Ecosystems as Complex Systems

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1 Introduction

Population biologists often attribute the stability of natural ecosystems to their inherent complexity, that is to the enormous number of species within them and to the multitude of interactions between these species. Simple models of few species predator-prey systems often exhibit instability, as do human experiments in single-crop agriculture. But in the tropical rainforests, where diversity is the rule, the ecosystem often remains the same, despite perturbations. In some manner, the wealth of interactions within a rich ecosystem tends to maintain that ecosystem in a stable equilibrium, meaning that the species' populations and amounts of different resources tend to remain the same despite small perturbations to the system from the outside.

Evolutionary biologists also know that populations of species can sometimes adapt to different environments quite easily. For example, during the Industrial Revolution in Britain in the nineteenth century, the smoke from the factories often accumulated on the ground as soot. Before this revolution, a usually white moth, Biston Betularia proliferated the countryside. But during the days of the sooty revolution, the common form of the moth changed from white to black, thereby taking advantage of the black surroundings and avoiding flying predators. Hence, the population of Biston Betularia had adapted to better suit its changed environment.

How does an ecosystem, or populations of species therein, maintain its (their) stability, and at the same time maintain its (their) ability to adapt to novel perturbations in the ecosystem itself? The answer lies in Dobzhansky's famous quote about normality: "The norm is diversity." In the following paper I will describe some of the mechanisms that give rise to diversity in ecosystems, and how this might affect the stability and adaptability of these ecosystems.

2 Genotype v. Phenotype

Ever since the landmark pea-crossing experiments of the monk Mendel in the nineteenth century, biologists have debated about the determinants of the characters of an individual organism, and how they are inherited. Some say that environmental factors completely determine the organism, while others say that one's ancestral line ultimately decides the organism's shape. Of course, the present-day wisdom says that a combination of the two factors: environmental and hereditary, is the shaper of the individual organism.

Standard genetics says that an organism's phenotype is completely determined by its genotype and its environment. The organism's genome, inherited at birth from a mixture of its parents' genomes, is composed of discrete pairs of bits called alleles, one coming from each parent. The particular combination of alleles at a particular genetic site determines the genotype of that organism. For example, if an organism's mother gave the organism an allele for red eye color, and the organism's father gave the organism an allele for white eye color, the organism's eye color genotype will be red/white. But the organism, will in general not have one red eye and one white eye, nor will have pink eyes, nor red, nor white. This depends upon the interaction of the organisms' genotype with the environment. In hot environments, the organism might have red eyes, while in cold environments, the organism might have white eyes. The organisms' expression of its genotype in a particular environment is called its phenotype. The dependence of an organism's phenotype on environment, for fixed genotype, is called the norm of reaction.

As examples of different norms of reaction, I present a figure illustrating several different dependences of phenotype on environment for fixed genotype. Notice in particular the highly nonlinear case C., which in aphids represents the effect of population density on the growth of wings.

In general, an organism's phenotype for a particular character is determined by many allelic sites, or genes. This degeneracy is called polygeny. At the same time, each gene affects several characters. This multiplicity is called pleiotropy. With polygeny and pleiotropy, understanding the effects of genetic and environmental differences upon the organism's phenotype is non-trivial.

As an example of pleiotropy, I present a figure illustrating the effect of the substitution of the amino acid valine for the amino acid glutamic acid at position 6 of the β chain of human hemoglobin, which results in sickle-cell anemia. Notice that this seemingly trivial substitution has highly deleterious effects, namely the destruction of the sickle cells and a clogged-up circulatory system, which then cause many other physiological side-effects.

2.1 Mutation

One might think that the units of genetics, alleles, cannot not change, but au contraire, the change of alleles, by genetic recombination, or genetic cross-over, is one of the main factors that allows populations of organisms to adapt to novel environments. This genetic recombination, often called mutation, is most often deleterious, resulting in the death, or inviability (cannot have children). But sometimes, the mutation is not deleterious, and actually enhances that allele's chance of being passed onto future generations. Thus mutation can be a source of genetic variation in a population. An increase of variation due to accumulation of spontaneous mutations can increase the mean viability of the population as a whole But mutation does not happen as often as you might think, and it is a random process, so that it can be "wished" for by a dreamer wanting to be some other species. Also, we must keep in the back of our minds, that an organism is born with mutations, by definition; an organism may not develop a mutation and hope to pass it down to its children. I present a table of some mutation rates per allelic site in various species. Notice that the Drosophila Melanogaster mutation rate per generation per genome is much higher than the others. Drosophila Melanogaster's mutation rate was measured per diploid genome per generation of flies, not per generation of cells, as in other species. Also notice, that in general, the mutation rates are small but finite, so that mutation can serve as a strong diversifying force on long time scales.

