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THIRD AUTUMN COURSE ON MATHEMATICAL ECOLOGY

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"Evolutionary Theory: An Introduction"

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EVOLUTIONARY THEORY
AN INTRODUCTION

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Overview

History

Key Issues

Basic population genetics

ESS theory

Assorted topics:

- Multiple alleles
- Diffusion approximations
- Multiple loci
- Frequency dependent selection
- Spatially varying environments

Some History of Evolutionary Theory

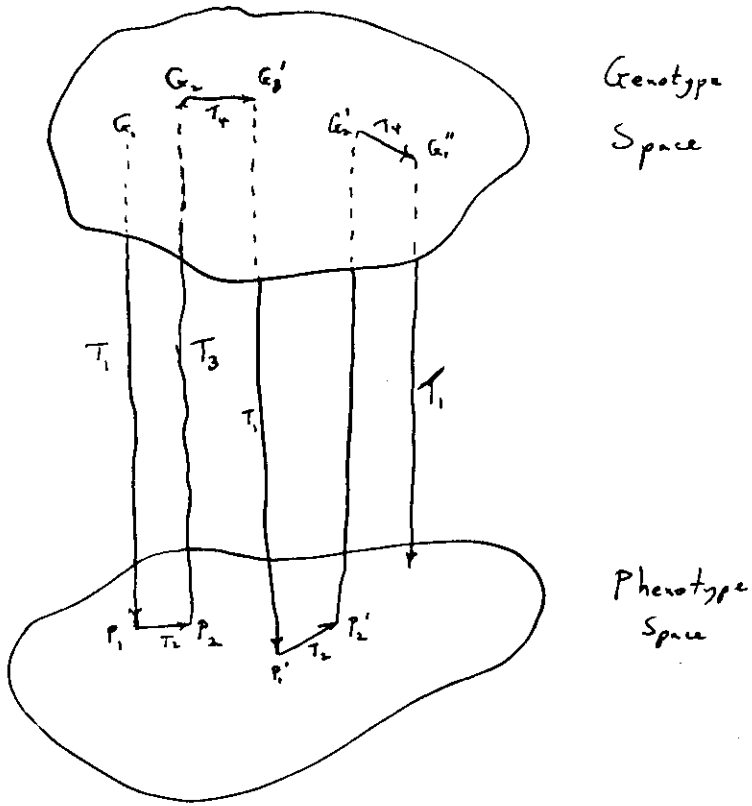
1. Darwin - 2 key points made in the Origin:
   (a) Evolution has happened (existing organisms descended from ancestral forms)
   (b) Main cause of evolutionary change was natural selection of variations which in their origin were non-adaptive.

   Without genetics, assumed acquired characteristics
2. Weissmann - germ line independent of soma, so acquired inheritance cannot occur.
3. Rediscovery of Mendel's laws - chromosome theory of heredity. Debate between Mendelians and biometric school over continuous versus discrete traits.
4. Population genetics (Fisher, Haldane, Wright) Theoretical basis for effects of selection, mutation, drift.
5. Modern synthesis (Dozhansky, Mayr, Simpson, Stebbins) - neo-Darwinism (natural selection on Mendelian pops.) sufficient to explain observations on geographic variation, fossils.

Key Issues in Evolutionary Theory

1. Origin of Life - how did entities with properties of multiplication, heredity and variation arise?
2. Evolution of the genome - how are chromosomes organized, how did recombination arise, is most of the genetic complement neutral, is DNA selfish, evolution of plasmids, viruses.
3. What maintains genetic diversity in populations Why are there so many different types of organisms? Evolutionary ecology - life history theory, pop. structure affecting evolution.
4. Levels of selection - when are kin, group, trait group, sexual selection important factors in genetic change? Evolution of behavior, ESS theory.
5. Cultural transmission and selection - phenogenotype approaches. Epigenetics.
6. Large-scale features of evolution - macroevolution - puncuated equilibria

See Evolution Now (J. Maynard Smith, ed.)
W.H. Freeman, 1982.



Evolution from the point of view of population genetics.

