

Chaos and Evolution

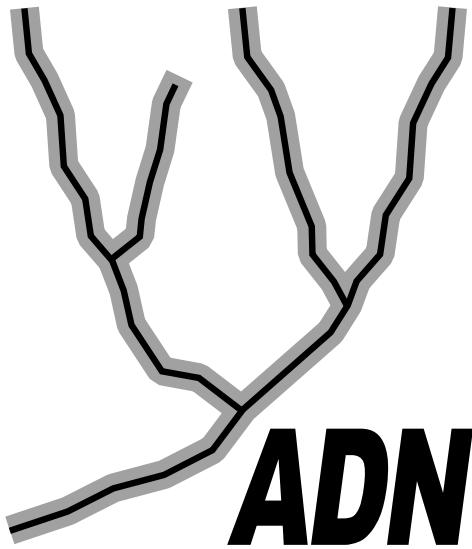
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1996



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The Adaptive Dynamics Network at IIASA fosters the development of new mathematical and conceptual techniques for understanding the evolution of complex adaptive systems.

Focusing on these long-term implications of adaptive processes in systems of limited growth, the Adaptive Dynamics Network brings together scientists and institutions from around the world with IIASA acting as the central node.

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The pivotal role of evolutionary theory in life sciences derives from its capability to provide causal explanations for phenomena that are highly improbable in the physicochemical sense. Yet, until recently, many facts in biology could not be accounted for in the light of evolution. Just as physicists for a long time ignored the presence of chaos, these phenomena were basically not perceived by biologists.

Two examples illustrate this assertion. Although Darwin's publication of "The Origin of Species" sparked off the whole evolutionary revolution, oddly enough, the population genetic framework underlying the modern synthesis holds no clues to speciation events. A second illustration is the more recently appreciated issue of jump increases in biological complexity that result from the aggregation of individuals into mutualistic wholes.

These and many more problems possess a common source: the interactions of individuals are bound to change the environments these individuals live in. By closing the feedback loop in the evolutionary explanation, a new mathematical theory of the evolution of complex adaptive systems arises. It is this general theoretical option that lies at the core of the emerging field of adaptive dynamics. In consequence a major promise of adaptive dynamics studies is to elucidate the long-term effects of the interactions between ecological and evolutionary processes.

A commitment to interfacing the theory with empirical applications is necessary both for validation and for management problems. For example, empirical evidence indicates that to control pests and diseases or to achieve sustainable harvesting of renewable resources evolutionary deliberation is already crucial on the time scale of two decades.

The Adaptive Dynamics Network has as its primary objective the development of mathematical tools for the analysis of adaptive systems inside and outside the biological realm.

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Abstract

There is growing interest in applying nonlinear methods to evolutionary biology. With good reason: the living world is full of nonlinearities, responsible for steady states, regular oscillations, and chaos in biological systems. Evolutionists may find nonlinear dynamics important in studying short-term dynamics of changes in genotype frequency, and in understanding selection and its constraints. More speculatively, dynamical systems theory may be important because nonlinear fluctuations in some traits may sometimes be favoured by selection, and because some long-run patterns of evolutionary change could be described using these methods.

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Chaos and Evolution

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The idea that nonlinear dynamics (Box 1), and especially deterministic chaos, play an important role in biology has become more influential in recent years. Chaos has been suspected, and sometimes firmly documented, at the biochemical^{1,2}, organismal^{3,4}, and population^{5,6} level. Nonlinear changes in phenotypes, gene frequencies, or population sizes must affect the evolution of character traits (and clades), and the ways we study evolution.

In linear systems, only two kinds of long-term dynamics are possible: either the system stays constant, or it grows or declines at a constant rate. A familiar ecological example is the model for density-independent population growth,

$$N_{t+1} = rN_t$$

the population stays constant if

$$r = 1,$$

and grows or declines at the constant rate r if r is greater or less than 1, respectively.

Box 1. Types of nonlinear dynamics

Three types of dynamics relevant to biology can only occur in nonlinear systems: limit cycles, quasicycles, and chaos. A dynamical system may be 'attracting' — i.e., the system resumes its dynamics after a perturbation — under these regimes as well as under equilibrational dynamics.

