

# The Positive Role of Parasites in the Origins of Life

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**Abstract**—One problem in the origins of life is how parasitic side-reactions can be mitigated. It is known that spatial self-organisation can help with this, making autocatalytic chemical systems more robust to invasion by parasitic species. In previous work we have shown that in such scenarios parasitic reactions can actually be beneficial. Here we demonstrate for the first time a system in which the presence of a parasitic autocatalytic cycle is not only beneficial but actually necessary for the persistence of its host. This occurs due to the effect the parasite has on the spatial organisation of the system; the host-parasite system is more stable than the host alone, despite the fact that the parasite's direct effect on its host is purely negative. We briefly discuss the implications for the origins of life.

**Keywords**—origins of life, reaction-diffusion, parasites, spatial self-organisation

## I. INTRODUCTION

Approaches to the origins of life have traditionally been divided into two camps, the “metabolism-first” and “replicator-first” approaches. Central to both approaches is the notion of an autocatalytic network, i.e. a set of molecules that react with an energy-containing substrate in such a way as to produce more molecules from the same set. In the information-first paradigm the members of this set are taken to be complex, information-carrying molecules such as RNA (e.g. [1]), whereas the metabolism-first view holds that only simpler molecules were involved originally, with genetic mechanisms arising later.

A problem for both approaches is the possibility of parasitic side-reactions, i.e. autocatalytic subnetworks that can benefit from the reactions in the original network, without contributing any of its products. Partly for this reason, a consensus seems to be emerging between the two approaches, in which the earliest ancestors of modern organisms are seen as existing in “compartments” that contain both a metabolic substrate and an information-carrying replicator. As well as preventing the autocatalytic compounds from dispersing into their environment, the compartments separate one individual from another, localising the effect of parasites and allowing them to be weeded out by natural selection. (See [2] for further discussion.)

Another approach to the problem of parasites was given by Boerlijst and Hoegeweg [3], who showed that hypercycles (a particular type of autocatalytic network) were resistant to parasites when embedded in a spatial setting, without any

compartmentalisation. This research has spawned a substantial body of literature on the question of how the effect of parasites is modulated by spatial dynamics.

In [4], [5] we presented a situation in which the existence of a parasite in a system can actually give a positive benefit. This occurs because the parasite affects the behaviour of dissipative “spot” patterns that form in the system, increasing their ability to invade new, uncolonised areas of the space.

In this work we go even further, presenting a situation where the parasite is not only beneficial but is actually necessary for survival, in the sense that the primary autocatalytic set will drop to zero concentration unless the parasitic set is also present. In ecological terms, the relationship thus becomes one of mutualism rather than parasitism. Similarly to our previous work, this occurs because of the parasitic reaction's effect on the dynamics of spontaneously formed spatial structures, in this case damping oscillations that would otherwise destroy them. The stabilising effects of predatory species are well-known in population dynamics, but we believe the importance of such effects has so far been understated in the origins of life. After presenting our model, we conclude by discussing other positive roles that parasites could play in prebiotic systems.

## II. THE MODEL

Our model is based on the Gray-Scott reaction scheme, an extremely minimal model of autocatalysis that was first studied in a spatial context by Pearson [6]. The reactions are



Here,  $X$  represents an energy-bearing substrate (or “food”) that is continually fed into the system, while  $A$  represents an autocatalytic system that constructs itself out of this substrate.

The trimolecular Reaction 1 is unrealistic if we consider  $A$  to represent a single molecule; it should instead be thought of as a rough approximation to the kinetics we would expect if  $A$  were a more complex autocatalytic set. Reaction 2 represents the gradual decay of  $A$  into an inert product  $P$  that has no further effect on the system.