Species	Base Pairs per genome	Mut. Rate per Genome per generation
Bacteriophage Lambda	4.7×10^4	0.001
Bacteriophage T4	1.8×10^5	0.002
Salmonella typhimurium	3.8×10^6	0.001
Escherichia Coli	3.8×10^6	0.002
Neurospora crassa	4.5×10^7	0.003
Drosophila Melanogaster	4.0×10^8	0.93

3 The Hardy-Weinberg Theorem & Variation

If we assume that the population size is infinite, that we have random mating, that we have no selection of subpopulations due to fitness differences and that we have no allele flow from external populations. We also assume that in our population, at a particular allelic site of n possible alleles, that we have a probability p_n that we have an allele of type n. Since we have random mating, and equal viabilities of each allele, it is trivial (using Mendel's laws of gene transmission) to prove that p_n remains constant for all generations. We do not care about the phenotypic effect of the combination of the i^{th} and the j^{th} allele and the environment, nor the genotype of the organism. But with the aforementioned conditions, allele frequencies are conserved quantities. This lack of evolution is called the Hardy-Weinberg equilibrium, with the equilibrium reached in only one generation. This is called a neutral equilibrium since every different set of initial gene frequencies gives rise to a different equilibrium. An equilibrium's basin of attraction has measure zero, yet there are an uncountable number of different equilibria, assuming smoothness of gene probability.

However, if we start to relax some of the above assumptions, we find that our conserved quantities are no longer conserved. In this section I will try to enumerate the different effects a relaxed Hardy-Weinberg assumption has on the time evolution of gene frequencies.

3.1 Sex-linked alleles

Consider the HW model with a sex-linked allele, that is the X chromosome carries the allele. Since females have two X chromosomes, and males have one

X chromosome and one Y chromosome, this disparity will cause evolution of gene frequencies with time.This evolution can be seen from the equation:

$$p_{t+1} = p_0 + (p_1 - p_0) \times \left[\frac{1 - (-\frac{1}{2})^{t+1}}{1 - (-\frac{1}{2})} \right]$$

However, $\lim_{n \to \infty} p_{n,t} = p_{nHW}$, that is as the number of generations gets large, the difference between the sex-linked gene frequencies and the Hardy-Weinberg Equilibrium gene frequencies goes to zero. I have included a graph of the time evolution of the above equation for gene frequency. Notice the large oscillations.

3.2 Finite Population Size

Another assumption of the Hardy-Weinberg equilibrium is the infinite population size. When a modeller decides to relax this assumption, he will see random genetic drift creep into his simulation. Suppose we have N diploid members of our population, and that N is fixed for all time. Thus we consider $2N$ total alleles as being our gene pool. Suppose that at generation 0, the gene pool has i_n copies of allele A_n, and that there are $M < 2N$ different alleles. By the Wright-Fisher markovian model, we can determine the probability $P_{i_n j_n}$ that given i_n copies of A_n in one generation, there are j_n copies of A_n in the next generation. We find:

$$P_{i_n j_n} = \begin{pmatrix} 2N \\ j_1 .. j_M \end{pmatrix} \prod_k \left(\frac{i_k}{2N} \right)^{j_k}$$

The eigenvalues of this matrix are difficult to calculate, but we know that the largest nonunit eigenvalue is the one that dominates the dynamics of the system of allele frequencies. The largest nonunit eigenvalue is $1 - \frac{1}{2N}$. Thus the rate of convergence to fixation is $1 - \frac{1}{2N}$ per generation. Therefore, for large N, we have stability of the equilibrium allele frequency, yet for small N, we see the phenomenom of random genetic drift, or small sampling effect, which is a destabilizing factor in populations. This allows a small population to easily go from fixation of one allele to fixation at another allele, which might be introduced by a mutation, or by a slight gene flow from another population.

