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Non-linear protocell models: Synchronization and Chaos

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Abstract We consider generic protocells models allowing linear and non-linear kinetics for the main involved chemical reactions. We are interested in understanding if and how the protocell division and the metabolism do synchronize to give rise to sustainable evolution of the protocell.

Keywords Protocell · Self-Replication · Dynamical model · Synchronization

1 Introduction

Protocells could be lipid vesicles, or micelles, endowed with some rudimentary metabolism and should contain “genetic” material, being able to grow, reproduce and evolve. While viable protocells do not yet exist, their study is important in order to understand possible scenarios for the origin of life, as well as for creating new “protolife” forms which

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are able to adapt and evolve (8). This endeavor has an obvious theoretical interest, but it might also lead to an entirely new “living technology”, definitely different from conventional biotechnology.

Theoretical models can be extremely useful to devise possible protocell architectures and to forecast their behavior. What can be called the “genetic material” of a protocell is composed by a set of molecules which, collectively, are able to replicate themselves. At the same time, the whole protocell undergoes a growth process (its metabolism) followed by a breakup into two daughter cells. This breakup is a physical phenomenon which is frequently observed in lipid vesicles, and it has nothing to do with life, although it superficially resembles the division of a cell (5). In order for evolution to be possible, some genetic molecules should affect the rate of duplication of the whole container, and some mechanisms have been proposed whereby this can be achieved.

In this paper we address an important issue in protocell research: the genetic material duplicates at a certain rate, while the lipid container grows, in general, at another rate. When the container splits into two, it may happen that the genetic material has not yet doubled: in this case its concentration would be lower in the daughter protocells. Hence through generations, this density might eventually vanish. On the other hand, if the genetic material growth were faster than the container, the former would accumulate in successive generations.

So, in order for a viable population of evolving protocells to form, it is necessary that the rhythms of the two processes are synchronized. In some models (like the Chemoton (4) this is imposed a priori in the kinetic equations, but it is unlikely that such a set of exactly coupled reactions springs up spontaneously in a single step. It is therefore interesting to consider the possibility that such synchronization could be an emergent phenomenon, without imposing it a priori.

It has previously been shown that this may indeed happen when one takes into account successive generations of protocells (11). Even if at the beginning the two processes take place with different paces, they asymptotically approach synchronization, in the sense that both \(i\) the time interval between the formation of a protocell and the moment when it doubles its size and \(ii\) the time required for doubling the genetic material change, do vary and generation after generation, they tend to the same value.

The present paper explores under which conditions such synchronization takes place, in the interesting case where there are several different kinds of replicating molecules in each protocell, and they can affect each other’s replication rates in a non-linear way. It therefore expands our previous studies, which had considered synchronization \(i\) when there is only one kind of replicating molecule in each protocell \(ii\) when there are different kinds of replicators in each protocell, but they do not directly affect each other’s replication rate and \(iii\) when there are different kinds of replicators in each protocell, which affect each other’s replication rates, and the kinetic equations are linear. Note that the model as a whole is always non-linear, due to the division of the lipid container: linearity refers only to the set of kinetic equations for the replicators in a protocell.

Synchronization is studied here using abstract models belonging to the “surface reaction models” family (briefly, SRMs); let us observe that other architectures have been introduced, for instance the internal reaction models (IRM)s (2). The difference is that, in the former case, the reactions which lead to the formation of the new genetic material and those devoted to the formation of new membrane molecules take place close to the protocell outer surface, while in IRMs they both take place in the interior of
the vesicle. The modeling level is fairly abstract, so the results should hold for different
detailed protocell architectures. SRMs are inspired by the so-called “Los Alamos
bug”, a model of protocells where the genetic material is composed by strands of PNA
(9; 10) which should be found in the vesicle membrane. According to this hypothesis,
different PNA’s may influence the growth rate of their “container” by catalyzing the
formation of amphiphiles (which form the protocell membrane) from precursors. On the
other hand IRMs are supported among other, by the RNA-cell architecture proposed
by (7; 6; 13).

