffusion step the subfields are shifted one cell diagonally.

Note that this cellular automaton is a straightforward representation of the physical process modelled (see ref. [6]), i.e. catalysis and replication of molecules. Partial differential equations could be constructed which simulate this cellular automaton; such a reformulation, however, would introduce extra, ad hoc, parameters.

### 3. Results

#### 3.1. Development of spirals

First we study the spatial behaviour of a set of molecules which are part of a pre-defined hypercycle. In plate 1A the nine members of this hypercycle are distributed at random in the space; 50% of the cells are empty. The nine molecule species have identical replication and decay parameters (table 1:  $\text{self}[1..9] = 1$ ;  $\text{decay}[1..9] = 0.2$ ). Each species catalyses one other member of the

hypercycle; catalysed replication is much stronger than non-catalysed replication (table 1:  $c[2, 1] = c[3, 2] = \dots = c[1, 9] = 100$ ). After each time step there are two diffusion steps.

Plate 1B shows the situation after 500 time steps. Spiral structures have developed containing all members of the hypercycle in catalytic order. Each species grows towards its catalytic supporter; species 2 (red) grows towards species 1 (purple), species 3 (orange) grows towards species 2 and so on. As a result of this directional growth the spirals rotate.

Plate 1C shows the situation after 1500 time steps. Most spirals occur in couples; a spiral rotating clockwise is close to a spiral rotating anticlockwise. Some spirals have disappeared. This happens when two spirals rotating in the opposite direction come too close to one another. The number of molecules of a species between the two spirals is then reduced. If by chance a species dies out, then temporarily the species that gives catalytic support to the extinct species takes over the complete region of the double spiral. Because this species now no longer gets catalytic support, the region formerly occupied by the double spiral is taken over by other nearby spirals.

After 1500 time steps the pattern remains constant: the centres of the spirals do not move and all spirals have the same rotation time, which in this case is approximately 110 time steps.

The middle of a spiral acts as a centre of growth for the entire spiral. This is demonstrated in plates 2A–2C. As a starting pattern we use the situation of plate 7A; a stabilized pattern of a hypercycle that consists of 6 members. In plate 2A the molecules in the middle and the periphery of the huge single spiral are labelled (only the labelled molecules are coloured; each colour represents three molecule species that are adjacent in the hypercycle). In plate 2B, after 30 time steps, the descendants of the labelled molecules in the periphery have reached the edge of the region of the spiral. The labelled molecules in the middle of the spiral have increased in number. After 200 time steps (see plate 2C) the molecules from the middle have taken over the complete spiral region and the molecules from the periphery have disappeared. This direction of growth is caused by the catalytic waves that travel from the middle towards the periphery of the spiral. Note that although the spirals rotate, growth is not rotational.

Thus in this incompletely mixed medium, cyclic catalysis has remarkable self-structuring properties: from a random start a stable situation with interlocking rotating spirals emerges. The spirals are often in couples and the middle of a spiral is the centre of growth of the entire spiral. This spatial self-structuring changes the selectional properties of the hypercycle. In this study we will focus on one specific property, namely the vulnerability of a hypercycle to parasites.

### 3.2. Stability against parasites

We infected the situation of plate 1C randomly with 100 “deadly” parasites. The parasite has the same replication and decay parameter as molecules in the hypercycle. It gets catalytic support from species 2 (red); this catalytic support is twice as strong as the support that species 3 gets

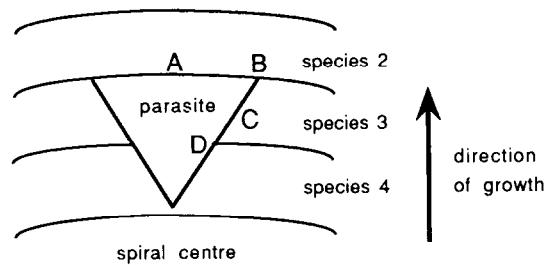


Fig. 2. Schematic diagram of a growing parasite. The parasite gets catalytic support from species 2. See text for further explanation.

from species 2 (table 1:  $c[\text{parasite}, 2] = 200$ ). The parasite does not give catalytic support to any member of the hypercycle. Plates 3A–3C show the situation after 30, 60 and 110 time steps. The parasite starts to grow in regions where it is near its catalytic supporter, species 2. However, the parasite grows in the direction of the periphery of the spirals. Eventually, at the boundaries of the spirals the parasite is wiped out; after 160 time steps the parasites have vanished completely.

