# Cascade of Complexity in Evolving Predator-Prey Dynamics

Nicholas Guttenberg and Nigel Goldenfeld
*Department of Physics and Institute for Genomic Biology,*
*University of Illinois at Urbana-Champaign, 1110 West Green Street, Urbana, Illinois, 61801-3080.*

We simulate an individual-based model that represents both the phenotype and genome of digital organisms with predator-prey interactions. We show how open-ended growth of complexity arises from the invariance of genetic evolution operators with respect to changes in the complexity, and that the dynamics which emerges shows scaling indicative of a non-equilibrium critical point. The mechanism is analogous to the development of the cascade in fluid turbulence.

Experiments on digital organisms represent one of the most accurate and informative methodologies for understanding the process of evolution[1]. Systematic studies on digital organisms are especially informative, because the entire phylogenetic history of a population can be tracked, something that is much more difficult— but not impossible[2]—to do with natural organisms. Experiments on digital organisms can be performed over time scales relevant for evolution, and can capture universal aspects of evolutionary processes, including those relevant to long-term adaptation [3, 4], ecological specialization[5, 6] and the evolution of complex traits[7].

Despite this progress, the way in which evolution leads to ever increasing complexity of organisms remains poorly understood and difficult to capture in simulations and models to date. Is this because these calculations are not sufficiently realistic, extensive, or detailed, or has something fundamental been left out? In this Letter, we argue that two fundamental aspects of evolutionary dynamics, with the character of symmetries, have been omitted, thus causing complexity growth to saturate.

The first feature is that the evolutionary dynamics must be invariant with respect to changes in the complexity of the evolving organisms. That is, if there are inhomogeneities which encourage organisms to have a specific complexity, then these will act to prevent the complexity of the system from continually increasing. This invariance is similar in spirit to that which lies at the heart of the Richardson cascade in turbulence[8, 9]. Here, a hierarchy of length-scales exists due to a transport of energy by scale-invariant processes between a large length scale and a small length scale. The largest and smallest features of the flow are determined by where the invariance is broken. In the biological case, processes invariant to changes in complexity will allow the dynamics to produce structures of arbitrarily high complexity. We will see below, in an explicit model, the effects of different genetic operations with regard to this invariance criterion. This criterion can also apply to the way that the fitness of an organism is determined in the dynamics, either explicitly or implicitly.

The second feature is that there must be some advantage which can only be gained by an organism in the system being more complex than the organisms it competes with. Competitive interactions can drive such a dynamic; for example, if competition can be thought of as one organism setting the environmental problem that the other organism must solve. The resulting co-evolution favors an increase in complexity over a decrease, because for the problem-setter, simplifying the problem does not exclude an organism already able to solve the problem. This factor has the same function as viscosity in turbulent flows: it sets the directionality of the relevant transport.

These two features have precisely the same mathematical role in evolutionary models as the mechanisms of energy transfer and viscous dissipation do in fluid turbulence. Thus, the open-ended growth of complexity in our model, and the existence of a hierarchy of structures at all scales in turbulent flows are mathematical consequences of the same underlying dynamics. It is not important for this argument what is the direction of energy flow in the turbulence case: in fact, the direction depends on dimensionality, with the possibility of the accumulation of large-scale structures in two-dimensional turbulence through the so-called inverse cascade.

The implications of this dynamical systems argument are far-reaching, and impose constraints on how digital evolution models should be built. For example, despite its popularity, the "fitness landscape" [10, 11, 12] picture of evolution does not satisfy these constraints, and is conceptually insufficient to account for the open-ended growth of complexity. To illustrate our points, we now show how open-ended growth of complexity emerges from underlying dynamical rules in a simple caricature of an evolving ecosystem.