Systems on limit cycles repeat themselves regularly. Neighbouring points on a limit cycle stay near one another; if one population is at the high point in this period-two cycle when another is at the low point, they will stay exactly out of phase.

Quasicycles resemble limit cycles, but the periods of the oscillations vary; the system never precisely repeats itself. Neighbouring points remain near one another in quasicycles. Quasicycles often occur when periodic subsystems (e.g., physiological cycles) are coupled.

Chaotic oscillations do not have regular periods or amplitudes. Neighbouring points tend to separate from one another at an exponential rate. This is why one cannot predict the system's behavior for more than a short time, even though the underlying mechanisms are deterministic.

Intuitively this sort of model suggests that most biological systems are nonlinear:

population size and other biological variables clearly vary over time without either disappearing or growing infinitely. Indeed, most biological models — whether describing physiology, changes in gene frequencies, population growth, or interacting populations — are nonlinear. Such nonlinear systems can have several kinds of behavior not possible in linear systems, including cycles, quasicycles, or chaos (see Box 1). “Nonlinear dynamics” refers to these dynamical regimes that can occur only in nonlinear systems, and terms like “nonlinear methods” refer to special methods for analyzing them.

There are four areas where nonlinear dynamics may prove important to evolutionists. First, because the evolutionary process — as modeled in population genetic equations — itself is nonlinear, one can use nonlinear dynamics theory to analyze gene frequency change in response to deterministic forces like selection. Second, because many physiological and developmental mechanisms are nonlinear, the outcome of selection can be limited (or expanded) in ways that would not be anticipated without a nonlinear perspective. Third (and more speculatively), selection may sometimes promote nonlinear dynamics, including chaos, in some traits. Fourth (and still more speculatively), one can use nonlinear approaches to consider how adaptive peaks change as external conditions change.

Nonlinear dynamics and the genetics of populations

Even in simple models, gene and haplotype frequencies, and mean fitness, can change cyclically or chaotically. When such dynamics occur, Wrightian adaptive topographies no longer exist, because no fitness function⁷ can be maximized. Perhaps more important, the traditional problem of explaining the maintenance of genetic diversity can disappear. We briefly review some results, and then consider their implications for evolutionary biology.

Simple genetic models

Complex dynamics can occur even under constant selection. Stable limit cycles — in gene and haplotype frequencies and mean fitness — can occur even in two-locus, two-allele models with constant selection and epistasis^{8,9}, and in one-locus, two-allele models with fertility selection¹⁰.

Recombination may have varied effects in multilocus models, depending on selective regimes or genetic systems. For constant selection, complex dynamics are most likely to occur under intermediate recombination rates¹¹. This is because Fisher's 'fundamental theorem of natural selection' applies when recombination rates are high (because the loci evolve almost independently) or low (because chromosomes behave like single genes). Populations therefore move up fitness gradients¹². On the other hand, in a haploid frequency-dependent model, intermediate recombination rates tended to stabilize dynamics¹³. Clearly the problem of the dynamical consequences of recombination is still a wide-open field.

Models with more ecological realism

Cycles and chaos occur more easily under frequency- and density-dependent selection. Cycles result from frequency-dependence in models of the evolution of recombination¹⁴ and of Dawkins' "battle of the sexes" game¹⁵. Both cycles and chaos occur in linear frequency-dependent models⁷ and in models that allow both gene frequencies and population size to vary¹⁶⁻¹⁸.

Complex dynamics — and the coexistence of multiple genotypes — may thus be more likely in more ecologically realistic models. Quasicycles arise in a model of the evolution of gynodioecy¹⁹; because the parameter space was only partly explored, chaos may also occur in this model. A host-pathogen coevolutionary model by May and Anderson²⁰ shows a wider range of behaviour. If population size is constant at carrying capacity, selection is frequency-dependent, and (for plausible parameter values) can lead to equilibria, cycles, or chaos. If population size is regulated by the disease, both gene frequency and population size can fluctuate cyclically or chaotically. Finally, Holt²¹ showed that when population dynamics are cyclic or chaotic, peripheral populations may maintain more genetic variation than they would under equilibrial dynamics.

