

Autocatalytic Sets and the Growth of Complexity in an Evolutionary Model

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A model of s interacting species is considered with two types of dynamical variables. The fast variables are the populations of the species and slow variables the links of a directed graph that defines the catalytic interactions among them. The graph evolves via mutations of the least fit species. Starting from a sparse random graph, we find that an autocatalytic set inevitably appears and triggers a cascade of exponentially increasing connectivity until it spans the whole graph. The connectivity subsequently saturates in a statistical steady state. The time scales for the appearance of an autocatalytic set in the graph and its growth have a power law dependence on s and the catalytic probability. At the end of the growth period the network is highly nonrandom, being localized on an exponentially small region of graph space for large s . [S0031-9007(98)07953-8]

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A characteristic feature of chemical, biological, economic, and social evolution is that it produces a complex network of interactions among the component species or agents involved in it. Understanding the mechanisms responsible for the origin of such networks and their movement towards greater complexity is an important issue. One mechanism, based on quasispecies and hypercycles [1], proposed in the context of prebiotic chemical evolution has self-replicating entities as its components. Another proposed mechanism starts from simpler components that are not individually self-replicating but can collectively form an autocatalytic set (ACS) [2–4]. The present work attempts to explore the latter mechanism quantitatively through a mathematical model.

The model has two main sources of inspiration. One is the set of models studied by Farmer, Kauffman, Packard, and others [3,5] and by Fontana and Buss [6] (see also [7,8]). Like these models the present one employs an artificial chemistry of catalyzed reactions, albeit a much simpler one, in which populations of species evolve over time. To this we add the feature, inspired by the model of Bak and Sneppen [9], that the least fit species mutates. Unlike the Bak-Sneppen model, however, the mutation of a species also changes its links to other species. This allows us to investigate how the network of interactions among the species evolves over a longer time scale. We find that for a fixed total number of species, the network inevitably evolves towards a higher complexity as measured by the degree of interaction among species and their interdependence. The increase is triggered by the chance appearance of an ACS, is exponential in time, and leads to a highly nonrandom organization.

The system is described by a directed graph with s nodes. The nodes represent the components or species, and the directed links represent catalytic interactions among them. A link from node j to i means that species j catalyzes the production of i . The graph is completely described by specifying the adjacency matrix $C \equiv (c_{ij})$,

$i, j = 1, \dots, s$. c_{ij} equals unity if there is a link from j to i , and zero otherwise. A link from a node to itself is prohibited (diagonal entries of C are zero); this corresponds to the exclusion of self-replicating species.

At the initial time ($n = 0$), the graph is random. That is, c_{ij} (for $i \neq j$) is unity with probability p and zero with probability $1 - p$. Thus on average every row and column of C has $m \equiv p(s - 1)$ nonzero entries, representing the average number of links to and from a node. p is the probability that a given species will be a catalyst for another given species.

The graph is updated at discrete time steps ($n = 1, 2, \dots$) as follows: One selects the “mutating node” of the existing graph by a rule to be specified below, removes all the existing incoming and outgoing links to and from that node, and replaces them by randomly chosen links with the same catalytic probability p . That is, if the selected node is i , the i th row and column of C are reconstituted according to the same rule as in the previous paragraph. This changes the graph into a new one. A mutating node is selected afresh, and this process is iterated over many time steps.

The mutating node at any n is taken to be the one with the “least fitness” at that time step. Associated with every node i is a population $y_i \geq 0$, or a relative population $x_i \equiv y_i/Y$, $Y \equiv \sum_{j=1}^s y_j$. The population depends upon a continuous time t , and its evolution between two successive graph updates (i.e., while the graph C remains fixed) is given by

$$\dot{y}_i = \sum_{j=1}^s c_{ij}y_j - \phi y_i. \quad (1)$$

From this it follows that x_i has the dynamics

$$\dot{x}_i = \sum_{j=1}^s c_{ij}x_j - x_i \sum_{k,j=1}^s c_{kj}x_j. \quad (2)$$

The x_i dynamics depends only on C and not on ϕ . The time between two successive graph updates is assumed

long enough to allow the fast variables x_i to reach their attractor configuration, denoted X_i . X_i is a measure of the fitness of the species i in the environment defined by the graph. The set of nodes with the smallest value of X_i is called the set of least fit nodes. The mutating node is picked randomly from the set of least fit nodes. For the cases that have arisen in our simulations, the set of least fit nodes depends only on the graph and not upon the initial values of x_i .