*Complexity saturation in digital ecosystems:-* Tierra[13] and Avida[14] are systems of digital organisms, which are represented as self-replicating programs in a Turing complete language. In principle any program or behavior can then be encoded with a sufficiently large genome. In Tierra, organisms exist in a linear space for which each point in space is associated with an instruction and replication occurs via a loop which copies the contents of the

space at an offset. In early work on the Tierra model it became evident that the dynamics were not neutral with respect to the size of replicating programs. Evolutionary pressure favored smaller programs as they replicate with fewer instructions and out-produce the larger programs in the system. This led to the development of interesting parasitic behavior in which a program would use a neighbor's replication code to decrease its length, i.e. the complexity of organisms did not increase. When this was corrected by a change in the way in which resources were allotted, the length of organisms was observed to increase in bursts, but eventually saturated for longer and longer intervals[13], a finding attributed to insufficient richness of the environment[15].

In Avida, there is a two dimensional grid, each cell of which contains a program, and replication occurs between cells. Selection is based on an organism's ability to solve a particular mathematical problem. Avida uses an information-theoretic definition of complexity based on the information learned by the organism from its environment[7]. For evolution occurring in a single niche, it is found that this complexity increases for some time, then saturates to a value determined by the maximum information associated with the niche (the potential complexity) [16].

A similar pattern of saturation in the level of complexity is found in 'WebWorld'[17, 18, 19, 20]. Here, species are described by a set of features that may be either present or not, and the total rate of predation between species is determined by summing over a random interaction matrix for each feature possessed by the predator and each possessed by the prey. The total number of features possessed is found to increase in the presence of interactions above the neutral case. However, the increase in complexity is eventually limited by the predefined set of features, there being no possibility of creating new features in the model.

In summary, these and other digital ecosystems appear to lack the drive to increasing complexity that arguably is present in real biological systems.

*Foodchain:-* We now present an abstract minimal model of an evolving predator-prey system, which we call "Foodchain". This model exhibits the potential for an open-ended growth of complexity. Organisms in this model exist in a two-dimensional space and interact with each other. The detailed mechanics of replication are abstracted away (unlike Tierra and Avida)—during replication, genetic operators (point mutation and gene duplication) are applied to the genomes, which are of fixed length 2048, to produce the genome of the offspring. In 'Foodchain', fitness is determined solely by interactions between organisms, as they attempt to eat a random neighbor each timestep. A certain amount of energy is introduced to each living organism every time step, and replication occurs when an organism has an adjacent empty grid cell and a sufficient amount of energy.

Each organism has a fixed-length string of letters as its genome. These letters can be upper or lower case, so that each letter is one of 52 possible letters. All but eight letters are inactive and do not influence the interactions between organisms. Of the eight active letters, four are offensive (A,B,C,D) and four are defensive (a,b,c,d).

The predator-prey interactions are determined by organisms' genomes. A particular organism is not predisposed to be predator or prey, and may even be able to eat its own offspring. The comparison between genomes consists of matching contiguous substrings of offensive letters in the organism attempting predation with defensive letters in the prey. If the predator has a sequence of offensive letters that is not matched in the prey by a corresponding defensive string, the prey organism dies and the predator gains a percentage of its energy. A neutral letter or letter of a different type ends a sequence.

This interaction rule satisfies the condition that fitness in the system should depend only on relative quantities as well as the condition that in interactions between different complexities, higher complexities produce a benefit for the organism. If a particular organism only has a defensive string of length $L$, then a predator with an offensive string of length at least $L + 1$ will always be able to eat it; thus there is always a structure at a higher complexity which can bypass a particular defense.

When an organism replicates, its genome is subject to change from mutation and other genetic operations. Point mutations occur at a rate $r_m$ per letter and set the mutated letter to a random letter, which may be the same as the original. Gene duplication occurs at a rate $r_d$. In gene duplication, three random values between zero and the length of the genome are generated: a start position $i_{start}$, ending position $i_{end}$, and an offset $i_{ofs}$. The sequence between $i_{start}$ and $i_{end}$ is stored in memory and written back into the genome starting at $i_{start}+i_{ofs}$. The genome is treated as being periodic as in microbial DNA, so if $i_{end} < i_{start}$ the reading process proceeds through the end of the genome and wraps around to the beginning.