Why does a parasite grow towards the periphery of a spiral? In fact, the parasite simply follows the general direction of growth in a spiral, as mentioned in the previous section, although it is not “pushed” outwards by a molecule that it catalyses, because it does not give catalysis. Let us take a closer look at what happens in a parasite region. In fig. 2 a parasite region is shown schematically. Four types of competition take place, all in different subregions:

(i) In subregion A the parasite competes with species 2. The parasite gets catalytic support from species 2 and is therefore far stronger than species 2.

(ii) In subregion B the parasite competes with species 3. Both get catalytic support from species 2. The parasite gets stronger support; it gains ground over species 3.

(iii) In subregion C the parasite also competes with species 3. In this subregion no catalytic support is given to either species; the competition is neutral.

On the next two pages the following colour plates are shown:

Plate 1. Development of a spiral wave pattern in a spatial hypercycle system. Colour molecule species: (1) purple; (2) red; (3) orange; (4) yellow; (5) dark green; (6) light green; (7) light blue; (8) middle blue; (9) dark blue. In order to increase contrast we do not show the state of a cell, but instead we show the majority molecule species in the 9-cell neighbourhood of the cell. If all 9 cells are empty the cell is white. (A)  $t = 0$ , random initialization; (B)  $t = 500$ ; (C)  $t = 1500$ . See text for further explanation.

Plate 2. Direction of growth within a spiral. Molecules in the middle and the periphery of a spiral are labelled. Plate 7A is used as a starting pattern. (A)  $t = 0$ , starting pattern with labelling; (B)  $t = 30$ ; (C)  $t = 200$ .

Plate 3. Parasite invasion. The situation of plate 1C is infected randomly with 100 parasites. If there is one parasite in the 9-cell neighbourhood of a cell, the cell is black. For other colours see plate 1. The parasite first starts to grow, but eventually it is wiped out completely and the spiral pattern is restored. (A)  $t = 30$ ; (B)  $t = 60$ ; (C)  $t = 110$ .

Plate 4. Parasite invasion in the centre of a double spiral. First the region of the double spiral is taken over by the parasite, but eventually the parasite is wiped out by other spirals. Colours as in plate 3. (A)  $t = 110$ ; (B)  $t = 550$ .

Plate 5. Parasite invasion in the centre of a single spiral. The parasite remains present as a cyst. Colours as in plate 3.  $t = 600$ .

Plate 6. Reinforcement of a spiral. In the situation of plate 7A all molecules that are not close to the centre of a single spiral are removed. The single spiral in the middle of the field is removed completely. The removed spiral reappears, although it is much smaller and not exactly at the same spot. (A)  $t = 0$ , situation after removal; (B)  $t = 60$ ; (C)  $t = 300$ .

Plate 7. The effect of diffusion. (A)  $t = 2000$ , 2 diffusion steps after every time step; (B)  $t = 500$ , no diffusion; (C)  $t = 500$ , 16 diffusion steps after every time step.

(iv) In subregion D the parasite competes with species 4. Although the parasite does not give catalytic support to species 4, there will always be some molecules of species 3 in the subregion (i.e. due to diffusion) which do give catalytic support to species 4. This causes species 4 to be far stronger than the parasite.

In summary, the parasite wins in subregions A and B. This causes it to grow towards the periphery of the spiral. The parasite loses in subregion D. This leads to a narrowing of the parasite region. If the parasite is not completely eliminated by the catalytic wave of species 4, then it is eliminated by the catalytic wave of species 5. Only after seven catalytic waves does the parasite again meet species 2 and only then can it grow towards the middle of the spiral. This growth, however, does not compensate for the loss incurred through the catalytic waves, so the parasite grows towards the periphery of a spiral.

### 3.3. Parasite invasion in the middle of a spiral

The inability of a parasite to grow towards the middle of a spiral explains the sequence in plates 3A–3C, but what happens if a parasite invades right in the middle of a spiral? In plate 4A such an event has taken place: a single parasite has been introduced into the middle of a spiral and after 110 time steps the parasite has taken over the complete domain of a double spiral. However, by doing this it has destroyed the double spiral, and now it has to compete with other spirals. We already know that a spiral gains ground over a parasite, so in plate 4B after 550 time steps the parasite invasion is wiped out completely and the region of the “killed” double spiral is taken over by other (double) spirals.