The dynamics (1) is an approximation of the rate equations in a well stirred chemical reactor with a nonequilibrium dilution flux ϕ when the reactants necessary for the production of the molecular species in the graph are fixed and in abundance, and spontaneous reaction rates are much smaller than catalyzed reaction rates. Then reaction rate is limited by and proportional to catalyst concentration. The i th species grows via the catalytic action of all species j that catalyze its production and declines via a common death rate ϕ . That all catalytic strengths are equal is an idealization of the model. This dynamics might also be relevant for economics (e.g., positive feedback networks) as well as certain kinds of ecological webs. A justification for selecting the least fit node to be mutated in a molecular context is that the species with the least population is the most likely to be lost in a fluctuation in a hostile environment. Alternatively, in an ecological context, certain fitness landscapes might be such that a low fitness is correlated with a smaller barrier to mutation (see the arguments in [9]). In economics a correlation between fitness and survival is at the heart of evolutionary game theoretic models like the replicator equation; the elimination of the least fit is an extreme idealized case of this correlation. Keeping the total number of species constant in the simulation is another idealization of the model.

In Fig. 1 we plot the total number of links $l(n)$ in the graph versus the graph update time step n . Three runs with $s = 100$ are exhibited, each with a different value of

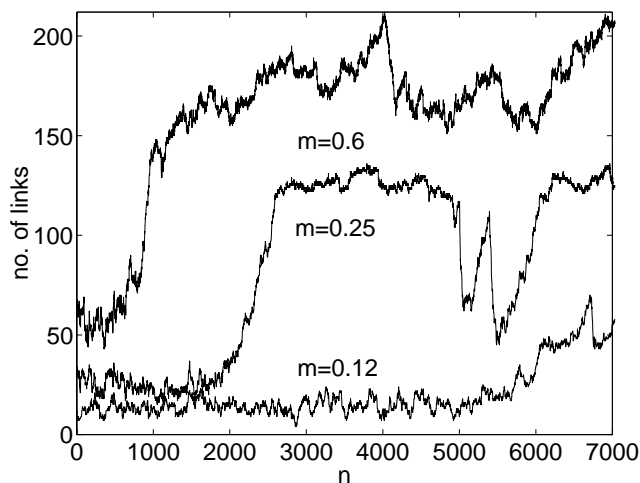


FIG. 1. Total links versus n for three runs with $s = 100$.

p (or m). We have studied the parameter range m from 0.05 to 2.0 and $s = 50, 100$, and 150. For fixed m, s we have conducted several runs with different random number seeds. The runs shown in Fig. 1 are typical of the runs with the same parameter values.

The curves have three distinct regions. Initially the number of links hovers around the value expected for a random graph, $l \sim ms$. The second region is one of rapid increase, in which l rises several fold. The third is a statistical steady state with many fluctuations where the average connectivity is much higher than the initial one.

The increase in l over time is a consequence of selection. In a “random run” in which the mutating node is chosen at random from among all the s nodes instead of from the set of least fit nodes, l keeps fluctuating about its initial average value ms . Note that under selection l rises even though the average connectivity of the species that replace the mutating species is the same as in the initial random graph. One may be tempted to give the following naive explanation of the increase: From (1) it is clear that the larger the number of species that have links to the species i , the greater is the rate of increase of y_i . Therefore the species that do well (population wise) are those that have more links coming in, and conversely those that do not do so well are deficient in incoming links. Hence selecting the least fit species amounts to selecting the species that have lower connectivity than average. If these are replaced by species that have the old average connectivity, it is no surprise that the number of links increases.