Implications for population genetic studies

Adding population dynamics to population genetic models not only makes complex dynamics more likely; it also points to the danger of the traditional assumption that population size can be ignored. A recent phenotypic model of the evolution of conspecific brood parasitism²² showed that the dynamics of phenotype frequencies and population size generally depend on one another, except in the special case of populations tending to stable equilibria.

These studies show that chaotic gene frequency changes are possible, but we do not know whether they actually occur. This issue can only be resolved empirically²³. Unfortunately there are no extant data sets adequate to test such a hypothesis for even a single population. At present we can point only to the possibility of chaotic change in gene frequencies.

It should be possible in the coming years to change this situation through carefully designed studies. These would need to use short-lived organisms to obtain sufficiently long time series, and genotypes would need to be readily identifiable. Knowledge is growing rapidly of specific loci and their effects in organisms like *Caenorhabditis*, *Drosophila* and *Tribolium*. Methods for identifying genotypes are becoming faster and more affordable. Methods for testing hypotheses about dynamics are becoming more sophisticated^{5,23}. For these reasons, we believe that well-designed laboratory studies of genetic dynamics may soon be within reach.

These results suggest caution in the application of optimality approaches. The central assumption of these models — that some quantity like mean fitness is maximized — may often be violated. ESS approaches have a similar difficulty because ESS's are defined as joint, constrained local optima. This does not negate the potential usefulness of optimality and ESS approaches in studying selection — only in predicting its outcome.

Nonlinear dynamics and gene substitutions

In the previous section we discussed ways in which nonlinear dynamics can promote the coexistence of multiple genotypes. However, nonlinear genetic dynamics can also promote gene substitutions.

Complex spatial patterns — including spiral waves and chaos — can develop in nonlinear models of host-parasitoid systems²⁴. Without a spatial component to the model, inefficient parasitoid genotypes fail to invade the population. Surprisingly, adding a spatial component creates new nonlinearities that allow mutants to invade, depending on where they are introduced. These invasions can lead to complete replacement²⁵. Thus, the emergence of spatial patterns determines the outcome of natural selection. This result calls into question the definition of 'invasibility,' which underlies much of population genetic theory.

Even without spatial dynamics, we speculate that there may be times when genotypes that are usually less fit can invade a chaotic resident population. This is because the complex shape of the chaotic attractor may include small regions where the net effect of the invader-resident interaction favours the “less fit” mutants. Invasions may succeed when the resident population is on such a vulnerable part of its attractor, while they would be unable to do so the rest of the time. This speculation requires that the invaders appear in sufficiently large numbers that their initial dynamics are initially nonlinear (they are approximately linear if initial numbers are very small).

Can the nonlinear dynamics underlying phenotypes sway selection?

The nonlinear dynamics of biochemical, cellular, physiological, neuronal and ecological systems can limit or enlarge the range of phenotypes reachable by selection. Small changes in parameters governing the system can cause it to qualitatively change its dynamics, that is, to 'bifurcate', say, from a steady-state to a stable limit cycle (Box 2).

Box 2. What are bifurcations?

To understand what bifurcations are, consider one of the simplest nonlinear population models, the logistic map

$$X_{t+1} = r X_t(1-X_t).$$

For $r < 3$, the system reaches a steady state. At this “bifurcation point,” the stable equilibrium suddenly becomes unstable, and a stable limit cycle of period 2 simultaneously appears. Further increases in r cause a sequence of bifurcations in which the stable limit cycle of period n becomes unstable and simultaneously a new stable cycle of period $2n$ appears. As r passes the value 3.57, a chaotic attractor appears. Even within the chaotic region, there are bifurcation points; for example, there are values of $3.57 < r < 4$ for which there are stable limit cycles.

Bifurcations occur in physiologically plausible models of the cell cycle²⁶, ventilation rate, and many other physiological processes³. In the cell cycle model, changing a rate

parameter can cause the system to move between cycle periods that vary chaotically and periods that are constant²⁶. In the ventilation model, changing a rate parameter can cause the system to move between normal and arrhythmic breathing³. There is some empirical evidence that these bifurcations also occur in real physiological systems³. Bifurcations also occur in models of population dynamics; an ongoing series of experiments with *Tribolium* beetles has shown that a biologically-based model of population dynamics can predict bifurcations that can be induced in the laboratory²⁷. Thus, bifurcations may be built in to some basic physiological and demographic processes.