3.3 Heterozygosity & Inbreeding

Another effect of finite population size is can be seen by measuring the heterozygosity of the population. An organism is called homozygous at an allelic site if both of the alleles are the same. Otherwise the organism is heterozygous at that allic site.The heterozygosity is the percentage of the population which is heterozygous at a particular allelic site. Heterozygosity is a measure of genetic diversity of the population. A population with a large, stable heterozygous fraction at equilibrium is said to exhibit a polymorphism. In a finite population the heterozygosity often tends to zero very quickly due to inbreeding.

Inbreeding is due to the finite population size, in which many relatives mate with each other. These relatives have a chance, depending on how close a relative they are, to have identical alleles by descent from an ancestor. The offspring of these two relatives may end up with two copies of an allele from the same ancestor. Thus, for a finite population, random genetic drift will cause the heterozygosity to decline at a rate of $\frac{1}{2N}$ per generation. Hence, the effect of inbreeding in small populations is that genetic diversity will decrease. Often, inbreeding leads to a less "fit" population due to a loss of diversity, and hence a loss of adaptability.

4 Selection as Source of Adaptability

We have not yet considered the advantage a certain genotype might have over other phenotypes. The effect of selection of genotypes on the genetic structure of a population is a null assumption of the Hardy-Weinberg Theorem. When we get rid of this assumption, we can explain the adaptability of populations to novel changes in the environment.

Selection acts on an arbitrarily defined quantity called fitness. In natural selection, differences in fitness cause a fitter genotype to survive and reproduce more often than a less fit genotype. In artificial selection, the breeder chooses which of his stock to breed together, so as to produce the next generation. The choices the breeder makes define the fitness of the phenotype. Only when the differences in phenotype is based on differences in the genotype, will selection occur. Hereafter, selection is defined as differential survival and reproduction of the genotype.

There a several different modes of selection (See the included figure). Selection can be stabilizing, tending to push a gaussian phenotype distribution to be more sharply peaked distribution around its mean value. Selection can be directional, tending to increase or decrease the mean value of a phenotypic distribution. Lastly, selection can be disruptive, maintaining the mean value, but increasing the deviation from the mean, and often splitting a single population into two different populations.

4.1 Dominance

As an example of the effect of natural selection on the evolution of allele frequency, let us assume we have two alleles in a population that is Hardy-Weinberg except for the differential survival and reproduction of genotypes. We assume that our population has the following genotype frequencies and genotype fitnesses:

Genotype	AA	AB	BB
Frequency	p^2	$2pq$	q^2
Fitness	1	$1 - s_1$	$1 - s_2$

We can calculate the mean fitness of an individual in the population as:

$$\begin{aligned} w_{mean} &= p^2 + 2pq(1 - s_1) + q^2(1 - s2) \\ &= 1 - 2s_1q + 2s_1q^2 - s_2q^2 \end{aligned}$$

We can then calculate the change in allele frequency, q, after one generation:

$$\Delta q = \frac{pq}{2w_{mean}} \times \frac{dw_{mean}}{dq}$$

For the special case when an advantageous allele has dominant expression ($s_1 = 0$), the deleterious allele has recessive expression $s_2 = s$, we can calculate this quantity as:

$$\Delta q = \frac{-spq^2}{1 - sq^2}$$

From this equation, and a scratchpad, you can quite easily see that in order for the rate of genetic change to be great, both alleles must be common in the population. I have included a figure illustrating this concept, which happens to be a general principle.

4.2 Heterozygous advantage

If we do a similar analysis, but this time with heterozygous advantage:

Genotype	AA	AB	BB
Frequency	p^2	$2pq$	q^2
Fitness	$1-s_1$	1	$1-s_2$

we find that that change per generation of q is:

$$\Delta q = \frac{pq(s_1 p - s_2 q)}{1 - s_1 p^2 - s_2 q^2}$$

From this equation, we can find an equilibrium when $\Delta q = 0$: $q_{equil.} = \frac{s_1}{s_1+s_2}$. We can also show that this $q_{equil.}$ maximizes the mean fitness, but I won't do this here. However, the maximization of fitness at the equilibrium frequencies is a general concept, though in the general case, there will be multiple stable equilibria. Since the stable equilibrium allele frequency is nonzero, we have our first stable polymorphism, that has a basin of attraction of non-zero measure.