In order to consider the role of parasitic autocatalysis, we

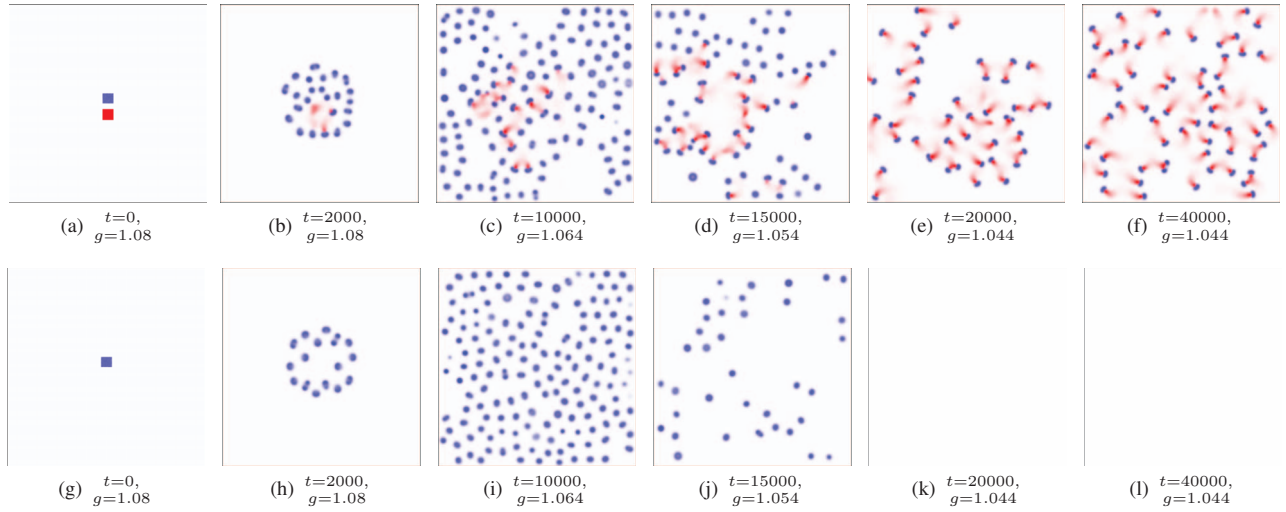


Fig. 1. Visualisation of the numerical experiments described in the text. The primary autocatalyst A is shown in blue and the parasitic autocatalyst B in red. F is not shown. Plots (a)–(f) show a run in which the parasite is present. Plot a shows the initial conditions. Plot (b) shows a number of self-replicating spots, at least one of which is “infected” with a tail of B. The global food availability parameter  $g$  is gradually reduced to 1.044, by which time only the spots with tails have survived (plot (e)). These can persist in the system for at least another 20000 time units (plot (f); see Fig. 2 for an enlarged version), and presumably indefinitely. Plots (g)–(l) show the results from a “control” run, which is identical except that no B is present in the initial conditions. The spots without tails disappear in this run as well, demonstrating that this occurs due to their inability to persist in a low- $g$  environment, rather than because the parasite infects every spot. The presence of B is necessary in order for A to persist even though the direct effect of B upon A is negative.

extend this model with the following two reactions:



B is an additional autocatalyst, which again is to be thought of as representing a system of several reactions rather than a single molecule. Rather than feeding on the substrate X, B feeds on the primary autocatalyst A, depleting it.

These reactions are modelled as taking place on a surface on which the species X, A and B can diffuse as well as reacting. X is continually fed into the surface at every point. This leads to a set of equations that can be parameterised as follows (see our earlier papers [4], [2] for more details):

$$\frac{\partial x}{\partial t} = D_x \nabla^2 x - x a^2 + f(g - x) \quad (5)$$

$$\frac{\partial a}{\partial t} = D_a \nabla^2 a + x a^2 - \alpha a b^2 - k_a a \quad (6)$$

$$\frac{\partial b}{\partial t} = D_b \nabla^2 b + \alpha a b^2 - k_b b. \quad (7)$$

Equations 5 and 6 are the standard Gray-Scott model as studied by Pearson, except that we have added a new parameter  $g$  to Equation 5 and the term  $-\alpha a b^2$  to Equation 6, representing the depletion of A by the parasitic reaction. Equation 7 is our addition, representing the dynamics of the parasitic autocatalyst B.