In this system the complexity is taken to be the longest functional string (separated into attack and defense complexities). The motivation for this choice is that it is directly related to the capabilities of the organism. It also represents the interaction between pieces of information in the organism's genome: together, a sequence of multiple letters have a certain functionality that, apart, they would not.

Point mutations do not satisfy the condition that the dynamics should be invariant to changes in complexity. If an organism has a particular active string of length $L$, there are $L$ chances for a point mutation to decrease the complexity, and 2 chances for a point mutation to increase the complexity. More specifically, if a mutation occurs at the first letter before or after the string, there is a 1/13 chance that the length of the active string in-
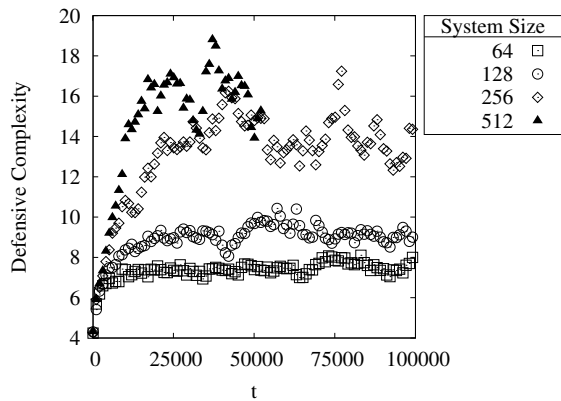
FIG. 1: Defense complexity versus time in Foodchain for system sizes 64, 128, 256 and 512 square grids. Duplication rate is set to 0.1 and mutation rate is set to 0.01.
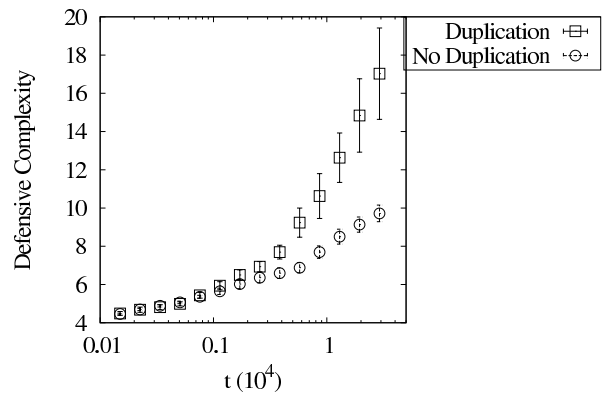


FIG. 2: Effect of gene duplication on the rate of complexity increase. Defense complexity is plotted for gene duplication rates of 0 and 0.1. The system is of linear size 256 and has a mutation rate of 0.001.

creases by 1. If a mutation occurs anywhere within the string, there is a 12/13 chance that the active length will decrease. The average resultant length $L'$ of an active string initially of length $L$ after a single point mutation is given by:

$$\langle L' \rangle = \frac{3}{4}L - \frac{1}{2} - \frac{1}{4L} \qquad (1)$$

The dynamics of point mutations tends to decrease the active length because there are many more ways to decrease it than to increase it. This entropy pressure competes against the selection pressure due to the advantage that results from having a sequence of higher active length. The magnitude of the advantage, and thus the selection pressure, is independent of the absolute sequence length, whereas the entropy pressure scales with the sequence length. Therefore, there is an equilibrium active string length (complexity) at which the entropy pressure is balanced against the selection pressure.

Gene duplication on the other hand operates equally on sequences of different active lengths so long as the active length is much smaller than the total genome length. The probability that the gene duplication region cuts a sequence of length $L$ is $L/L_{genome}$. If a particular sequence is captured, its length will at least be preserved and may increase by an amount proportional to the average sequence length in the organism if the write region is adjacent to another sequence of the same type.

Point mutations are necessary to fully explore the genetic space, but if the point mutation rate is too high, the complexity cascade is inhibited. The next section examines the results of simulations for a variety of point mutation rates and system sizes in order to probe this effect.

Every hundred timesteps the system-wide population, average energy, average attack complexity, and average defense complexity are stored for analysis. The attack and defense complexities are taken to be the longest con-

tiguous string of attack and defense functionality. The simulation is run for different initial random seeds in order to extract the mean behavior of these quantities with simulation time.