The paper is organized as follows. For the sake of completeness in sections 2 and
3 we recall the main features of SRMs, the main mathematical techniques to study
synchronization and the results of our previous studies. Section 4 is devoted to the
introduction of some non-linear replicators kinetic models and the description of their
synchronization properties. Finally, in the last section some critical comments and in-
dications for further work are reported, in particular aiming at understanding the out-
come of regular behaviors once the involved kinetic equations exhibit chaotic regimes:
embed chaos to make it disappear.

2 Surface Reactions Models

Let us suppose the protocell to be a spherical vesicle, with an aqueous interior and a
lipid-phase membrane composed by amphiphilic molecules. In a way inspired by the
Los Alamos bug hypothesis, we will suppose that the membrane grows by addition of
amphiphiles, which are formed close to its external surface by suitable precursors,
under the catalytic influence of some molecules which are found in the vesicle, hereby
called Genetic Memory Molecules, GMMs for short. In general, only a fraction of these
catalysts will be effective, namely those which are close enough to the outer surface (it
is assumed that these molecules can be found in the lipid phase). We will also suppose
that the membrane is composed by a single kind of amphiphilic molecules.

Let us then consider a single protocell, and let \( C \) be the quantity of its lipid con-
stituent (moles of amphiphiles). Then the growth of \( C \) in time is assumed to be propor-
tional to the vesicle surface \( S \), times a function of the concentration of the catalysts
in the membrane, times a function of the density of the precursors which are found
outside, close to the membrane. We will assume that this last term is not influential,
e.g. that either precursors are buffered, so that their concentration is kept constant, or
that there are saturation effects and the concentration of the precursors is high enough
to saturate.

Moreover, we will also assume i) that the rate of spontaneous formation of am-
phiphiles in the outer medium is negligible with respect to the one due to the catalytic
effects of the replicators and ii) that diffusion is fast enough to ensure that, on the
time scale of the model, the catalyst concentration is homogeneous in the lipid phase:
so, if there is a single kind of catalyst, and if \( X \) denotes its quantity (moles) in the
protocell lipid phase, its concentration is \([X] = X/V_L\), where \( V_L \) is the volume of the
lipid phase.

Therefore, under the above hypotheses

\[
\frac{dC}{dt} = S(V_L) \dot{f} \left( \frac{X}{V_L} \right)
\]  (1)
Where \( \hat{f} \) denotes a function of the concentration that will be specified later on, and the dependency of \( S \) upon the volume \( V_L \) has been emphasized. We will assume that \( S \) is proportional to \( V_L^{\beta} \), with the parameter \( \beta \) ranging in the interval \([2/3, 1]\) : in a spherical micelle, where the volume of the lipid phase equals the total volume, \( S \) would be proportional to \( V_L^{2/3} \), while in a vesicle with a very thin membrane it would be almost proportional to \( V_L \) itself. Since \( V_L = C/\rho \) (with \( \rho \) constant), by redefining the arbitrary function which appears in the r.h.s. of the previous equation we obtain

\[
\frac{dC}{dt} = C^\beta \hat{f} \left( \frac{X}{C} \right)
\]  

(2)

If, as it is often the case, we assume \( \hat{f} \) to be proportional to \( X/C \) to some power \( \gamma \) we obtain

\[
\frac{dC}{dt} = \alpha C^{\beta - \gamma} X^\gamma
\]  

(3)

A particularly important studied case is when the growth of the container is assumed to be proportional to the concentration of the catalyst molecules, i.e.

\[
\frac{dC}{dt} = \alpha C^{\beta - 1} X
\]  

(4)

Eqs. (3) and (4) describe the continuous growth of the protocell up to a certain size, which is then followed by the breakup in two daughter cells. Although this is a complicated process, we will assume for simplicity (as it is done in most models) that the cell breaks when it reaches a certain size, i.e. when \( C(t) \) equals a fixed value \( \theta \) and that the initial value of \( C \) of each of the two daughter cells is \( \theta/2 \).