There is a second possible result of a parasite hitting the middle of a spiral. An example is shown in plate 5: the parasite has been intro-

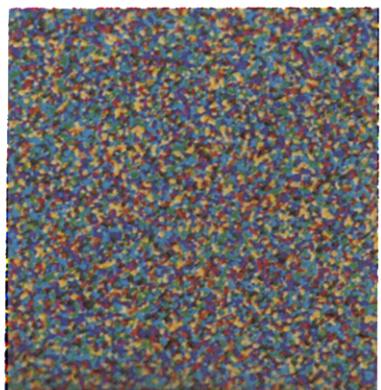


Plate 1A

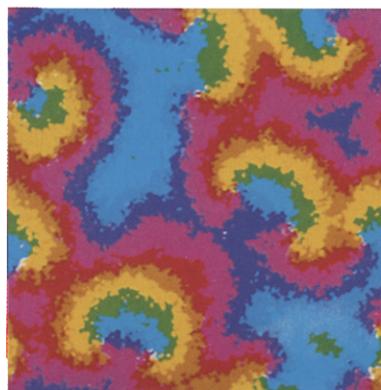


Plate 1B

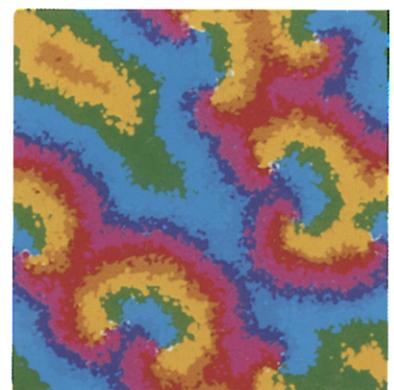


Plate 1C

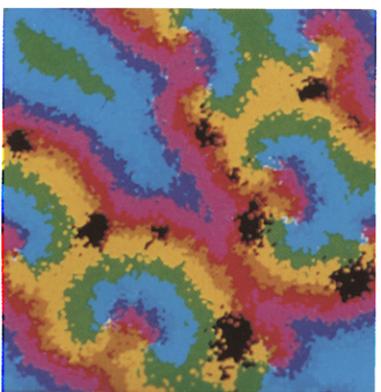


Plate 3A

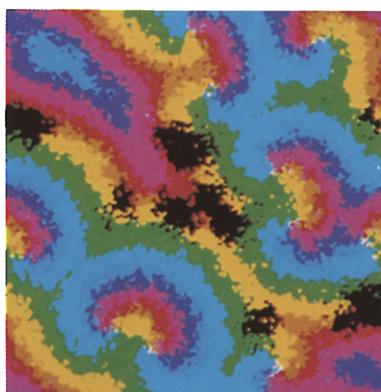


Plate 3B

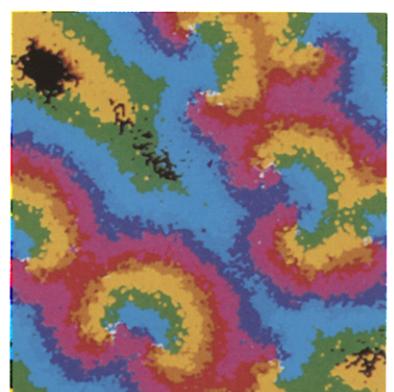


Plate 3C

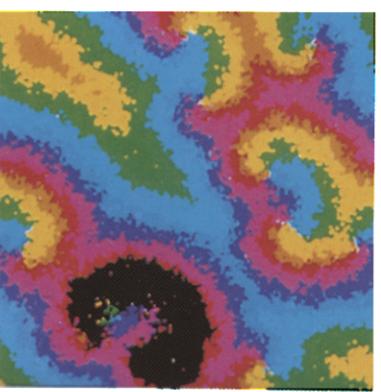


Plate 4A

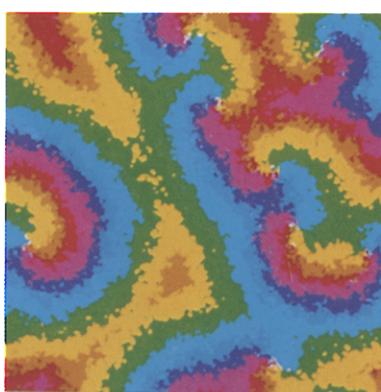


Plate 4B

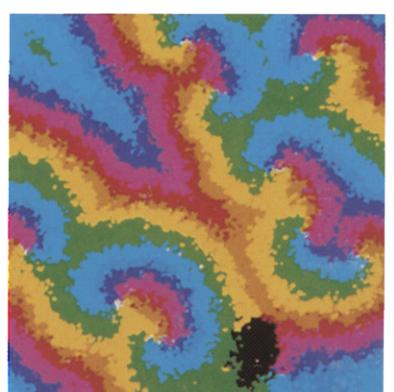


Plate 5