In this work we keep all of the parameters apart from  $g$  fixed, with the following values:  $D_x = 2 \times 10^{-5}$ ;  $D_a = 1 \times 10^{-5}$ ;  $D_b = 1 \times 10^{-6}$ ;  $f = 0.022$ ;  $k_a = 0.09$ ;  $\alpha = 0.8$ ;  $k_b = 0.002$ . Note that the diffusion rate and decay rate of B are an order of magnitude lower than the corresponding values of A. This is needed in order for B to persist in the system: if the parameters of B are instead chosen to be of a

similar magnitude to those of A, it will simply deplete the local concentration of A and then decay, since it has no way to spread to neighbouring spots of A.

The parameter  $g$  represents the general availability of the “food” species X. During the integration of the system, we vary this parameter over time in the following way: from  $t = 0$  to  $t = 2000$  we let  $g = 1.08$ . After  $t = 2000$  we decrease  $g$  linearly until it reaches the value  $t = 1.044$  at  $t = 20000$ . After this,  $g$  is held constant at the value 1.044. The reason for this procedure is that when  $g = 1.044$  it is very difficult to find initial conditions such that any pattern will persist. By starting with a higher value of  $g$  and then decreasing it, we side-step this difficulty.

Following Pearson we integrate the system using the forward Euler method with  $\delta x = 0.01$  and a system size of  $2.56 \times 2.56$  with von Neumann boundary conditions. We use a time step of  $\delta t = 0.3$ . The initial values are  $x = 1.08$ ,  $a = 0.005$ ,  $b = 0$ , except for a small square of side 0.05 in which  $x$  and  $a$  are both equal to  $0.3 \pm 10\%$  random noise, and, in the test runs only, another square the same size in which  $b = 1.0 \pm 10\%$  random noise.

### III. RESULTS

Fig. 1 shows the results of one “test” run in which the parasite is present, and one “control” run in which it is not. We have replicated each of these experiments 12 times, with qualitatively the same results in every case. (See Fig. 3.) The initial square of A rapidly becomes a pattern of self-replicating spots, which is a well-known behaviour of the Gray-Scott system. In the test run, some of these come into contact with the region of B, which is then able to propagate itself by feeding on them. A pattern then arises consisting of moving spots of A followed by “tails” of B.