This explanation is not correct. First, this argument is unable to explain the observed fact that there is a long region of almost constant l in the graphs before it starts to increase. Second, in this region the mutating nodes tend to have a larger proportion of outgoing links than average, which more or less balances out their deficiency in incoming links. The real explanation, which we substantiate in detail below, is the chance appearance of an ACS in the graph.

Since (2) does not depend on ϕ , we can set $\phi = 0$ in (1) without loss of generality for studying the attractors of (2). For fixed C the general solution of (1) is $\mathbf{y}(t) = e^{Ct}\mathbf{y}(0)$, where \mathbf{y} denotes the s dimensional column vector of populations. It is evident that if $\mathbf{y}^\lambda \equiv (y_1^\lambda, \dots, y_s^\lambda)$ viewed as a column vector is a right eigenvector of C with eigenvalue λ , then $\mathbf{x}^\lambda \equiv \mathbf{y}^\lambda / \sum_i y_i^\lambda$ is a fixed point of (2). Let λ_1 denote the eigenvalue of C which has the largest real part; it is clear that \mathbf{x}^{λ_1} is an attractor of (2). By the theorem of Perron-Frobenius for non-negative matrices [10] λ_1 is real and ≥ 0 and there exists an eigenvector \mathbf{x}^{λ_1} with $x_i \geq 0$. If λ_1 is nondegenerate, \mathbf{x}^{λ_1} is the unique asymptotically stable attractor of (2), $\mathbf{x}^{\lambda_1} = (X_1, \dots, X_s)$. In practice, we found in our simulations that λ_1 was usually nondegenerate, except for very sparse graphs. This is not surprising in view of the well known level repulsion in random matrix theory, which implies that

repeated eigenvalues are very improbable for a generic matrix.

An ACS is defined as a subgraph whose every node has at least one incoming link from a node that belongs to the same subgraph. This definition is meant to capture the property that an ACS has “catalytic closure” [3]; i.e., it contains the catalysts for all its members. The simplest ACS is a 2-cycle. The following hold: (i) An ACS always contains a cycle. (ii) If a graph has no ACS, then $\lambda_1 = 0$ for the graph. (iii) If a graph has an ACS, then $\lambda_1 \geq 1$. (iv) If $\lambda_1 \geq 1$, then the subgraph corresponding to the set of nodes i for which $x_i^{\lambda_1} > 0$ is an ACS. We will call this subgraph the “dominant ACS” of the graph. These properties, which we first observed numerically, can be proven analytically from graph theory [11]. It follows from (iv) that members of the dominant ACS completely overshadow all other species population wise.

For $m < 1$ the initial random graph is sparse. For example, with $s = 100$, $m = 0.25$, there are on average only 25 links. Most of the nodes are singletons, some pairs have a single link among them, and there are a few chains or other trees with two or more links. The probability of there being a cycle is small [$\sim O(m^2)$]. Figure 2 shows how λ_1 evolves. Since λ_1 remains zero for $n < n_1 = 1643$, it is clear that there is no cycle in the graph in this period. When there are no cycles, then $y_i \sim t^r$ for large t , where r is the length of the longest path terminating at i . Then $X_i = 0$ for all i except the nodes at which the longest paths in the graph terminate. Define $s_1(n)$ as the number of species i for which $X_i \neq 0$ at the n th time step. This is plotted in Fig. 3. (The lower curves in Figs. 2 and 3 correspond to a random run with $s = 100$, $m = 0.25$.) Since the mutant can be any least fit node, the chains can be disrupted over time. In particular, if the mutating node happens to be the “nearest neighbor” of a node that is the terminating point of the

longest chain, after mutation the latter node can become a singleton and join the ranks of the least fit.