We also suppose, like in Los Alamos bug, that also the replication of the \( X \)-type molecules takes place near the external surface, by virtue of buffered precursors which are also in this case not taken explicitly into account. Replication takes place in a thin boundary of width \( \delta \) near the surface, therefore only the molecules which are found in this volume can contribute to the synthesis of new molecules. Therefore \( dX/dt \) should be proportional to \( S \delta \) times a function of \( X/C \), so

\[
\frac{dX}{dt} = S(V_L^\beta) \hat{g} \left( \frac{X}{C} \right) = C^\beta \hat{g} \left( \frac{X}{C} \right)
\]  

(5)

Assuming \( \hat{g} \) to be proportional to \( X/C \) raised to some power \( \nu \) we then obtain

\[
\frac{dC}{dt} = \eta C^{\beta - \nu} X^\nu
\]  

(6)

The case where the continuous growth phase is described by Eqs. (4) and (6) will be referred to as the “single replicator” case.

Note that in deriving Eqs. (5) and (6) it has been implicitly assumed that replications needs the presence of externally supplied precursors, which are available only close to the outer boundary of the membrane. This is consistent with a template duplication mechanism, like that of nucleic acids; if replicators were e.g. interacting polypeptides, and precursors were available in the whole lipid phase, then all the \( X \) molecules in the lipid phase could contribute and, instead of Eq. (5) we would get

\[
\frac{dX}{dt} = C \hat{g} \left( \frac{X}{C} \right)
\]  

(7)
And, if \( g \) is proportional to \( X/C \) raised to some power \( \nu \)

\[
\frac{dX}{dt} = \eta C^{1-\nu} X^\nu
\]

(8)

So the difference between the two cases would amount to choosing \( \beta = 1 \) in the second one. But it has already been shown (11) that, while the kinetics is influenced by the value of the geometric parameter \( \beta \), the achievement of synchronization is not, so that one can limit to analyze the case \( \beta = 1 \) to draw general conclusions. We therefore come to the conclusion that, as far as synchronization is concerned, both a “nucleic-acid based” and a “polypeptide based” hypothesis are described by the formalism used here and therefore behave in much the same way.

A particularly important case, whose analysis is quite simple, is the one where both exponents \( \gamma \) and \( \nu \) are equal to one, so

\[
\begin{align*}
\frac{dC}{dt} &= \eta C^{0} X \\
\frac{dX}{dt} &= \eta C^{0} X
\end{align*}
\]

(9)

Which can be straightforwardly generalized to the case of \( N \) different linearly interacting replicators:

\[
\begin{align*}
\frac{dC}{dt} &= \alpha X C^{\beta-1} \\
\frac{dX}{dt} &= \eta C^{\beta-1} X
\end{align*}
\]

(10)

Where \( \alpha \) and \( X \) are \( N \)-dimensional vectors and \( M \) is the \( N \times N \) matrix of kinetic coefficients. The same arguments used above concerning the role of \( \beta \), we can limit ourselves to consider the case \( \beta = 1 \) in the study of the synchronization phenomenon.

Next step is to consider two replicators, \( X \) and \( Y \), in the same protocell whose growth rate is assumed to be non-linear. If they do not interact directly, and their effect on the growth of the lipid container is linear, then the continuous growth is described by the following equations, analogous to Eq. (6)

\[
\begin{align*}
\frac{dC}{dt} &= \alpha' X + \alpha'' Y \\
\frac{dX}{dt} &= \eta' X^{\nu} C^{\beta-\nu} \\
\frac{dY}{dt} &= \eta'' Y^{\nu} C^{\beta-\nu}
\end{align*}
\]

(11)