*Results:-* The average defensive complexity of organisms in the system as a function of time is plotted in Fig. 1 for different system sizes. These simulations use a gene duplication rate (per replication) of 0.1 and a mutation rate of 0.01 per letter. The complexity increases with time for short times, but then saturates at a value which depends on the system size. We observed that in a system with no gene duplication, the increase in complexity was logarithmic with time, whereas the system with gene duplication exhibited super-logarithmic complexity growth (Fig. 2). Increasing the system size beyond 256 has diminishing returns, as the change from 256 to 512 is less than the change from 128 to 256.

When the mutation rate is decreased to 0.001, the saturation at low system sizes is unchanged, but at high system sizes the saturation point increases. These results are shown in Fig. 3. This suggests that a large mutation rate creates a specific maximum complexity value due to entropy pressure, and that a small system size creates a different specific maximum complexity value. Thus the system will increase in complexity until it reaches the first of those maxima. When the data are plotted in terms of variables which reflect the asymptotic complexity scaling, they collapse onto a single curve. This is analogous to finite size scaling around a critical point in which the system size creates a departure from criticality and causes the scaling to saturate.

The data collapse takes the form of $r^a(C - C_0) = f(r^a S^b)$ where $f(x)$ scales as $x$ when $x \to 0$ and $f(x)$ approaches a constant when $x \to \infty$. The data are found to collapse for $a = 0.6 \pm 0.2$, $b = 2 \pm 0.1$, and $C_0 = 6.65 \pm 0.1$. The error in these quantities was determined by varying them around the point of best collapse and monitoring
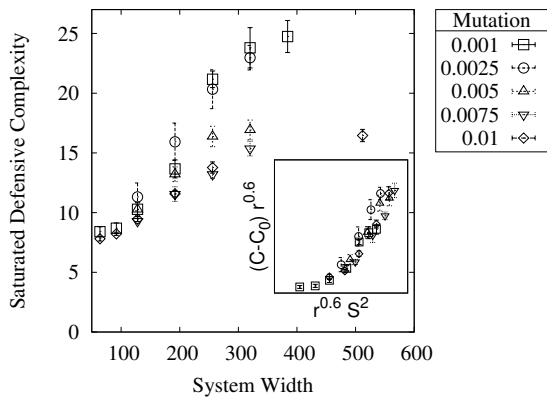
FIG. 3: Dependence of maximum defensive complexity on system size and mutation rate. The inset shows that the data collapse onto a single curve when plotted with a dependent variable $(C - 6.65)r^{0.6}$ and independent variable $r^{0.6}S^2$.

the quality of the collapse. The $S^2$ dependence is indicative that the total population is the relevant quantity when determining finite-size effects. The value of $C_0$ is consistent with the complexity one would generate by randomly generating strings of length 2048 with a proportion of defense characters to alphabet size equal to that observed in the smallest systems. That is to say, at the asymptote corresponding to high mutation rate and low system size, the complexity of strings is due entirely to evolutionary pressures on the relative proportions of the different characters, rather than spatial organization within the genome.

The saturation due to large point mutation rate can be understood as being due to its complexity dependence as discussed earlier and in terms of the Eigen error threshold [21, 22], but the observed scaling exponent is not at this time understood. The system size scaling is surprising as it is not obvious a priori that the complexity of an organism's genome should be related to the size of the space the organism lives in (in contrast with turbulence, in which the complexity of the flow is expressed in the distribution of velocity throughout the system).

It is possible that the connection between system size and complexity in 'Foodchain' is a result of the fixation of complexity-decreasing mutations. For a finite population of organisms with a set of traits that may be present or absent in each organism, the fluctuations in the population and the dynamics of reproduction will eventually cause the trait to be either present or absent in every member of the population. The probability of a particular mutation going to fixation is $P(s) = (1 - \exp(-2s))/(1 - \exp(-4Ns))$, where $s$ is the selective advantage and $N$ is the population size[23, 24, 25].