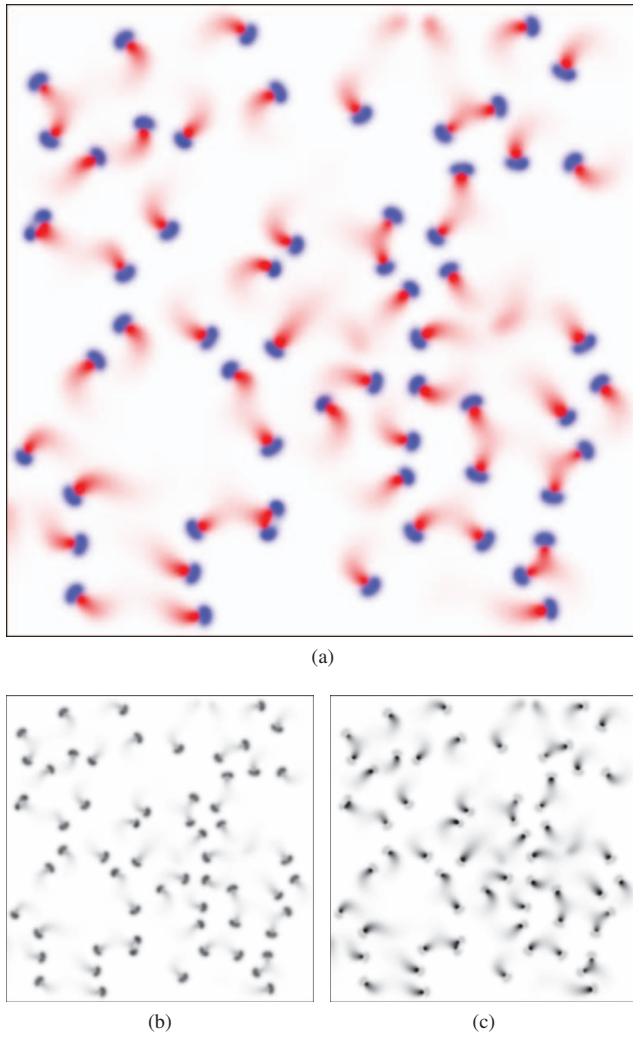


Fig. 2. (a) An enlarged version of Fig. 1f, showing the fully developed spot-tail pattern, with A shown in blue and B in red. Plots (b) and (c) show the same data in black and white; in plot (b) the primary autocatalyst A is shown in a darker shade than B, while in plot (c) the parasitic autocatalyst B is shown darker than A.

This spot-tail configuration is shown in more detail in Fig. 2. These spot-tail patterns are in constant motion, with the spot of A continually moving away from its tail of B. The spots disappear when they move into an area where there is too much B, but tend to fission if the local concentration of B is low, so that an approximately constant population is maintained. When a spot reproduces, its parasitic tail is “inherited” by each of its offspring. See [4], [2] for more on the formation and properties of these spot-tail patterns.

As we gradually reduce  $g$ , the spots without tails become unstable and begin to oscillate. That is, they deplete some of the substrate X locally and shrink to a smaller size as a result, but then the X recovers, resulting in an over-expansion of the spot of A and the cycle repeats. Occasionally the amplitude of the oscillations becomes such that the local concentration A is insufficient for it to recover after depleting X, and the spot disappears. When  $g$  drops below about 1.05 this cycling behaviour prevents the spots without tails from reproducing