We suggest that bifurcations can sometimes correspond to abrupt changes in the range of phenotypes reachable by selection, regardless of whether they are bifurcations in physiological or population dynamics. We outline the sense in which we believe that bifurcations may sometimes affect selective outcomes, and the sense in which this kind of effect may be similar regardless of whether the traits involved are physiological, developmental, or demographic.

As a first example, consider a physiological trait with a stable cycle of period p , governed by a parameter m . Selection might favour a slight increase in m with the same period. Near a bifurcation of the underlying physiology, substitution of an 'increased- m ' allele would not have this effect: increasing m causes a change to a cycle of different period (or to chaos). As a second example, consider a trait related to the intrinsic rate of population growth r , such as fecundity at some age i , b_i . Again, selection might favour a slight increase in b_i if the increase led to a larger steady-state population, but not if it led to the onset of population cycles. Near a bifurcation of the population dynamics, this favored phenotype cannot be achieved.

This purely verbal argument is supported by a theoretical study of the evolution of iteroparity versus semelparity^{28,29}. Ferrière and Gatto (Box 3) found that selection can favour one or the other life history, depending on whether bifurcations in population dynamics occur along a trade-off curve between adult survival and juvenile recruitment.

Bifurcations also restrict the applicability of some widely used models. Quantitative genetic approaches assume that fitness varies smoothly as a function of small allelic substitutions. This is not necessarily true near bifurcations of the physiological or population dynamics (and need not be true in nonlinear systems anyway). Optimality models are also likely to be misleading near such bifurcations, because fitness may not vary smoothly as a function of phenotypic value, and because some phenotypes may not be reachable.

Is chaos sometimes favoured by selection?

If physiological or population dynamics are sometimes chaotic, it seems natural to ask whether this is because chaos is caused by selection. Two distinct cases are possible: chaos itself may be favoured by selection, or it may be a byproduct of selection on another trait³⁰. We cannot presently distinguish between these hypotheses for any instance of biological chaos, but it is worth speculating about at least two kinds of mechanisms suggested by Conrad³¹ as candidates for adaptive chaos.

First, chaos may be adaptive in some cases of *defence behaviours*. An example may occur in the behaviour of foraging birds³². Bird vigilance can be characterized by the durations of scans (when the bird lifts its head and looks around) that alternate with feeding periods. Data from some species whose vigilance is likely to be anti-predatory (sandpipers, doves, finches) show typical features of chaos, such as high short-term predictability of scan and interscan durations, which decays exponentially over time. The long-term unpredictability may prevent waiting predators from timing their attack accurately, while the short-term predictability permits animals feeding in groups to improve their joint vigilance. This coordination also allows a whole flock to reduce the amount of time spent without surveillance by any of its members.

Second, chaos can provide maintenance and disturbance dissipation mechanisms: systems whose parts vary independently can be more efficient and persistent than tightly coupled systems. For example, chaos may help to prevent entrainment in neural networks. Physiologically plausible models of neurons can exhibit chaos³. It is conceivable that, without chaos, either very dull pacemaker activity, or highly explosive global neural firing patterns, would emerge. Neural chaos may maintain the functional independence of different parts of the nervous system; it could also provide a basis for adaptive chaos in behaviour.

Another important example was recently provided by Schaffer and collaborators³³. In challenging the view that group selection should make chaos rare in natural populations^{34,35}, they considered a spatially subdivided metapopulation whose local dynamics are governed by the Ricker or logistic map, plus local noise. This metapopulation was subject to stochastic global perturbations. Metapopulations with chaotic dynamics persisted longer than those with equilibrial or cyclic dynamics; cycles generally produced the least persistent metapopulation. These results are due to chaos' property of sensitive dependence on initial conditions, which promotes asynchrony and decoupling among local populations, increasing metapopulations' ability to withstand perturbations.

Adaptive chaos: could it really evolve?

