BI 2113 - Ecology and Evolution

9/9/20

ECOLOGY AND EVOLUTION

BI 2113 - Semester 3

Wildebeest migration

→ To understand the mechanism - one has to look inside.

Wildebeest muscle efficiency - 62%

Reproduction mechanism - herd comes together and mate and give birth together. (Jan - March)

400k outta 2 million give birth to 80% of calves in 2-3 weeks

→ Why do they migrate?

They migrate through Serengheti plains (area = 500k km²)

They migrate following the places where they can get food which depends on the rains the plains get — monsoon makes the southern region greener — so they take a 650 km circuit. (They track the rain from 80-100 kms)

Migration gives the population two advantages —

1. Additional food — which allows them to survive & reproduce
2. Avoiding predators - predators can't follow them beyond their territories. Only 25% are killed by predators while Residents are affected by it by 75%.

Migrators : 1.3 million.

Resident population : ~ 5000

→ How they impact the ecosystem

1. Their death : 0.7% die by drowning and their death feeds a lot of scavengers and returns nutrients to the atmosphere. Feeds birds, fish, crocs and allow microbes to grow on their bones.
2. Tree cover : Earlier, forest fires would burn down young trees and tree cover was rapidly declining. But as wildebeest eat a lot of grass and hence reduced dry grass which would feed the fire. Hence, increase in wildebeest population reduced forest fires which increased tree cover. This increase in tree cover has led to increase in populations of birds, elephants, herbivores and other species.

Discussion

1. What and how exactly do they follow resources?
2. How do calves recognise their mothers?: Imprinting
3. Site fidelity: No

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Lecture 02

- Structural Organization of Life: From biomolecules to biosphere
- If were studying with an individual in focus, the population, ecosystem and biosphere becomes its environment.
- An individual affects the environment and is affected by it in turn

Temporal Organization

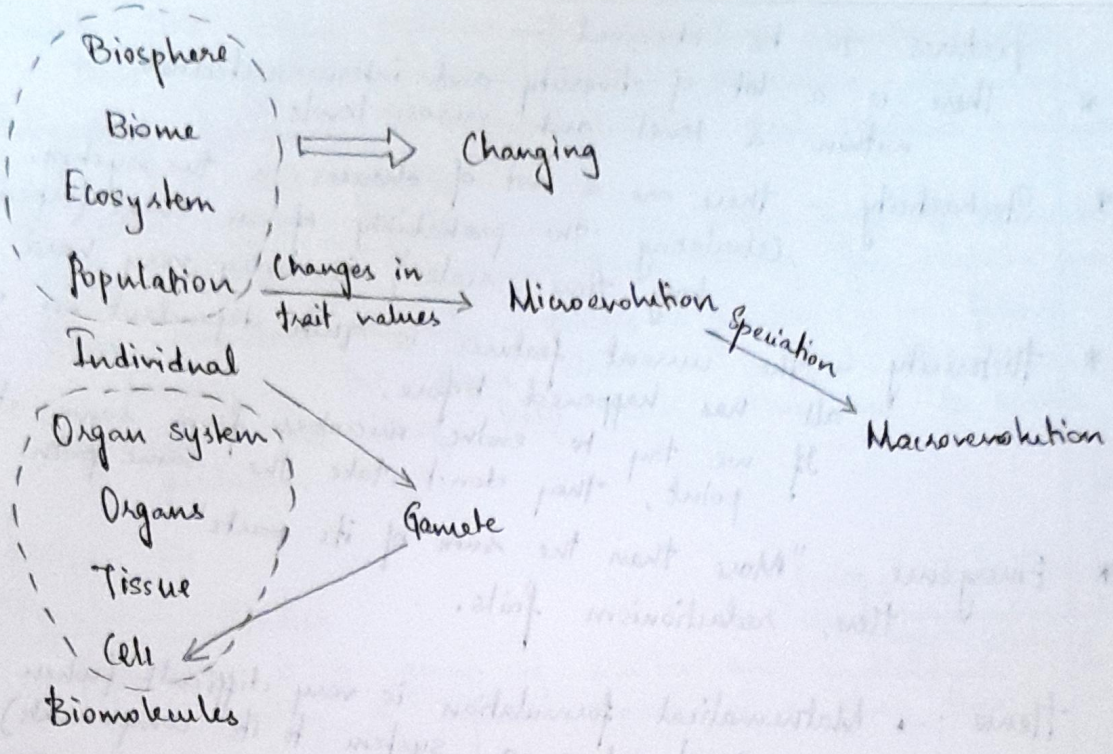
* Short timescale – generational timescale
An individual produces gametes which through fertilisation produce a zygote (cell) which then develops (tissue – organ – organ system) into an individual.
This can be affected by the environment.

* Longer timescale – several generations' time
There are certain trait frequencies in a population. Change in trait frequency (over several generations) is called microevolution. Continued it causes speciation (through natural selection etc) and Macroevolution. This happens ~~every~~ over very long timescale.

All this time, the environment is changing continuously. So, experiments/studies are instantaneous as its impossible to reproduce the same system exactly.

In the structural organisation, the lower level can easily affect the upper levels because in biology, small changes ~~tendencies~~ have a tendency to cascade.

When thinking of a problem, think about it in context of the whole picture.



Discussion

* Can change in theory / knowledge at a smaller scale be affect theories at large scale ? - Not always.
* Changes in higher level affecting lower level : Eg: Forest fires
* Is there evolution of ecosystem — one being chosen over another because it has stabilising factors ?

Could succession be called evolution ? If so, where is natural selection coming from.

Stability of evolution — its resilience.

* Macroevolution v/s saltation
* What's the definition of a species ?
  - Morphology (Based on features - Darwin)
  - Fertile offspring
  - Genetic Basis ?
* What is the fundamental unit that natural selection operates on ? Individuals or a group ?