A more interesting case is when the replicators directly interact each others. For instance, let us suppose that the kinetics is second order, so that the rates of production of new \( X \) and new \( Y \) should be proportional to the frequency of encounters between \( X \) and \( Y \) molecules. The total number of encounters per second, in the lipid phase, is then \( S \delta [X][Y] \approx V_L^{\beta-2} \delta X Y \). By observing that \( V_L \) is proportional to \( C \) and by lumping some constants in the terms \( \eta' \) and \( \eta'' \) one then gets

\[
\begin{align*}
\frac{dC}{dt} &= \alpha' C^{\beta-1} X \\
\frac{dX}{dt} &= \eta' C^{\beta-2} X Y \\
\frac{dY}{dt} &= \eta'' C^{\beta-2} X Y
\end{align*}
\]

(12)

Where we have assumed for simplicity that only one kind of molecule, say \( X \), catalyzes the growth of the membrane, i.e. set \( \alpha'' = 0 \) in Eq.(11). In the more general case where the rate of production of \( X \) is proportional to the concentration of \( Y \) times some power \( \nu \) of \( X \), and similarly for \( Y \), the previous equations generalize to
\[
\begin{align*}
\frac{dC}{dt} &= \alpha' C^{\beta-1} X \\
\frac{dX}{dt} &= \eta' C^{\beta-1-\nu} X^\nu Y \\
\frac{dY}{dt} &= \eta'' C^{\beta-1-\nu} X Y^\nu
\end{align*}
\]  

(13)

Eqs. (11) and (12) are examples of cases with two non-linear interacting replicators. More general non-linear equations will be introduced in section 4.

3 A summary of previous results

The equations of SRMs lend themselves to a nice analytical technique, which has been applied in order to study their behavior, and which has been complemented by numerical simulations\(^1\).

Starting with an initial quantity of container \(C\) at time \(T_0\) equal to \(\theta/2\), we assume that once \(C\) reaches a critical value \(\theta\), the protocell will divide into two equal protocells each one of size \(\theta/2\). Let \(\Delta T_0\) be the time interval needed to double \(C\) from this initial condition, and let \(T_1 = T_0 + \Delta T_0\) be the time when the critical value \(\theta\) is reached. Since the initial value for \(C\) is fixed, \(\Delta T_0\) is a function of the initial quantity of replicators, \(X_0\). The final value of \(X\) in the protocell, just before the division will be denoted by \(X(T_1)\).

Because we assume perfect halving at the division, each offspring will start with an initial concentration of replicators equal to \(X_1 = X(T_1)/2\). Then the continuous growth process starts again, until the next doubling time will be reached, \(T_2 = T_1 + \Delta T_1\), and the third generation will start with an initial value \(X_2 = X(T_2)/2\), and so on.

We generalize the preceding discussion with the following equations, which refer to the \(k\)th cell division cycle that starts at time \(T_k\) and ends at time \(T_{k+1}\):

\[
\frac{\theta}{2} = \int_{T_k}^{T_{k+1}} \frac{dC}{dt} dt, \quad X_{k+1} = \frac{1}{2} X(T_{k+1})
\]  

(14)

Note that in general \(X(T_{k+1}) \neq 2X(T_k)\) and therefore the time needed to double the value of \(C\) is not constant between two successive generations. The problem of synchronization therefore amounts to finding under which conditions the following equality asymptotically holds:

\[
\lim_{t \to \infty} [X(T_{k+1}) - 2X(T_k)] = 0
\]  

(15)

The existence of first integrals of the equations describing the continuous growth phase, allows us to prove synchronization. In fact we are able to obtain a discrete map \(X(T_{k+1}) = F(X(T_k))\), and the search for conditions which allow synchronization are equivalent to the existence of a fixed point.

There is no general form for the first integrals, which depend upon the kind of SRM model which is considered; several examples can be found in (11; 3; 2) and the main results are hereby summarized. Before doing so, it is however interesting to consider which kind of behaviors one can imagine to find. The possible alternatives are the following:

---

\(^1\) Numerical simulations have been performed using Matlab’s standard solver for ordinary differential equations ode45 with parameter “nonNegative”. This function implements a Runge-Kutta method with a variable time step for efficient computation and prevents the variables to become negative, for details see Matlab website (http://www.mathworks.com).
1. **Synchronization**: in successive generations (as $k \to \infty$) the time for duplication of the protocell, $\Delta T_k$, and the time required to duplicate the genetic material, $\Delta T_{gk}$, approach the same value (for the reasons highlighted above, this condition is satisfied when Eq.(15) holds).