T_1 - epigenetic laws

T_2 - ecological transformation, survival, fecundity

T_3 - reverse epigenetic laws

T_4 - genetic laws - recombination, mating.

Basic Population Genetics

Genotype - total genetic complement of an individual, capable of being passed on to offspring

Phenotype - collection of all characters displayed by an individual, controlled by genetics, environment, development

Population genetics - study of variation of genes within a population through time and space

Evolution - change or potential change in genetic structure of a population

Natural selection - process whereby if individuals of one genotype have on average more offfspring than another, then the first genotype will spread through the population.

Note: population genetics allows us to investigate the mechanisms of the process of evolution, rather than just describing the patterns we observe as the products of evolution (phylogeny).

Basic assumptions of simple models:

1. A gene controlling a character is at one location (locus) on a chromosome or several such locations (multiple loci).
2. At any particular locus the gene may have one of a number of possible forms (alleles)
3. Organism may be haploid(one allele at each locus) or diploid (2 alleles at each locus)

So in diploid case, AA and aa individuals are homozygous at that locus, Aa individuals are heterozygous. If after time passes, only one allele persists at a locus, that allele is "fixed", is more than one allele persists at the locus, the population is "polymorphic" at the locus.

Basic Forces of Population Genetics

1. Natural Selection - changes in gene frequencies in a population due to traits giving differential survival and reproduction among individuals, and these traits are heritable.

2. Mutation - genes change form during the life cycle of an individual in a way that can be passed to offspring. If one allele mutates at a higher rate than another, get change in gene frequency due to "mutation pressure".

3. Random genetic drift - change in genetic structure in a population due to random sampling error in finite populations. Important when populations are small, when new populations are started in isolated areas (founder effects).

4. Other forces:
   - Selection at other levels - group, kin, sexual
   - Gene flow - migration or dispersal of individuals with different genetic structure into a population.
   - Segregation distortion (meiotic drive) - unequal production of gametes in heterozygous individuals - difficult to tell apart from gamete selection.

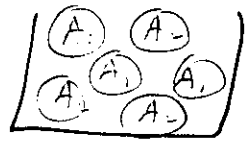
Basic Population Genetics

A: Hardy-Weinberg equilibrium – 1 locus, 2 alleles - diploid

Assume:

1. Discrete non-overlapping generations (no age/size structure)
2. Random mating or random union of gametes
3. ∞ - population size (no sampling phenomena)
4. No selection, no mutation, no gene flow
5. No sex differences

Then view a pot of gametes from which next generation is drawn.



Let p = initial frequency of A_1
$1-p = q$ = " " " A_2
} gamete frequencies

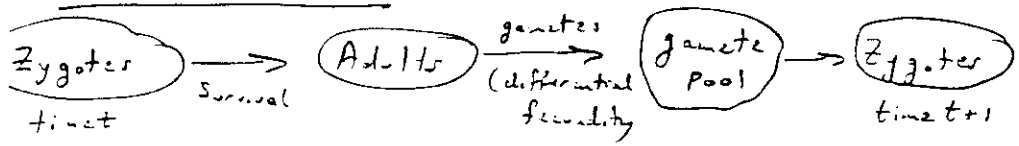
Then in 1st generation, the genotype frequencies are

$A_1A_1 : A_1A_2 : A_2A_2$

$p^2 : 2p(1-p) : (1-p)^2$ H-W equilibrium

Shows, unlike in blending inheritance in which variance in population declines, under simplest assumptions of Mendelian genetics, variation is maintained.

Selection Model — A_1, A_2 alleles



x_t = frequency of A_1 at time t (in zygotes)

Assume zygotes at time t in H-W equilibrium

N_t = # zygotes at time t

l_{ij} = $P[$ survive from zygote to adult if A_iA_j $]$

$2m_{ij}$ = mean # gametes produced by A_iA_j individ.