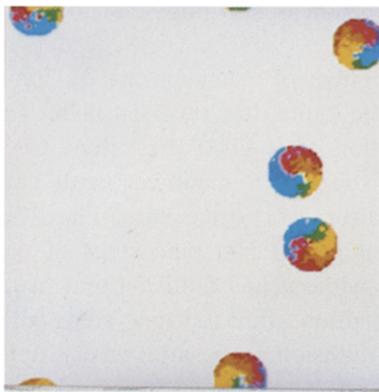


Plate 6A

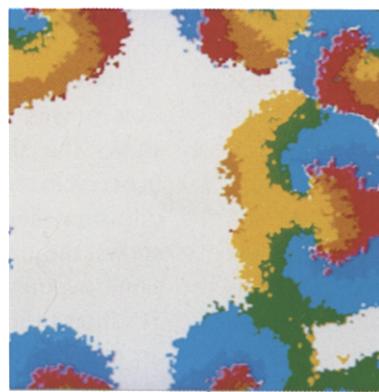


Plate 6B

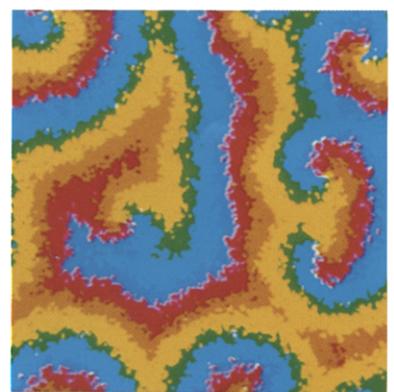


Plate 6C

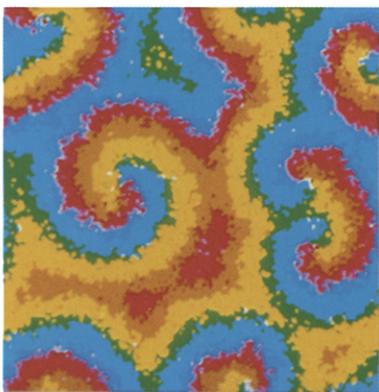


Plate 7A

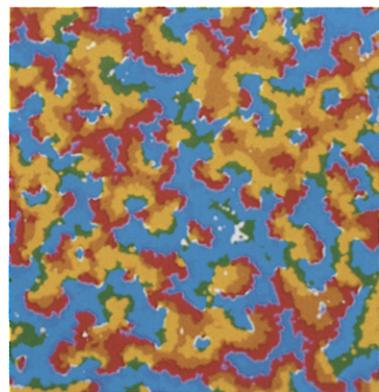


Plate 7B

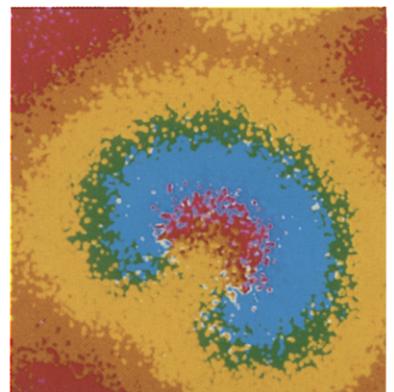


Plate 7C



Plate 2A



Plate 2B



Plate 2C

duced into the middle of a single spiral and again it has taken over the domain of the spiral. Now, however, after 600 time steps the parasite still is not wiped out by other spirals. The situation appears to be stable in time; the parasite forms a relatively small "cyst".