If there are sometimes long-term group-level advantages to chaotic population dynamics, are there ever short-term, individual advantages? A life history study^{28,29} (Box 3) showed that selection on several demographic parameters should often lead to chaos, especially in late-maturing species and when selection is constrained by steep trade-offs between traits.

Should we expect chaotic or stochastic mechanisms to be favoured, where temporal variability is advantageous as a basis for defence behaviours or for maintenance and disturbance dissipation? Chaotic mechanisms may more often evolve for several reasons: (1) Biological systems are often intrinsically nonlinear — the 'raw material' for generating chaos is already present. For example, population dynamics are generally nonlinear because of density-dependence. Chaos may thus be an easy way to generate variability and uncertainty. (2) By contrast with random noise, chaos involves high short-term predictability that may also be selectively advantageous. It may be more difficult for organisms to achieve the same ends with autocorrelated stochastic processes. (3) Even if stochasticity is intrinsically generated by a biological system, it may not produce sufficient levels of variability. Because chaotic systems have sensitive

dependence on initial conditions, chaos can serve to magnify the internal randomness of the system.

Box 3. Nonlinear dynamics and the evolution of itero- vs. semelparity

Ferrière and Gatto²⁸ studied the evolution of adult survival S and recruitment to adulthood P , when there is a tradeoff between them (bold curves in figures below). What phenotype $\{S, P\}$ is the ESS? To each resident phenotype pair $\{S_{res}, P_{res}\}$ we can attach an invasion boundary — a curve separating phenotypes able to outcompete the residents from those outcompeted by the residents. If the invasion boundary of a phenotype is just tangent to the trade-off curve, that phenotype is an ESS.

The dynamics of the resident population greatly affect the evolutionary outcome. Ferrière and Gatto used a delayed Ricker model that assumes overcompensatory density-dependence (but other density-dependent functions would give similar results):

$$X_{t+1}^{res} = P X_{t-\alpha+1}^{res} e^{-(X_t^{res} + X_{t-1}^{res} + \dots + X_{t-\alpha+1}^{res})} + S X_t^{res}$$

where X_t^{res} denotes the adult resident population size at time t .

The shape of the invasion boundary is thus crucial, and it depends on the dynamics of the resident population³⁷. It is a straight line if the dynamics are equilibrial, and a strictly convex curve if the dynamics are nonlinear. The invasion boundary of any resident phenotype passes through $\{S_{res}, P_{res}\}$ itself, and through $(S=1, P=0)$.

If the trade-off curve is entirely contained in the region of stable equilibria (a), the ESS is semelparous. If part of the trade-off curve is in the region of nonlinear dynamics [which may happen for a different value of age at maturity a (b)], the ESS is iteroparous. Finally, a polymorphism can be an ESS only if at least one of the phenotypes involved belongs to the region of complex dynamics (c).

Chaos may be selectively favourable in some circumstances, but cycles or equilibria are surely favoured in others. It is time, however, to abandon the prejudice that selection always favours constancy at the biochemical, organismic, and population levels.

Nonlinear dynamics in long-term evolution

The evolutionary walk along a limited-mutation pathway

In the discussion above, we considered evolution in the neighbourhoods of evolutionary attractors — cycles, quasicycles, and chaotic attractors — that did not change. But changes in selection and in the availability of mutant genotypes can affect both the location and the qualitative nature of these attractors. Recently there has been much interest in using nonlinear methods to study these long-term evolutionary changes.

Metz and his collaborators³⁶ have analyzed how evolutionarily stable strategies (ESS's) come into being and disappear, and how they become (or cease to be) attracting, as the environment changes. An ESS is a combination of traits such that, once established in a population, cannot be outcompeted by any other feasible phenotypes. Their theory assumes clonal reproduction or haploid genetics, and that mutations occur randomly but only one at a time (thus response to selection is strictly mutation-limited).

This theory also assumes that the sign of a simple function — the invader's long-term stochastic growth rate — decides the outcome of an attempt by phenotype Y to invade the set of resident phenotypes X . This growth rate is calculated as the dominant Lyapunov exponent (Box 4) of the resident community dynamics^{37,38} (Box 4). Clearly a population growth rate like this is not a fixed quantity — it depends on the environment and on the composition and dynamics of the resident population. Therefore it can change if the resident population evolves.