	A_1A_1	A_1A_2	A_2A_2
adults	$x_t^2 l_{11} N_t$	$2x_t(1-x_t) l_{12} N_t$	$(1-x_t)^2 l_{22} N_t$
gametes produced	$2l_{11}m_{11} x_t^2 N_t$	$4l_{12}m_{12} x_t(1-x_t) N_t$	$2l_{22}m_{22}(1-x_t)^2 N_t$

$\Rightarrow$ frq. of A_1 in gamete pool (and thus in zygotes at $t+1$) is

$$x_{t+1} = \frac{\left(2l_{11}m_{11}x_t^2 + 2l_{12}m_{12}x_t(1-x_t)\right)N_t}{\left[l_{11}m_{11}x_t^2 + 2l_{12}m_{12}x_t(1-x_t) + l_{22}m_{22}(1-x_t)^2\right]2N_t}$$

$$= \frac{W_{11}x_t^2 + W_{12}x_t(1-x_t)}{W_{11}x_t^2 + 2W_{12}x_t(1-x_t) + W_{22}(1-x_t)^2} = W(x_t)$$

and

$$N_{t+1} = W(x_t)\,N_t$$

$W(x_t)$ = mean population fitness

W_{ij} = absolute fitness of A_iA_j

If W_{ij} depends on N_t, get density dependent selection. If W_{ij} depends on x_t, get frequency dependent selection.

If W_{ij}'s constant, above is equivalent to dynamical system

$$x' = F(x) = \frac{W_{11}x + W_{12}x(1-x)}{W(x)}$$

then can show

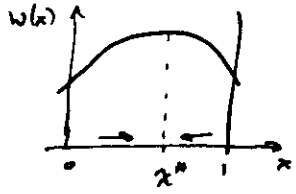
$$\Delta x = x' - x = \frac{x(1-x)}{2W(x)}\frac{d}{dx}(W(x))$$

so fixed points are $x = 0, 1$ and critical points of $W(x)$ in interior of $(0,1)$

Case 1: $W_{12} = \frac{W_{11}+W_{22}}{2}$ Heterozygote complete intermediate

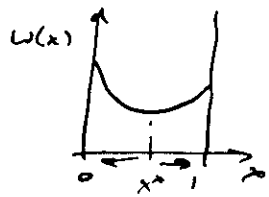
then $W(x)$ is linear, get $x \to 0$ if $W_{11} < W_{22}$ & $x \to 1$ if $W_{11} > W_{22}$

Case 2: $W_{12} \neq \frac{W_{11}+W_{22}}{2}$ - $W(x)$ is parabola



Heterozygote superior

x^* stable equil.



Heterozygote inferior

x^* unstable

initial conditions control final state

C: Mutation & Selection Mutation $A_1 \underset{v}{\overset{u}{\rightleftharpoons}} A_2$

With no selection

$$x' = x(1-u) + v(1-x) = v + x(1-u-v) = G(x)$$

So at equil, $x = x' = \frac{v}{u+v}$

If assume selection operates betwn zygote & adult stage & mutations occur just during meiosis, then with selection,

$$x' = G(F(x)) = v + (1-u-v)F(x)$$

Defining $\phi(x) = x^{2v}(1-x)^{2u}\left(W(x)\right)^{1-u-v}$,

then can show $x' - x = \frac{x(1-x)}{2\phi(x)} \frac{d}{dx}\left(\phi(x)\right)$

so equilibria are 0, 1, critical points of $\phi(x)$ in $(0,1)$

In absence of mutation, $\phi(x) = W(x)$ as in selection case

In absence of selection, $\phi(x) = x^{2v}(1-x)^{2u}$ which is maximized at the equil $x^* = \frac{v}{u+v}$

In general, there is a trade-off between the two maximum principles — called "mutation/selection balance". Fisher's Fundamental Thm. of Nat. Selection relates increase in fitness of pop. under selection to amount of additive genetic variation in the pop.

Game Theory & Evolutionarily Stable Strategies

Applied when costs + benefits of a behavior depend upon behavior of other population members.

Specify: (a) Set of behavioral responses an organism can have: e.g. display, attack, escalate, etc.

(b) Set of possible strategies - rule specifying what behavior will occur in any particular situation

Pure: eg. always attack under circumstance A

Mixed: e.g. attack w.p. .5 & display w.p. .5 under A

(c) Payoffs

$E[A, B]$ = expected change of fitness for organism adopting strategy A against opponent which adopts B.