This last result looks puzzling. Why do the other spirals not take over the parasite region just as in the previous case where a double spiral was "killed" by a parasite? The key to the answer to this question is that a single spiral is not an independent entity. This is demonstrated by the removal experiment in plate 6A: all molecules in plate 7A that are not close to the middle of a spiral are removed; the single spiral in the middle of the field is removed completely. In plate 6B after 60 time steps the spirals start to grow again and in plate 6C after 300 time steps the spiral pattern has re-appeared. The single spiral that was removed has recovered, although it is a bit smaller and it is not exactly at the same spot. It turns out that, on a toroidal field, for each spiral rotating clockwise there must also be a spiral rotating anti-clockwise.

Apparently the spirals reinforce each other. This causes the cyst in plate 5 to remain present: the remaining spirals constantly induce the reformation of the "killed" single spiral. At any time, therefore, the parasite region is in contact with all species of the hypercycle; there is always catalytic support for the parasite. The phenomenon is restricted to single spirals; a double spiral is not reinforced.

We conclude that the stability against the parasite in this hypercycle system is a counter-example of the conclusions of the ODE hypercycle model [1, 7]. In the worst case the parasite can destroy one spiral and remain present as a cyst.

### 3.4. Diffusion

What happens to the self-structuring properties of the hypercycle when the medium is mixed more thoroughly? One might expect the spiral pattern to become unstable at higher rates of

diffusion, for diffusion is an undirected force of dispersal.

However, the effect of a higher rate of diffusion proves to be quite the reverse. Plate 7A shows the situation after 2000 time steps of a hypercycle consisting of 6 members, with two diffusion steps after every time step. Plate 7B shows the situation after 500 time steps of the same hypercycle without any diffusion and plate 7C shows the situation after 500 time steps with as many as 16 diffusion steps after every time step. The main effect of increased diffusion is that the spiral pattern enlarges dramatically. Without diffusion the catalytic waves are thin; with diffusion they become thicker and thus more effective in eliminating molecules that receive no catalysis. This means that parasites are removed more easily when there is diffusion; e.g. the parasite in plates 3A–3C is fatal when there is no diffusion.

Intuitively one might suspect that for high rates of diffusion things must muck up, because an infinite diffusion rate means complete mixing and therefore the differential equations results should hold, i.e. the hypercycle should be vulnerable to parasites. The results, however, indicate that for increasing rates of diffusion the spirals become bigger and bigger (if the space is large enough) and more stable against parasites. This result surely holds up to 32 diffusion steps after every time step; we did not investigate higher rates of diffusion because in that case the spirals become too big for our cellular automaton.

## 4. Conclusion and discussion

*4.1. Molecules with a hypercyclic interaction show spatial self-structuring. The existence of a spiral wave structure causes a hypercycle to be resistant to parasites.*

The spiral wave is a well known pattern in excitable media which has been studied most thoroughly with respect to the Belousov–

Zhabotinskii reaction, both experimentally and theoretically (e.g. refs. [8, 9]). Most theoretical models are formulated in terms of partial differential equations (c.f. ref. [10]); spiral wave solutions are found in cellular automata models as well ([4, 11–13]). Spiral waves have been shown to play a role in cell to cell communication in *Dicytostelium discoideum* (e.g. ref. [14]) and in neuro-muscular tissue (e.g. ref. [15]). Our analysis suggests a role for spiral waves in the enabling of evolution of co-operation.

We tested our model extensively for robustness of the self-structuring property. It turns out that the precise definition of the cellular automaton does not affect the development of the spirals. We examined for instance asynchronous updating of the cells, a non-toroidal field and other neighbour cells that can give catalytic support. Furthermore, the individual molecule species in the hypercycle can (to some extent) differ in their parameters. The number of species in the hypercycle does affect the formation of spiral waves; it appears that for the given parameters the hypercycle needs to consist of at least five members, otherwise no stable spirals are formed. For hypercycles of four or less members the resistance against any parasite is lost. The parameter dependence of spiral development and resistance to parasites is an intricate matter, which exhibits a wide variety of ordered, chaotic and complex behaviour. We will report on this in a future paper [16].