4.3 Homeostasis

The inquisitive physicist may wonder what the two opposing forces are that maintain this stable, non-trivial equilibrium. The biologist would answer that biselection against homozygotes serves as the two opposing forces. This opposition is called genetic homeostasis. Evidence for genetic homeostasis is the common aforementioned phenomenom of inbreeding depression. Inbreeding depression increases homozygosity, and thus reduces fitness. One reason for the low fitness of homozygotes is their reduced capacity for developmental homeostasis. Developmental homeostasis is "the capacity of an individual's genotype to produce a proper, well-formed, adaptive phenotype in the face of perturbations that can occur during the course of development." (Futuyma) The emergence of a "normal" phenotype is canalized along proper channels and resists diversion into other paths.

Selection is the source of the adaptability of a population to its environment. But selection can only work if there are multiple genotypes on which differential fitness to work. These multiple genotypes are maintained by heterozygous advantage. Yet if a stable polymorphism does not in general exist, there are fleeting polymorphisms caused by spontaneous mutation, which allow for selection to occur in the general situation when there are few heterozygotes.

5 A More Complex Model

5.1 Epistasis

Until now, we have treated single alleles as our basic units of heredity. Under this scheme, the naive physicist might think that the joint effect of many alleles on the phenotype would be the sum of the effects of each allele on the phenotype. But the complexity theorist knows better: the whole is greater than the sum of its parts. It is quite common for alleles in an organism's genome to effect the expression of the other alleles. This is what makes genetics such a challenging discipline, and is known as epistasis, and is due to the pleiotropic effects of a gene on many different characters.

5.2 Linkage Disequilibrium

Often epistasis maintains a phenomenom known as linkage disequilibrium, in which two or more alleles for different traits tend to be associated with each other. For example, the flowers of the primrose Primula vulgaris have two types of styles and two different types of anthers. Yet there is a strong interaction between the anther and style alleles, since there is a strong association of types of anthers and types of styles, producing two common forms of the plant.The "pin" form, with long style and low anthers, is inherited as recessive to the "thrum" form, with short style and long anthers. The homostylous form, with both short or both long is produced very rarely by genetic crossing over during meiosis.

Assume that we have have a diploid parent, with a chromosome consisting of two allele-pairs, A_1B_1 & A_2B_2. During meiosis, or gamete formation, the haploid gametes have an R% chance of being A_1B_2 & A_2B_1, by genetic recombination. The other two possible gametes are A_1B_1 & A_2B_2, which are the same as the parental gametes. When R is close to zero, A & B are tightly linked. On the other hand, when $R = frac12$, A & B are loosely linked. Given the four possible haploid gametes, there are ten possible diploid offspring genotypes. From the recombination frequency, R, and Mendel's laws of inheritance, we can easily deterimine the time dependence of the ten genotype frequencies. However, it is more convenient to keep track of chromosome frequencies, since we only have to track four quantities. The four chromosome frequencies are given as:

$$x_1(t+1) = x_1(t) - R \times D(t)$$
$$x_2(t+1) = x_2(t) + R \times D(t)$$
$$x_3(t+1) = x_3(t) + R \times D(t)$$
$$x_4(t+1) = x_4(t) - R \times D(t)$$

where $D(t) = x_1 x_4 - x_2 x_3$, is the linkage disequilibrium between genes A & B. We can calculate the time evolution of $D(t)$ quite trivially: $D(t+1) = (1-R)D(t)$, so that D converges to zero at a rate of (1-R) per generation. This is called the two-locus Hardy-Weinberg equilibrium.