Typical assumptions:

(i) ∞ - population size - ie. ignore genetic relatedness between individuals

(ii) random mixing - each encounter with an individual is chosen at random from all individuals adopting B

(iii) asexual reproduction - strategies represented in successive generations with frequencies proportional to their overall payoff in the previous generation.

Def: A population is in an <u>evolutionarily stable state</u> if its genetic composition is restored by selection after a small enough disturbance (note: pop. could be monomorphic or polymorphic here)

Def: An <u>evolutionarily stable strategy</u> (ESS) is a strategy such that, if all individuals adopt it, no mutant strategy can successfully invade.

In pairwise contests, I is an ESS if for any other strategy J either $E(I,I) > E(J,I)$

or

$E(I,I) = E(J,I)$ and $E(I,J) > E(J,J)$

Often, simple cases are expressed as matrix game

Ex: Parental care – parent either guards or deserts offspring

$P_0, P_1, P_2 = P[\text{egg survives} \mid \text{cared for by } 0,1,2 \text{ parents}]$

$p = P[\text{male mates with } 2^{nd} \text{ ♀} \mid \text{male deserts } 1^{st} \text{ ♀}]$

$p' = P[\text{" " " " " " } \mid \text{male guards } 1^{st} \text{ ♀}]$

Assume $p \geq p'$

$V = \#\text{eggs laid by ♀ if she deserts}$

$v = \#\text{eggs " " " " guards}$

Assume $V \geq v$

Assume male & female mates only during one year.

Assume $P[$male is father of offspring of female with which he mates$]$ is independent of deserting or guarding

Assume at most 2 matings:

♀

♂ \ ♀	G	D
G	vP_2 ♀ payoff; $vP_2(1+p')$ ♂ payoff	VP_1; $VP_1(1+p')$
D	vP_1; $vP_1(1+p)$	VP_0; $VP_0(1+p)$

Get 4 possible ESS's

#1 Both G $\quad vP_2 > VP_1$ & $P_2(1+p') > P_1(1+p)$

#2 ♂ G, ♀ D $\quad VP_1 > vP_2$ & $P_1(1+p') > P_0(1+p)$

#3 ♂ D, ♀ G $\quad vP_1 > VP_0$ & $P_1(1+p) > P_2(1+p')$

#4 Both D $\quad VP_0 > vP_1$ & $P_0(1+p) > P_1(1+p')$

So if $P_2 \gg P_1$ & $p \approx p'$, #1 is ESS

if $P_0 \approx P_1$, then #4 is ESS

Can get cases in which both #2 & #3 are ESS – & history matters.

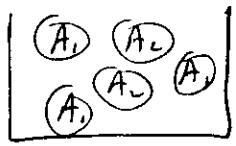
Problems with ESS Theory

1. No population genetics – purely phenotypic approach – particularly difficult when point of interest concerns genetic variability in the pop. (e.g. evolution of sex ratio)
2. Misleading to think of animals as optimizers – but standard pop. genetics models lead to maximization principles
3. There are developmental constraints, so optimum strategies may not be reachable – need more emphasis on construction of phenotype set.
4. Essentially equilibrium approach – ignores dynamics. Even if an ESS exists, due to nonlinearities pop. may not get to it.
5. Usually assume conflict not affected by information gained during the conflict – need to consider suite of behaviors through time à la dynamical state-space approach.

Random Drift

Consider finite pop. size – let pop. size = N (constant)

Diploid 2-allele case, have $2N$ total alleles in pop. Assume



have large pool of gametes, but only select $2N$ to form next generation

Question: Does freq. of alleles change with time?

Let X_t = # A_1 alleles in generation t (values $0, 1, 2, \ldots, 2N$)

Assume random sampling, then with $x = \frac{X_t}{2N}$ = freq. of A_1, X_{t+1} is binomially distributed with parameter x.