2. The initial concentration of the genetic material vanishes in the limit of infinitely many divisions; in this case, given the above assumptions, the growth of the container ends and the whole process stops.

3. The initial concentration of the genetic material grows unbounded as $k \to \infty$. This results in a limitation of the previous equations to modeling a protocell, which indeed lack a rate limiting term for the growth rate of $X$.

4. The two rates $\Delta T_k$ and $\Delta T_{gk}$ oscillate in time with the same frequency. This condition is not equivalent to synchronization *strictu sensu* but it would nonetheless allow sustainable growth of the population of protocells. Therefore this condition might be called *super-synchronization*. Note that in principle super-synchronization does not require equality of the two frequencies, but that their ratio be a rational number.

5. The two rates $\Delta T_k$ and $\Delta T_{gk}$ exhibit a non regular time behavior. In the case of a single replicator whose replication rate given by Eq.(6) with $0 < \nu < 2$, with linear growth for the container, one can show that synchronization is always achieved provided that $\alpha$ and $\eta$ are positive (these conditions are fairly obvious, otherwise it would make no sense to speak of growth). The result holds also if one takes a realistic geometry into account (i.e. if one uses the volume of a spherical shell instead of the approximation $S \approx V_L^3$). It also generalizes to more general forms of the function which describes the growth of the protocell or that of the replicators (12). More generically, we can prove that once the container growth follows some non-linear power law, i.e. $\gamma \neq 1$ in Eq.(4), then a necessary condition to have synchronization is $0 < \nu < \gamma + 1$.

Let us observe that the interesting case of parabolic growth, i.e. $\nu < 1$, fits the above condition. On the other hand, we can prove and it has been numerically checked that in the case of a very steep increase $\nu \geq 2$, the system does not approach synchronization: If $\nu = 2$ then synchronization can be obtained only with a very specific choice of the involved parameters, whereas if $\nu > 2$ synchronization can never be achieved, according to the initial amount of $X$, in the long run either $X_k$ diverges or goes to zero.

A further generalization is worth discussing. In section 2 we have assumed that $[X] = X/V_L$, which is correct as long as $X$ itself does not appreciably contribute to the volume of the lipid phase. But if the quantity of $X$ becomes large, and $X$ itself is a lipophilic compound which contributes to the container, this formula should be substituted by $[X] = X/(V_L + V_X)$. This can be rewritten, by rescaling the kinetic constants, as $X/(C + r X)$. It has been analytically shown and numerically confirmed that also in this case synchronization is achieved when $\alpha$ and $\eta$ are positive.

If there are several replicators in the same cell, but they do not interact directly, namely the system is modeled by Eq.(11), one again finds synchronization if $\alpha, \eta$ and $\eta'$ are positive. In the linear case, $\nu = 1$, with two replicators, only the fastest replicator survives in the final population of protocells, while if $\nu < 1$ both survive, their relative proportion being a function of $\eta/\eta'$. This is consistent with similar behaviors observed in population dynamics. On the other hand if there is mutual interaction, namely the protocell can be described by Eq.(12) or Eq.(13), then synchronization cannot be always achieved.
The case of several linearly interacting replicators Eq.(10) has been analytically studied and a complete discussion of the different cases can be found in (2). The most relevant results are that the behavior of the system is ruled, in the long time limit, by the eigenvalue of the matrix \( M \) with largest real part, denoted by \( \lambda_{LRP} \). If \( \Re(\lambda_{LRP}) > 0 \), and if the corresponding eigenvector \( v_1 \) is non-negative\(^2\), synchronization is achieved. The asymptotic value of \( X_k \) is a multiple of \( v_1 \). A sufficient condition to guarantee that \( \lambda_{LRP} \) is real and positive and that the associated eigenvector is non-negative, is that the matrix elements \( M_{ij} \) are non-negative. This is an important case, corresponding to all \( X \) molecules, which directly interact, contribute to the synthesis of the others (mutual catalysis).