Anyhow the existence of a spiral pattern causes a hypercycle to be resistant to a large class of parasites. A parasite is only fatal to a hypercycle if it is able to grow towards the middle of a spiral. This is a difficult task, for it is against the direction of growth within the spiral; the parasite gets hardly any catalytic support in this direction and has to compete with the passing catalytic waves. It is not an impossible task; in order to be fatal a parasite should for instance have a low decay parameter (much lower than the species in the hypercycle; in plate 1C  $\text{decay}[\text{parasite}] \leq 0.1$ ) or it should get catalysis from more than one member

of the hypercycle. The properties of all members of the hypercycle are of importance for the outcome of the competition. This is in sharp contrast with the ODE model [1] where the parasite competes with only a single member of the hypercycle (i.e. species 3 in fig. 4).

A parasite that is unable to grow towards the middle of a spiral cannot destroy a hypercycle. The greatest harm such a parasite can do is to invade in the middle of a single spiral and remain present as a cyst. In a spiral pattern that consists only of double spirals the parasite is always wiped out.

#### *4.2. Resistance to parasites implies positive selection for a strong altruistic property. This contradicts generally accepted selection theory and is caused by spatial structuring.*

We have shown that in an incompletely mixed medium a hypercycle can be resistant to parasites. A “parasitic” mutant that receives increased catalytic support from a member of the hypercycle but does not give catalytic support to any molecule is wiped out. This implies that there is positive selection for molecules giving catalytic support.

We tested the robustness of this property by strengthening the parameters of the parasite. It appears that in the situation of plate 1C a parasite with a lower decay parameter (table 1:  $\text{decay}[\text{parasite}] = 0.15$ ), a higher self-replication parameter (table 1:  $\text{self}[\text{parasite}] = 2$ ) or a higher catalytic support parameter (table 1:  $c[\text{parasite}, 2] = 500$ ) still cannot destroy the hypercycle. In addition, a parasite that gives weak catalytic support (table 1:  $c[4, \text{parasite}] = 10$ ) is also wiped out. We conclude that selection for the giving of catalytic support is a robust property in this system.

In generally accepted selection theory (see refs. [17, 18]) this selection seems impossible, for the giving of catalytic support does not raise the number of copies of a molecule. In fact giving catalytic support is a so-called strong altruistic property, for a molecule that receives catalytic

support destroys its “benefactor”. This description of the selection process is on the level of the individual molecules. However, in the system we described selection on the long run appears to take place at the level of the spirals; in competition with a parasite the spirals act as integrated entities. Because a spiral consists of more than one species there appears to be inter-specific “group selection” on the individual level, i.e. there is selection for “helping” (giving catalytic support to) another molecule species. In 4.3 we will discuss the specific structure of the spirals which causes this phenomenon.

#### *4.3. The spirals consist of a “breeder” structure. In this type of structure, group selection is possible.*

Within a spiral there is a strikingly unequal distribution of long-term fitness: the molecules in the middle of a spiral generate the offspring of the entire spiral whereas the molecules in the periphery of a spiral disappear (as shown in plates 2A–2C). Thus the spatial self-structuring “creates” a small subclass of molecules which dominates reproduction: the breeders.

Breeders from different spirals are separated from each other: each spiral acts as if it were a super-organism whose cell wall is formed by the molecules in the periphery. This structure permits group selection, for there is competition at the level of the spirals. If the breeders of a spiral all possess an altruistic property that increases the competition strength of the periphery, then this spiral will expand. If there is a non-altruistic “cheater” mutant amongst the breeders of a spiral, then the altruistic property will be lost within this spiral. However, the former region of the spiral will then be taken over by other spirals that are still altruistic.

The self-structuring origin of the “breeder” structure makes it an attractive alternative to previous models of group selection (e.g. refs. [19, 20], and in the context of hypercycles [21]), which

require behavioural and/or spatial pre-structuring.

#### *4.4. Spatial self-structuring can have a major impact on the outcome of selection processes. Therefore it should be taken into account in the study of pre-biotic evolution.*

In this study we have shown that spatial self-structuring alters a selection property of hypercycles, namely their vulnerability to parasites. Preliminary results show that other selection properties of the hypercycle such as “once-for-ever” selection and selection in joint hypercycles (see appendix A) also change as a result of the spatial self-structuring. We will report on this in a future paper.

Furthermore it seems plausible that the phenomenon of spatial self-structuring is not restricted to hypercycles. For instance, in the model of Farmer et al. [22] a cyclic catalytic network of polymers is formed. This network differs from the structure of a hypercycle in that the polymers are not self-replicative. However, the interactions in this network look very much like the interactions in the abovementioned Belousov–Zhabotinskii reaction [9], so the spiral structure may well emerge in this system too. Whether cyclic interaction structures are likely to appear and outcompete other structures in networks with random interactions is subject for further study.