Box 4. Lyapunov exponents and their use for evolutionists

Lyapunov exponents provide a way to identify the qualitative dynamics of a system — e.g., deciding whether it is chaotic. They can also be used to ask whether a system is invasible. This is because they describe the rate at which neighbouring trajectories converge or diverge (if negative or positive, respectively) from one another in orthogonal directions. If the dynamics occur in an n -dimensional system, there are n exponents.

Ecologists may notice that Lyapunov exponents are similar to the eigenvalues used in local stability analysis. In fact, they are generalizations of these eigenvalues. Instead of describing what happens near a fixed point, Lyapunov exponents describe what happens near an entire trajectory.

To ask whether a system is chaotic, we find the Lyapunov exponents of the linearized system near a trajectory. Since chaos can be defined as divergence (on average) between neighbouring trajectories, the presence of a positive exponent is diagnostic of chaos.

To ask whether a population is invasible, we study a new system: the resident population's dynamics embedded in an additional dimension (the number of invaders). Information on invasibility is given by the exponent corresponding to the new dimension. Positive values mean that the system diverges on average from the old attractor in this direction: the number of invaders will grow. Since Lyapunov exponents give the rates of divergence or convergence, this exponent is the long-term stochastic growth rate of the invader population.

The central point of this theory is that the meanders of evolution reflect the randomness of mutation, but major trends in evolving traits are deterministic. These trends are determined by 'singular phenotypes' (Box 5), which move and change their nature (bifurcate) as the environment changes.

Bifurcations and long-term evolution

Metz *et al.* classified the ways singular phenotypes can bifurcate in trait space. For example, an ESS (attracting or not) can be created or can disappear; a monomorphic ESS can become polymorphic, or vice versa. The analyses do not only concern ESS's: Lyapunov-unstable singular points can also occur. These points are like saddle-points:

populations initially approach them, but are subsequently repelled from them. These points can bifurcate into attracting ESSs, or vice versa.

Such bifurcations are local: they occur when the local stability of a singular phenotype changes. Global bifurcations can occur as well, when a singular phenotype abruptly appears or disappears (Box 6). One way to interpret local bifurcations is as models for long-term changes in the genetic composition of a population, while global bifurcations could describe speciation and extinction. A study of a predator-prey-resource model, revealed a global bifurcation pattern that resembles a succession of stasis, punctuated equilibrium and gradualism³⁹.

Box 5. Singular phenotypes and adaptive dynamics

Here we illustrate the Metz *et al.* theory of adaptive dynamics³⁶ for the case where the resident type can be described by one variable. The time unit of the dynamics is that taken to lose or fix a new mutation. Whether the mutant with trait Y can invade the resident with trait X is determined by the sign of the long-term stochastic growth rate of the invader — the dominant Lyapunov exponent (Box 4) of the X -type's dynamics^{37,38}.

$s_X(Y) > 0$: the mutant increases in frequency

$s_X(Y) < 0$: the mutant goes extinct.

What happens where $s_X(Y)$ changes sign? *Phenotypic singular points* are the points where the line separating the negative from the positive values intersects the 45° line (see figure). An ESS is a special case of such a singular point. Using linear stability analysis of $s_X(Y)$ around a singular point \hat{X} , Metz *et al.* classified the properties of singular phenotypes. Defining

$$c_{12} = \frac{1}{2} \frac{\partial^2 s_X(Y)}{\partial X \partial Y} \Big|_{Y=X=\hat{X}}, \text{ and}$$

$$c_{22} = \frac{1}{2} \frac{\partial^2 s_X(Y)}{\partial Y^2} \Big|_{Y=X=\hat{X}}, \text{ they showed that:}$$

- If $c_{22} < 0$, X is a local ESS; if $c_{22} > 0$, X is an “Evolutionarily Unstable Strategy” (EUS).
- ESSs and EUSs can be either attracting ($c_{12} < -c_{22}$) or repelling ($c_{12} > -c_{22}$).
- Attracting EUSs are surrounded by mutually invisable phenotypes, and the same applies to approachable ESSs if

$$c_{12} < 0.$$

This approach to evolutionary theory has important strengths. It provides an explicit (and explorable) connection between ecology and long-term evolution. Perhaps more important, it encourages study of how evolutionary and ecological changes on different timescales are interrelated.