It is however possible to imagine also cases where the network of reactions includes some negative terms (if they were all non-positive the system would of course die out). The most relevant phenomenon discovered in this analysis is the existence of an oscillatory behavior, super-synchronization, which is found when the eigenvalues with the largest real part are complex conjugate (see for instance figure 1).

Let us observe that the most striking result of the analysis of linear replicators is that they behave in a way similar to that of a Continuously Stirred Tank Reactor: in the present case vesicle splitting limits the asymptotic values, while in CSTR it is the outflow which does it. But the ratio of various replicators is the same in the two cases. The coefficient \( \alpha \) does not influence this ratio, nor the asymptotic division time, although it affects the actual value of the asymptotic quantities.

These latter cases show that non-linearity may lead to a halting of the growth, or to an unbounded growth of the molecules, hence preventing the protocell from a viable evolution. These behaviors can be associated with the presence of power laws growth rates, to tackle the question we thus consider more general non-linear models by introducing “squashing functions”.

\(^2\) Because eigenvectors are defined up to a multiplicative factor, by non-negative we mean that all the components can be made positive.
4 Non-linear models with squashing terms

The growth of the container is still assumed to be described by a linear equation as Eq.(4), for instance

\[ \frac{dC}{dt} = \alpha X \]  

(16)

We then select some possible kinetic equations for the replicators growth, that we now briefly describe and analyze with the help of dedicated numerical simulations.

4.1 Quasilinear models

One drawback of linear equations like eq.(10) is that the growth rate may undergo an unrealistic unlimited increase. In order to take physical constraints on the rates into account it is possible to introduce bounds which are never exceeded. Instead of fixing sharp thresholds, which would lead to discontinuous derivatives, we consider here "squashing functions", i.e. functions which are bounded both from below and from above, and which are never decreasing. They can be thought as activating functions for the reactions to start.

Let \( \sigma(x) \) be such a function, then we hypothesize the following replicator law in the case of \( N \) interacting molecules:

\[ \frac{dX_i}{dt} = C \beta \sigma \left( \sum_{k=1}^{N} \frac{M_{ik}X_k}{C} \right) \]  

(17)

In this case the behavior is similar to that of the corresponding linear model, i.e. Eq.(10), once we use the same coefficients \( M_{ij} \). In particular, synchronization in the linear models implies synchronization in the quasi-linear one. However sometimes we have observed super-synchronization in the linear case, while the corresponding non-linear version still synchronizes.

We frequently observe that the amounts of \( X \)-molecules rapidly saturate, so that the duplication times are largely unaffected by the exact values of the matrix elements \( M_{ij} \) and are not a function of \( \lambda_{LRP} \), as it would happen in the linear case. A second interesting observed feature is that cell duplication times are not affected by the value of \( \alpha \).

5 Second order kinetics

The quadratic model Eq.(12) can be generalized as to include \( N \) interacting GMMs, to give

\[ \frac{dX_i}{dt} = C \beta -2 \sum_{k=1}^{N} M_{ik}X_iX_k \]  

(18)

In the case of mutual catalysis the matrix coefficients are non-negative. Note that catalytic cycles can be modeled in this way by a proper choice of the matrix elements \( M_{ik} \). Generically this model does not show synchronization, except for very peculiar
relationships among the coefficients; in the case of only two GMMs one can provide an analytical answer.

On the other hand, once we introduce molecules able to self-replicate

\[
\frac{dX_i}{dt} = C\beta X_i X_k + C\beta -1 \eta_i X_i \tag{19}
\]

we can have synchronization. Let us observe that fixing non-negative matrix elements \(M_{ik}\) in a cyclic way one gets the well known hyper-cycle systems.

The synchronization fixed point depends on the value of \(\eta\) as shown in figure 2.