5.3 Linkage Disequilibrium with Selection

Now if we give a differential fitness to each genotype, the recursion for x_k becomes a little bit more complex. Define w_{ij} as the relative fitness of the i/j genotype, where i, j are labels $\in [1,2,3,4]$, labelling A_1B_1, A_1B_2,A_2B_1 & A_2B_2. We assume that $w_{14} = w_{23}$. Then:

$$x_k(t+1) = (x_k(t)\sum_{j=1}^{4} w_{kj}x_j(t) \pm RD(t)w_{14}) \div w_{mean}$$

where the plus values are for $k \in [2,3]$, and:

$$w_{mean} = \sum_{i=1}^{4}\sum_{j=1}^{4} w_{ij}x_i x_j.$$

Due to the non-zero linkage disequilibrium, the solution to this system of equations is non-trivial. But Feldman discusses the stability of the equilibria of the four-simplex $x_i \geq 0$, $\sum_{i=1}^{4} x_i = 1$. He says that if the stability of a simplectic boundary equilibrium (fixation to a vertex or to a 2-polymorphism on an edge) depends on the recombination frequency, R, then that equilibrium is stable for large R (loose linkage) and unstable for small R (tight linkage). We can define R_{min} as the minimum recombination rate needed for stability. We have found another genetic principle that gives the population stability and yet at the same time, maintains adaptability.

6 The Effects of Adaptation

6.1 Convergent Evolution

An interesting phenomenom seen right here in Tucson, that is due to adaptation, is called convergence. Due to our unusually dry environment, over many millions of years, many desert plants near Tucson, have become succulent, though they represent widely different taxonomic form. This degeneracy is another mark of the complexity of ecosystems and is evidence for the effect of the environment on the genetic structure of populations.

6.2 Evolutionary Stable Stategies

Another effect of adaptation is seen in the study of Maynard Smith's evolutionary stable strategies (ESS). These are strategies which fit into the ecosystem in such away that no other mutant strategy could invade the population under the influence of natural selection. While a strategy is a specification of what an individual will do in any situation in which it may find itself. A strategy may be pure (constant phenotype), or mixed (time-dependent phenotype). Notice that a mixed strategy is not a genetic polymorphism.

Biologists most often use ESS models to analyze animal behavior, for example, the predator-prey relationships of a complex food chain. Maynard Smith shows that, in general, by considering small displacements from equilibrium of a feed-forward system of Volterra's equations, with no lateral connections, that competition between prey species is destabilizing, whereas competition (generalist, rather than specialist predators) between the top predators is stabilizing. Competition at the intermediate level has inconsistent effects. Also, when the connectivity of the ecosystem destabilizes the system, its components oscillate out of phase, with a period longer than their period of isolation.

6.3 Coevolution of Species

Another effect of adaptation is the coevolution of species. Coevolution of two species means that the interaction of the two species tends to change the genetic structure of each species' population. This genetic feedback is one of the most effective non-random interspecific interactions that maintains the stability of the ecosystem. An example is the competition of the housefly Musca domestica and a blowfly Phaenicia sericata.In an experiment in which the two species competed in a large cage, the housefly population initially increased manifold relative to the blowfly. After 27 generations, the blowfly began an explosive increase, and the housefly generally became extinct after about 10 more generations. This experiment supports the theory that the low-frequency species adapted to increase its fitness relative to the high-frequency coevolving species.

6.4 Territorial Behavior

Probably the strongest stabilizing force in any ecosystem is territorial behavior. If the regulation of population numbers is regarded as due to territorial behavior, then natural selection must have acted by "group selection," as proposed by Wynne Edward. Most mathematical biologists have trouble accepting adaptation by group selection, except in cases where species are divided into small reproductively isolated groups. Wade gives a good account of the emergence of altruism (non-cannibalism)in flour beetles by group or kin selection.

7 Conclusion

Several factors affect the stability/adaptability balance in any ecosystem. Spontaneous mutation and selection on the advantage that mutation gives in a perturbed environment helps to maintain a specific polymorphism in an environment. This polymorphism, in turn, allows for developmental homeostasis of a population in its environment. Genetic recombination and selection on the recombinant chromosomes tends also to maintain a diversity, and hence homeostasis, and allows for speciation (genetic isolation). Lastly, the emergence of adaptive behavior, especially interspecific interactive feedback in the ecosystem's web of life, and non-random interaction between species, due to novel strategy-choice, lend great stability to any ecosystem. The complexity of an ecosystem (defined here as the number of species and the connectivity) does not necessarily lead to a balance between stability and adaptability, as very simple reasons can explain the observed persistence of ecosystems, even subject to perturbation. Yet the complexity of most ecosystems lends us many interesting phenomena to study.

8 References

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