Self-structuring is a well-known feature of cellular automata. Simple low-level transition rules can generate high-level spatial patterns. This spontaneous self-structuring has often been interpreted as a form of evolution (e.g. ref. [23]). In this study we use a different approach; we consider self-structuring as a substrate for selection [24]. The substrate has proved very fertile; an environment is created in which inter-specific group selection is possible.

We believe therefore that in the study of (pre-biotic) evolution it is important to look for self-structuring and examine its consequences.

## Acknowledgements

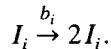
We thank Miss S.M. McNab for linguistic advice, and Drs. R. Bertin for discussion in the early phase of the investigations.

## Appendix A. The ODE model of the hypercycle

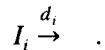
In the model  $n$  self-replicative molecule species are linked cyclically by catalysis (see fig. 3). The total number of molecules  $C$  is kept constant by an output flux  $\phi$ . Erroneous mutants are not included in the model. For analytical prove and further discussion see refs. [1, 7].

### A.1. Kinetic steps

Self-replication:



Decay:



Catalysed replication:

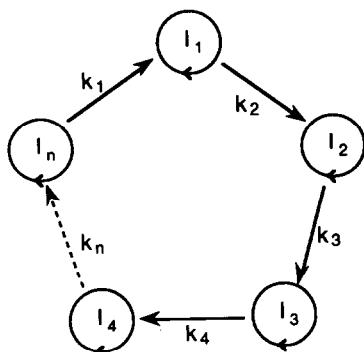
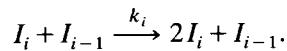


Fig. 3. Schematic diagram of a hypercycle. The hypercycle consists of self-replicative molecule species  $I_i$ ; each species provides catalytic support for the subsequent species in the cycle. After ref. [1].

Output flux:

$$I_i \xrightarrow{\phi} .$$

### A.2. Differential equations

$$\dot{X}_i = r_i X_i + k_i X_i X_{i-1} - \phi X_i, \quad r_i = b_i - d_i;$$

$$\sum_{i=1}^n X_i = C,$$

$C$  being the total number of molecules;

$$\phi = \left( \sum_{i=1}^n (r_i X_i + k_i X_i X_{i-1}) \right) / C.$$

### A.3. Summary of dynamical and selectional properties

#### Stability

The elementary hypercycle has only one attractor. At low dimensions ( $n \leq 4$ ) the attractor is an asymptotically stable fixed point, namely, a focus for  $n = 2$  and a spiral sink for  $n = 3$  and  $n = 4$ . In systems of higher dimensions ( $n \geq 5$ ) “permanence” has been proven, i.e. no molecule species vanishes; numerical integration provides strong evidence for the existence of a stable limit cycle.

#### Parasites

In fig. 4 a hypercycle with a so-called parasite is shown. The system appears to be competitive, i.e. the hypercycle and the parasite cannot co-exist. If the linear terms are neglected, the following relation holds:

if  $k_{\text{par}} > k_3$ , the parasite wins; the entire hypercycle becomes extinct;

if  $k_{\text{par}} < k_3$ , the parasite becomes extinct.

#### Competition between joint hypercycles

In fig. 5 two joint hypercycles are shown. The two cycles exclude each other (again neglecting the linear terms):

if  $k_1 > k_2$ , hypercycle  $\Gamma_1$  will outcompete  $\Gamma_2$ ;

if  $k_1 < k_2$ , hypercycle  $\Gamma_2$  will outcompete  $\Gamma_1$ .

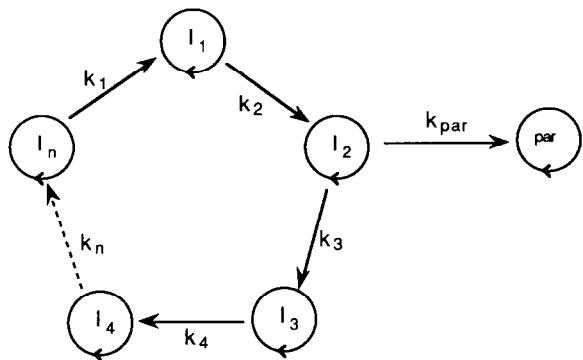


Fig. 4. Schematic diagram of a hypercycle with a self-replicative “parasitic” molecule species “par”. The parasite gets catalytic support from species  $I_2$  but does not give catalytic support to any molecule species in the hypercycle. After ref. [1].