Construct a Markov Chain

$$p_{ij} = P[X_{t+1} = j \mid X_t = i] = \binom{2N}{j} \left(\frac{i}{2N}\right)^j \left(1 - \frac{i}{2N}\right)^{2N-j}$$

$i, j = 0, 1, \ldots, 2N$

$P = (p_{ij})$ is transition matrix, $P \geq 0$, with

$f_i(t) = P[X_t = i]$ then knowing P & $\tilde{f}(0)$

$\tilde{f}(t+1) = \tilde{f}(t)\, P$ so $\tilde{f}(t) = \tilde{f}(0)\, P^t$

Have 2 "absorbing states" $X = 0, 1$ & if

$\pi_i = P[X(t) = 2N$ eventually for some $t \mid X_0 = i]$

then $\tilde{\pi} = \tilde{P}\,\tilde{\pi}$ gives absorption probabilities

Note: Any trajectory of process (i.e. any population) must eventually become fixed at 0 or 1.

Letting $Y_n = \frac{X_n}{2N}$ = gene freq. of A_1 & suppose $p_0 = \frac{X_0}{2N}$,

then $E[Y_n] = p_0$ and $Var(Y_n) = p(1-p)\left\{1-(1-\frac{1}{2N})^n\right\}$

$\to p(1-p)$ as $n \to \infty$

So drift acts to increase inter-population variance while decreasing within population variance.

Note: Since $\tilde{f}(t) = \hat{f}(0)\, P^t = \sum_{i=0}^{2N} c_i \lambda_i^t \vec{v}_i$ where

λ_i's are eigenvalues with $\vec{v}_i$ eigenvectors, can show 1 is a double eigenvalue of $\tilde{P}$ ($\overset{\text{fixation}}{\text{eigenvectors}}$ $\begin{pmatrix}1\\0\\\vdots\\0\end{pmatrix}, \begin{pmatrix}0\\\vdots\\0\\1\end{pmatrix}$)

& next largest eigenvalue is $1-\frac{1}{2N}$, which controls time to fixation.

Above is Wright-Fisher Model, there are lots of extensions, i.e. with selection, but generally do not use Markov Chain, but associated diffusion approximations.

Other topics

Diffusion Approximations Approximate solution paths generated by Markov Chain models by a continuous-state space Diffusion Process (Markov Process with continuous paths) in order to derive properties of solutions, e.g. asymptotic behavior, in situations when Markov Chain models are difficult to obtain analytic results. Derive Forward & Backward Equations:

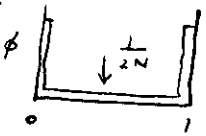
Wright-Fisher – $\frac{\partial \phi}{\partial t} = \frac{1}{4N} \frac{\partial^2}{\partial x^2}\left(x(1-x)\phi\right)$, $\phi(x,0) = \delta(x-p)$

no selection, no mutation

ϕ is density function for unfixed gene frequencies –

can show $\phi \approx 6p(1-p)\, e^{-t/2N}$ for t large

i.e. gene distribution of unfixed classes is uniform



Can analyze lots of different cases – mutation, selection, fluctuating selection, migration, etc.

(See J. Gillespie in M. Feldman (ed) Mathematical Evolutionary Theory, Princeton, 1989)

Multiple Alleles Similar to 2 allele case – there is Fundamental Thm that says mean pop. fitness increases under selection, and at equilibrium fitness is maximized. There are results which state when a full polymorphic equilibrium (i.e. with all alleles present) is stable vs. case when go to boundary equilibria.

Multiple Loci

Lots of open problems, but 2-loci theory well studied. Must deal with linkage & recombination. Some questions:

(a) If 2 loci control very different traits, can one use single locus theory & treat these loci independently?
No, due to linkage - usually.

(b) If 2 loci control some trait, can selection act to aggregate together alleles from different loci which function well together? Yes, sometimes

(c) Does Fundamental Thm carry over to multiple loci?
Not generally - many cases in which mean fitness can decline under selection.

Key question is whether level of linkage disequilibrium in natural pops. is low (then 1-locus theory may well be more or less OK to describe genetic dynamics), or not (then single locus results are misleading).

Frequency Dependent Selection - Fitnesses of genotypes depend on allele or gamete frequencies in the pop. One conclusion of work is that a stable polymorphism is possible without heterozygote advantage - can be used to explain large # of observed electrophoretic polymorphisms.