6 Second order kinetics without self-replication

Let us finally consider the case where there is no self-replication, but the GMMs mutually catalyze each other’s formation from existing precursors, in a way it requires the interaction of two molecules to produce a third one. The corresponding equations, neglecting possible saturation effects, are then

\[
\frac{dX_i}{dt} = C\beta -2 N \sum_{k=1}^{N} M_{ik} X_i X_k \tag{20}
\]

Where, in order to avoid self-replication, the matrix elements should have the form

\[
M_{ik} = \mu_{ik} (1 - \delta_{ij})(1 - \delta_{ik}) \tag{21}
\]

In this case we observe that sometimes synchronization is achieved, while in other cases extinction is the outcome. This result is related to the sparseness of the matrix
M. It is interesting to observe that, if a large fraction of the matrix elements is non-vanishing, synchronization is found, even when the frequency of negative $M_{ijk}$'s equals that of positive ones.

Considering a random initialization of $M_{ijk}$, increasing the number of GMMs (in a population of independent protocells) the sparsity coefficient decrease due to the random initialization of the non zero entries that decrease increasing the matrix dimensions.

Several simulations show that, considering an equal frequency of negative and positive entries, ten replicators (that correspond to a sparsity coefficient equal to 0.28) are sufficient in order to avoid death of the protocell.

7 Conclusions and remarks on the chaotic behaviors

The models considered so far are, from a mathematical point of view, generic non-linear systems of differential equations, that we know can exhibit chaotic behaviors. It is thus a natural question to consider how these non-regular behaviors can be associated to sustainable growth and evolution of protocells. In other words, why, even if you have a set of chemical reactions that exhibit a chaotic behavior, once you “embed” these reactions in a protocell, their behavior is more regular, in such a way the protocell can grow and divide?

In the following we provide a partial answer to this question by considering the Willamowsky-Rössler (1) system\(^3\), that exhibits chaotic behaviors (see figure 3) and it has been already used to model chemical reactions:

\[
\begin{align*}
\frac{dX}{dt} &= k_1 X - k_{-1} X^2 - k_2 XY + k_{-2} Y^2 - k_4 XZ + k_{-4} \\
\frac{dY}{dt} &= k_2 XY - k_{-2} Y^2 - k_3 Y + k_{-3} \\
\frac{dZ}{dt} &= -k_4 XZ + k_{-4} + k_5 Z - k_{-5} Z^2 
\end{align*}
\]

We observe that if the previous kinetic equations are introduced in a protocell, whose container varies as a function of some GMMs, then the Willamowsky-Rössler protocell becomes

\[
\begin{align*}
\frac{dC}{dt} &= \alpha X \\
\frac{dX}{dt} &= k_1 X - \frac{1}{C} k_{-1} X^2 - \frac{1}{C} k_2 XY + \frac{1}{C} k_{-2} Y^2 - \frac{1}{C} k_4 XZ + C k_{-4} \\
\frac{dY}{dt} &= \frac{1}{C} k_2 XY - \frac{1}{C} k_{-2} Y^2 - k_3 Y + C k_{-3} \\
\frac{dZ}{dt} &= -\frac{1}{C} k_4 XZ + C k_{-4} + k_5 Z - \frac{1}{C} k_{-5} Z^2
\end{align*}
\]

We observed that the system undergoes through a bifurcation as the parameters $\alpha$ varies: for small values of the latter the system still evolves chaotically following the Willamowsky-Rössler strange attractor, then as $\alpha$ increases the system presents, first a periodic orbit and then a fixed point, hence synchronization, see figure 4.

This implies that protocells, and possibly also real cells, during their evolution could have “tuned”, or have been selected, in such a way the coupling between container growth and metabolism/information reproduction, gives rise to a regular behavior, even if the latter systems could evolve (separately) in a chaotic way.

\(^3\) Similar conclusions have been found by analyzing a modified Lorentz system where the strange attractor is fully contained in the positive octant.
The addressed question is surely relevant to understand the emergence of sustainable forms of life, and thus deserves further investigations that will be presented elsewhere.

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References