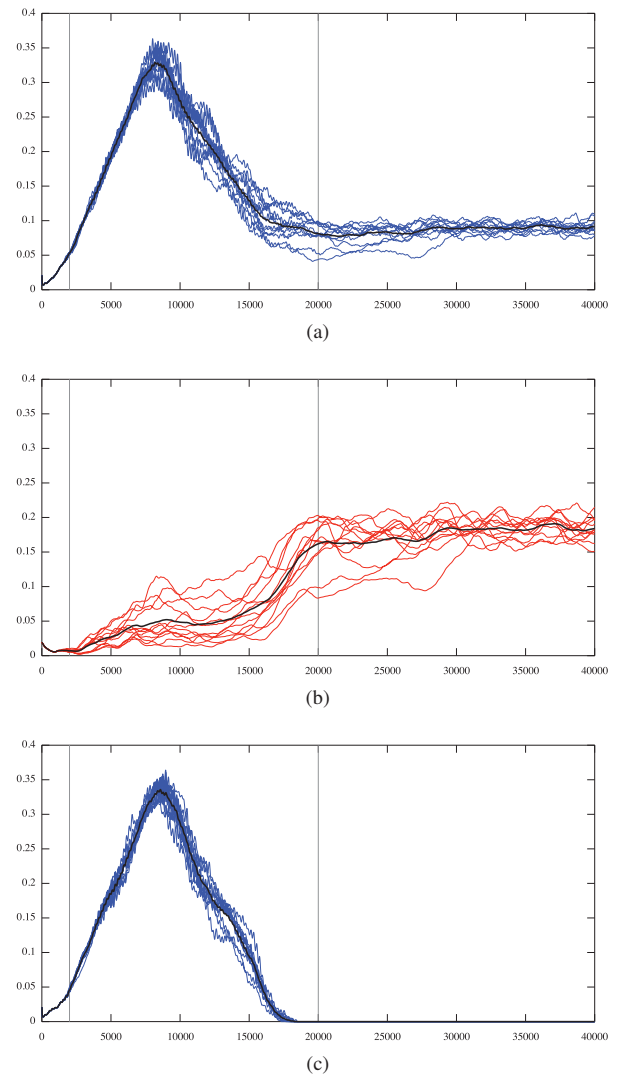


Fig. 3. Plots of the total amount of A and B in the system over time, obtained by integrating  $a$  and  $b$  over space at every time step. (a) and (b) show the amount of A and B respectively over the twelve test runs, and (c) shows the amount of A in the twelve control runs. (The concentration of B remains at zero throughout the control runs, since none is present initially.) In each plot the thin coloured lines are the time series for each individual run, and the thick black line represents the mean over all the runs. The thin vertical lines indicate the time period over which the parameter  $g$  is linearly reduced from 1.08 to 1.044. In the test runs, A reaches a stable mean amount of about 0.09, whereas in the control runs it always drops to zero.

and they cease to persist in the system. However, the spots that have a tail of the parasitic autocatalyst B are stabilised and continue to persist in the system indefinitely.

In the control runs, in which no B is added, all of the spots become unstable and the total concentration of A eventually drops to zero, as shown in Figure 3. This shows that the reason for the test run becoming dominated by the spot-tail pattern is not that the parasite B “infects” every spot, but rather that spots without a tail of parasite are unable to persist in the system once the value of  $g$  is low enough. Our test run therefore represents a case in which a parasitic side-reaction is not only beneficial but is actually necessary for the “survival” of the primary autocatalyst. This is due to its stabilising effect on the

dynamics of the spatial structures that form in the system.

Informally, we have also tried reducing  $g$  still further. Below about  $g = 1.042$  the spots with tails lose their ability to reproduce. They remain stable but tend to move in straight lines until they encounter the system's boundary or the tail of another spot, at which point they cease to persist. Below approximately  $g = 0.9$  they cannot persist at all.

#### IV. DISCUSSION

The idea that parasites or predators can lead to positive effects is well-known in biology. Examples include the origin of mitochondria as endosymbionts, and the ability of a top predator to stabilise the dynamics of an ecosystem. However, the literature on the origins of life tends to look upon their analogues in the chemical domain as a purely negative possibility that must be mitigated in order for pre-biotic metabolisms to persist.

Our work represents an existence proof in support of another possibility: that parasitic reactions can occasionally be beneficial. Although our example has many parameters whose values were chosen by hand, we suspect that beneficial parasitic reactions might not have been uncommon during the origins of life, for the simple reason that autocatalytic parasitic reactions can provide a mechanism for heredity. In our system, tails are "inherited" when spots fission; see also [7] for a mechanism of heredity based on autocatalytic subnetworks. Individuals bearing a beneficial parasite would have a differential advantage over those without, increasing the likelihood that such systems would be present in the pre-biotic world.

More generally, our work provides further evidence that spatial self-organisation should not be ignored when considering the origins of life. The current consensus view tends to envisage protocells as an autocatalytic set of chemical species (including a genetic mechanism), enclosed in a lipid bilayer membrane; but it tends to ignore all spatio-temporal dynamics apart from the fissioning of vesicles. However, there is a growing body of opinion that the time scale of individual behaviour must also play an important role in the properties and evolution of early organisms. This idea has arisen both from theoretical work [8], [9] and in work on wet experiments, in which oil droplets and vesicles have been shown to be capable of self-propelled motion, e.g. [10].

Our work adds to this by showing how easy it is for behavioural dynamics to arise in simple self-organising physical systems, and by demonstrating how such dynamics can be modulated in a heritable way by the presence of self-sustaining, autocatalytic parasitic reactions. In previous work, we have contributed to this view using models similar to the one presented here. In [4] we showed that parasitic reactions can lead to self-motility, thereby allowing their hosts to explore and colonise empty areas more rapidly, leading to an increased probability of survival through a mechanism we called a "behaviour-based hypercycle" [5]; in [2] we used an argument along the lines of [8] to show that processes on the behavioural level can help to reduce the need for efficient and highly selective enzymes in early proto-life. The present work shows that the dynamical modulation effect of parasites can

lead directly to more stable structures, enabling persistence in parameter regimes where it would not otherwise be possible.

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