In the context of the Foodchain model, each organism may have many strings of varying complexities only a few of which are responsible for the organism's reproduc-

tive success. The pivotal strings are not necessarily those of the highest complexity (short defense strings can still be important in defending against short attack strings held by other organisms, for instance). However, a mutation to the most complex string may turn it into a pivotal string even if it is not currently experiencing selective pressure. In the low mutation rate limit fixation of complexity-decreasing mutations imposes a limit on the maximum sustainable length $L$ of a particular string. We balance the rate of fixation of complexity-increasing mutations (which occur at a constant rate) with the rate of fixation of complexity-decreasing mutations (which occur at a rate proportional to $L$): $P(s) - LP(0) = 0$, where $P(0) \propto 1/N$[24]. This results in the scaling $L \propto N$, consistent with the system size scaling exponent observed in the data collapse.

In the simple 'Foodchain' model presented here, there is no separation between primitive organisms that compete with each other using structures of low complexity and organisms with very complex offensive and defensive strings. In order to generate a rich hierarchy of structures, some form of trophic structure would need to be represented in the system[26].

---

[1] C. Adami, Nature Reviews Genetics **7**, 109 (2006).
[2] S. Elena and R. Lenski, Nature Reviews: Genetics **4**, 457 (2003).
[3] C. Wilke and C. Adami, Trends in Ecology & Evolution **17**, 528 (2002).
[4] R. E. Lenski, C. Ofria, T. C. Collier, and C. Adami, Nature **400**, 661 (1999).
[5] C. Adami, C. T. Brown, and M. R. Haggerty, in *Proceedings of the Third European Conference on Advances in Artificial Life* (Springer-Verlag, London, UK, 1995), pp. 503–514.
[6] E. Ostrowski, C. Ofria, and R. Lenski, Am. Nat **169**, E1 (2007).
[7] R. Lenski, C. Ofria, R. T. Pennock, and C. Adami, Nature **423**, 139 (2003).
[8] L. Richardson, *Weather Prediction by Numerical Process* (Cambridge University Press, Cambridge, UK, 1922).
[9] A. N. Kolmogorov, Dokl Acad Nauk USSR **30**, 299 (1941).
[10] S. Wright, Proceedings of the Sixth International Congress on Genetics **1**, 356 (1932).
[11] S. Gavrilets, *Fitness landscapes and the origin of species* (Princeton University Press, Princeton, US, 2004).
[12] H. Orr, Nature Reviews Genetics **6**, 119 (2005).
[13] T. S. Ray, in *Scientific Excellence in Supercomputing: The IBM 1990 Contest Prize Papers*, edited by K. R. Billingsley, E. Derohanes, and I. H Brown (The Baldwin

Press, The University of Georgia, 1991), pp. 489–531.

[14] C. Adami and C. T. Brown, in *Proc. Artificial Life IV*, edited by R. Brooks and P. Maes (MIT Press, US, 1994), pp. 377–381.

[15] R. K. Standish, International Journal of Computational Intelligence and Applications **3** (2002).

[16] C. Adami, Complexity **8**, 49 (2002).

[17] G. Caldarelli, P. Higgs, and A. McKane, Journal of Theoretical Biology **193**, 345 (1998).

[18] B. Drossel, P. G. Higgs, and A. J. McKane, J. Theor. Biol. **208**, 91 (2001).

[19] A. McKane, The European Physical Journal B-Condensed Matter **38**, 287 (2004).

[20] M. Maron and C. T. Fernando, in *Complexity Workshop, Artificial Life X* (2006).

[21] M. Eigen, Naturwissenschaften **10**, 465 (1971).

[22] C. Wilke, BMC Evol Biol **5**, 44 (2005).

[23] S. Wright, Genetics **16**, 97 (1931).

[24] M. Kimura, Genetics **47**, 713 (1962).

[25] H. Orr, Genetics **155**, 961 (2000).

[26] B. Drossel, A. McKane, and C. Quince, Journal of Theoretical Biology **229**, 539 (2004).