Box 6. Local and global bifurcations in long-term evolution

When an environmental parameter ε changes, the long-term stochastic growth rate $s_X(Y)$ also changes. This can cause bifurcations in the singular phenotypes (Box 5).

For example, consider the case where two phenotypes compete, with Lotka-Volterra dynamics³⁶:

$$\frac{dn_{X_i}}{dt} = r_{X_i} \left[1 - \frac{\sum_j \alpha_{X_i X_j} n_{X_j}}{K_{X_i}} \right] n_{X_i} ,$$

with $r_{X_i} = 1 - X_i$, $\alpha_{X_i X_j} = e^{-\varepsilon(X_i - X_j)^2}$ and $K_{X_i} = e^{-X_i^2}$.

There is a singular point at $\hat{X} = 0$, which is an attracting ESS for small ε . As ε increases beyond 3, the resident population becomes invadable by nearby mutants, and then stable polymorphisms can develop (a). The curves in (a) give the mutant invasion rate $s_X(Y)$. Further increases in ε change the shape of the mutant invasion rate still more, allowing additional phenotypes to invade, and others to go extinct (a). The distribution of phenotypes over time is shown in the evolutionary tree in (b); the dashed vertical bars indicate the trait values that are numerous at that time, and the numbered points correspond to the graphs in (a).

These local bifurcations occur when the local stability of a singular phenotypic point (Box 5) changes³⁶. We show a schematic example in (c, left). The singular trait X_0 initially is an ESS: for any mutant trait Y , we have $s_{X_0}(Y) < 0$. As ε increases, the shape of $s_{X_0}(Y)$ changes (solid curves depict its graph before bifurcation; dashed curves, afterwards), and X_0 , though it remains a singular phenotype, loses its evolutionary stability. Instead, an attracting protected polymorphism develops.

A global bifurcation corresponds to the disappearance of a singular phenotype (c, right). Notice that even gradual changes in $s_X(Y)$ can cause the property of singularity to be passed on to a quite different phenotype (c). Thus a wild change in $s_X(Y)$ is not required to generate a pattern reminiscent of punctuated equilibrium.

On the other hand, there are still important limitations to the theory. First, it is not yet explicitly genetic, so it can only be treated as heuristic. Second, the dominant Lyapunov exponent may not always be an appropriate invasibility criterion. Finally, continuous mutations (instead of one-step mutations) are known to influence the evolutionary walk⁴⁰. Thus, there are important challenges ahead for this theory.

Conclusion

No simple dynamical system can be expected to capture the intricate nature of long-term evolution⁴¹. Attempts in this direction have nevertheless begun to appear in the literature. These mostly rely on verbal arguments, are not convincing, and mainly show how one can misinterpret the ideas of nonlinear dynamics theory, by treating

mathematical ideas as vague metaphors⁴²⁻⁴⁴. We think that the jargon of nonlinear dynamics can obfuscate, rather than clarify, when divorced from its mathematical roots.

On the other hand, ideas and methods from the theory of nonlinear dynamics may prove to be important to evolutionists. We have examined several distinct issues in the relationship between nonlinear dynamics and evolution. One issue is the effect of complex population and genetical dynamics on adaptation. When these nonlinearities operate, evolution cannot be assumed to be an adaptive process, and Wrightian fitness landscapes are not defined. Standard evolutionary models may thus be more limited in their scope than is generally appreciated. The second issue is whether short-term nonlinear dynamics at the phenotypic level can be adaptive. Chaos is not necessarily an indicator of pathology; under some circumstances, natural selection may favour chaotic phenotypes. A final issue is whether large-scale evolutionary patterns can usefully be studied using methods from nonlinear dynamics. An initial theory attempting to do this is still in its early stages of development, but may prove important in organizing thinking about how ecology, population genetics, and macroevolutionary patterns are interconnected.

Thus, chaos and other nonlinear phenomena can be important to evolutionists in several ways. Adding nonlinear analysis to the evolutionists' tool kit will require a grounding in the appropriate quantitative methods. We believe the potential benefits are well worth the effort.

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