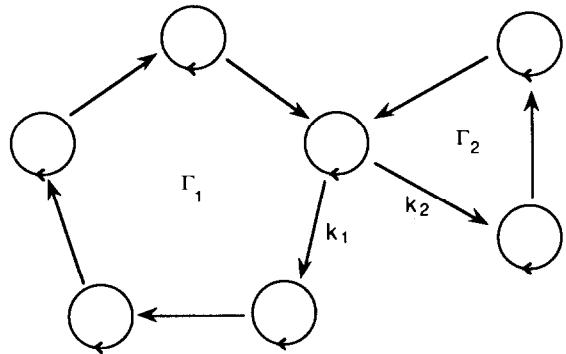


Fig. 5. Schematic diagram of two joint hypercycles. After ref. [7].

#### *Competition between disjoint hypercycles*

Selection of a hypercycle is a “once-forever” decision. A hypercycle, once established, cannot easily be replaced by any newcomer, since new species always emerge as one copy; the growth rate of a hypercycle is non-linear and therefore dependent of population size.

#### References

- [1] M. Eigen and P. Schuster, *The Hypercycle: A Principle of Natural Self-Organization* (Springer, Berlin, 1979).
- [2] J. Maynard Smith, *Nature* 280 (1979) 445.
- [3] T. Toffoli and N. Margolus, *Cellular Automata Machines: A New Environment for Modeling* (MIT Press, Cambridge, MA, 1987).
- [4] U. Frisch, B. Hasslacher and Y. Pomeau, *Phys. Rev. Lett.* 56 (1986) 1505.
- [5] S. Wolfram, *Theory and Applications of Cellular Automata* (World Scientific, Singapore, 1986).
- [6] T. Toffoli, *Physica D* 10 (1984) 117.
- [7] J. Hofbauer and K. Sigmund, *The Theory of Evolution and Dynamical Systems* (Cambridge Univ. Press, Cambridge, 1988).
- [8] S.C. Müller, T. Plessner and B. Hess, *Physica D* 24 (1987) 71.
- [9] J.P. Keener and J.J. Tyson, *Physica D* 21 (1986) 307.
- [10] J.J. Tyson and J.P. Keener, *Physica D* 32 (1988) 327.
- [11] D. Griffeath, *Notices Am. Math. Soc.* 35 (1988) 1472.
- [12] M. Gerhardt and H. Schuster, *Physica D* 36 (1989) 209.
- [13] M. Gerhardt, H. Schuster and J.J. Tyson, *Science* 247 (1990) 1563.
- [14] J.J. Tyson, K.A. Alexander, V.S. Manoranjan and J.D. Murray, *Physica D* 34 (1989) 193.
- [15] A.T. Winfree, *J. Theor. Biol.* 138 (1989) 353.
- [16] M.C. Boerlijst and P. Hogeweg, in: *Artificial Life II, SFI Studies in the Sciences of Complexity*, ed. C. Langton (Addison-Wesley, Reading, MA), in press.
- [17] J. Maynard Smith, *The Theory of Evolution*, 3rd Ed. (Penguin Books, Harmondsworth, 1975).
- [18] R. Dawkins, *The Selfish Gene* (Oxford Univ. Press, Oxford, 1976).
- [19] M.E. Gilpin, *Predator-Prey Communities* (Princeton Univ. Press, Princeton, 1975).
- [20] D.S. Wilson, *The Natural Selection of Populations and Communities*. (Benjamin-Cummings, Menlo Park, CA, 1980).
- [21] R.E. Michod, *Am. Zool.* 23 (1983) 5.
- [22] J.D. Farmer, S.A. Kauffman and N. Packard, *Physica D* 22 (1986) 50.
- [23] P. Tamayo and H. Hartman, in: *Artificial Life, SFI Studies in the Sciences of Complexity*, ed. C. Langton (Addison-Wesley, Reading, MA, 1988).
- [24] P. Hogeweg, *BioSystems* 23 (1989) 231.