The picture changes qualitatively when an ACS appears by chance in a mutation. Then λ_1 jumps from zero to one (at $n = n_1$). For concreteness let us say the ACS at $n = n_1$ is a 2-cycle between species 1 and 2. Then $\mathbf{x}^{\lambda_1} = (1/2, 1/2, 0, 0, \dots, 0)$. The key point is that both species 1 and 2 are absent from the set of least fit nodes and will not be mutated at the next time step. By definition the nodes which are not part of the dominant ACS of a graph with $\lambda_1 \geq 1$ have $x_i^{\lambda_1} = 0$ from property (iv) above, and hence constitute the set of least fit nodes. Therefore, as long as the dominant ACS does not include the whole graph, the mutating node will be outside it, and hence a mutation cannot destroy the links that make up the dominant ACS just before the mutation. *Thus the autocatalytic property is guaranteed to be preserved once an ACS appears until the dominant ACS engulfs the whole graph.* In the run of Figs. 2 and 3 this happens at $n = n_2 = 2589$, when $s_1(n) = s$. Whenever $s_1 < s$, λ_1 is a nondecreasing function of n . An increase in s_1 during $n \in (n_1, n_2)$ occurs whenever a mutant species gets an incoming link from the existing dominant ACS and hence becomes a part of it. (Note that s_1 itself need not be a nondecreasing function of n when it is $< s$, because the dominant ACS after a mutation can be smaller than the one before the mutation.)

There is another qualitative change in the evolution at $n = n_2$. Since the whole graph becomes an ACS, for the first time since the appearance of the ACS the mutant must now be from the dominant ACS itself. When the mutant happens to be a species which is playing an important catalytic role in the organization (a “keystone species”), the mutation can disconnect a number of other species from the main ACS, as evidenced from the substantial drop in s_1 at $n = 4910$. The final steady state in Fig. 1 is characterized by the fact that the mutating

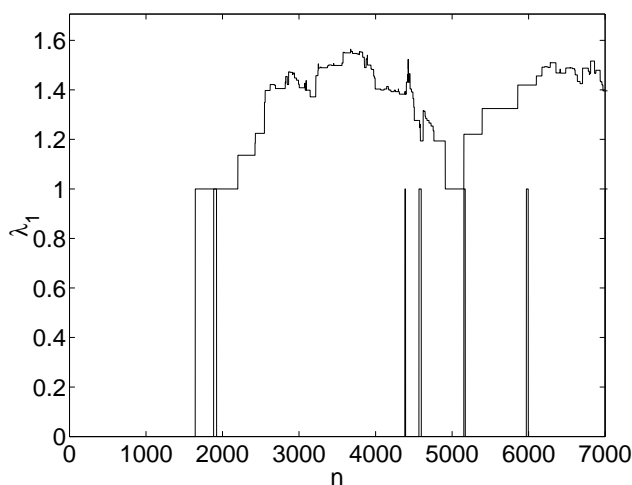


FIG. 2. λ_1 versus n for $s = 100$, $m = 0.25$. The upper curve is for the same run as the middle curve of Fig. 1. The lower curve is for a random run with $s = 100$, $m = 0.25$.

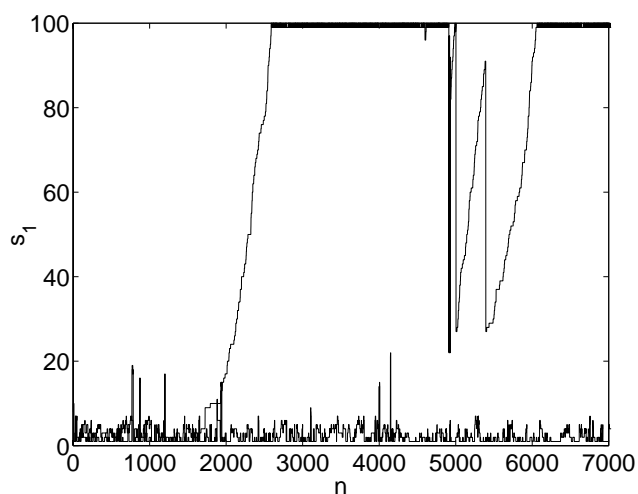


FIG. 3. s_1 versus n for the same runs as in Fig. 2.

node has, on average, the same total number of links (namely, $2m$) as its replacement.

The above picture holds for different values of m, s , as long as m is small enough and s large enough. For very small m the fluctuations in the final steady state are large; the ACS can even be destroyed completely. For sufficiently high values of m the initial random graph is dense enough to contain an ACS, hence the initial period with $\lambda_1 = 0$ is absent.

During the growth period $n \in (n_1, n_2)$, s_1 and l (locally averaged in time) grow exponentially. For example, $s_1(n) \sim s_1(n_1)e^{(n-n_1)/\tau_g}$. The m dependence of the “growth time scale” τ_g is shown in Fig. 4 and is consistent with $\tau_g \propto m^{-1}$. In a time Δn , the average increase in s_1 in a large sparse graph is given by $\Delta s_1 \sim ps_1\Delta n$, which is the average number of mutating nodes out of Δn which will get an incoming link from the s_1 nodes of the dominant ACS. Therefore $\tau_g \sim 1/p \sim s/m$. The average “time of arrival” $\tau_a \equiv \langle n_1 \rangle$ of an ACS in a sparse graph is given by $\tau_a \sim s/m^2 \sim 1/(p^2s)$, since the probability that a graph which does not have an ACS will get a 2-cycle at the next time step (3-cycles and larger ACSs being much less likely for small p) is $\sim p^2s$. Thus for any finite p , however small, the appearance and growth of ACSs in this model is inevitable.

The graphs generated at the end of the growth phase are highly nonrandom. The probability of a random graph with s nodes and on average m^* links per node being an ACS is $[1 - (1 - [m^*/(s-1)]^{s-1})^s]$, which declines exponentially with s when $m^* \sim O(1)$. This may be relevant to the origin of life problem for which naive estimates of the probability of a complex organization like a cell arising by pure chance on the prebiotic earth give exponentially small values. The present model provides an example whereby highly nonrandom organizations can arise in a short time by a mechanism that causes an exponential increase in complexity. The hypercycle is

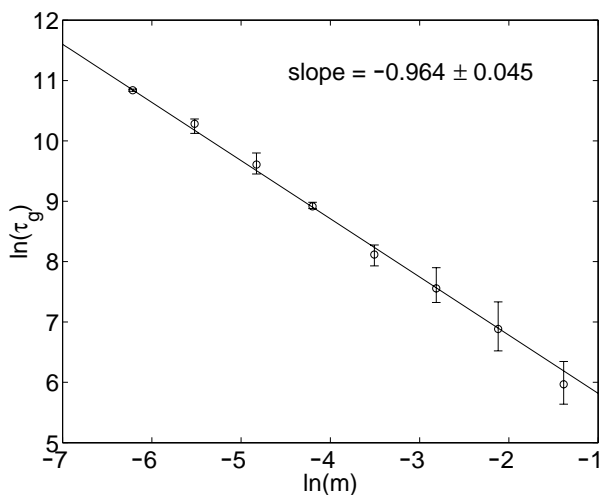


FIG. 4. Power law dependence of τ_g on m .

known to suffer from the short-circuit instability which reduces its complexity [12]. It is interesting that in the present model ACSs provide the system with the opposite kind of instability, in the direction of increasing complexity. Finally, this model provides an example of how selection for fitness at the level of individual species results, over a long time scale, in increased complexity of interaction of the collection of species as a whole. Note in Fig. 2 that in the random run ACSs come and go, whereas, when selection is operative, the system “cashes in” upon the novelty provided by an ACS that arises by chance. This is reminiscent of how ecosystems and economic webs capitalize on “favorable” chance events.

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