Features to be observed —

* There is a lot of diversity and interconnectedness — within a level and across levels.
* Stochasticity — there are a lot of chances in the system. Calculating the probability of an event (especially over long time scales) is very very hard.
* Historicity — the current features is quite dependant on what all has happened before. If we try to evolve microbes from same starting point, they don't take the same path.
* Emergence — "More than the sum of its parts" Here, reductionism fails.

Hence :→ Mathematical formulation is very difficult (when y. can't reduce a system to its components).
→ The experimental variables are very variable and hence its difficult to get similar results.
Hence its relation with reality are statistical & conditional

Due to these limitations, we have two approaches —

1. Figure out what is there — descriptive
2. Try to build simple models to explain concepts and processes

Discussion

* Are controls / substitutes used in studying ecological systems — depends on the question we're asking (eg. Predator-prey)
* The nature of trajectories (steady increase, sigmoid, bell curve etc) are the qualitative features studied
* What changes in the nature of study when we move from one level to another (molecular to cellular)?

Ans: No. of interlinks & interactions increases exponentially.

* In 1960s, they found a cure for a virus that infected wildebeests and cows. From this point their population exploded. They've existed in the plains in a few lakhs since 2.5 mya & they've been migrating for a long time.

(05) * While the virus explains the population growth recently, their migration pattern explains population sustainence.

Migratory behaviour is genetical. Note that learned behaviour is not genetic.

Its seen that proteomics is a better way of accounting for migratory behaviour than looking for certain genes.

* Other migratory animals - zebras & gazelles (?) - follow roughly the same circuit. In fact, there are hypotheses that they help each other out to evade predators.

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Ecology - Interactions among organisms and their biophysical environment (i.e biotic & abiotic)
And how this leads to their abundance & distribution patterns

Book on wildebeests - J Estes? Richard
" Newsworld"

Lecture 04

Population Studies

Ecology - Ernst Haeckel
"Oikos" : house/environment
"logia" : study of

Population is a group of similar ~~one~~ individuals, inhabiting a defined space at a given time.

Why - conservation of endangered species
Management of economically important
Management of pests
Fundamental unit of evolution

What is studied - Distribution (over space)
- AGE DISTRIBUTION - Demographics (age-structure, survivorship, birth rate)
Dynamics (how their numbers change over time & space)

Population dynamics studies how and why the size of population changes.

Example : Phlox drummondii - a herb - lives about a year.

The group of individuals that are chosen to study at a given time (without considering offsprings produced) are called a cohort.

✓ born at same time

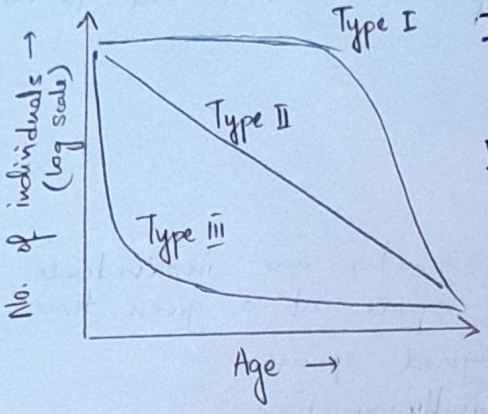
Refer the Cohort Life-Table
- No. of surviving individuals and proportion of surviving individuals (l_x) decreases monotonically over time of 362 days.
- Upto 299 days, all 996 individuals survive
- Then, avg. no. of seeds set per individual during that interval - m_x - is also tabulated. This can be in any pattern.
- Avg no. of offsprings produced by an individual is - Net per capita Reproductive rate : $R_0 = \Sigma l_x m_x$

- If $R_0 = 1$: Each individual is replacing itself
  $R_0 < 1$: Population will decline over time
  $R_0 > 1$: Expanding population.

Survivorship Curves.

It shows the no./proportion of individuals surviving to a certain age in each species.

Its a plot of "No. of Individuals in log scale" vs "Age"

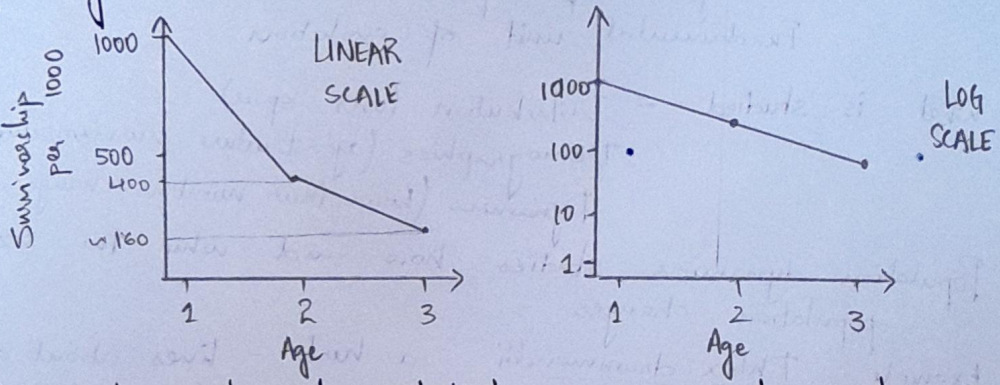


I : Mortality rates are low until late in life
Eg: P. drummondii, humans

II : Const. proportion of individuals throughout the life cycle
Eg. (?) deers, birds, common mud turtle

III : Juvenile mortality is very high
Eg: fish, turtle, insects.
desert plant
- Cleome

Note : log scale vs. linear scale



* If numbers to be plotted are very large, then linear scale won't be useful or convenient.
* In above case, 60% of individuals are dying between 1 & 2 and 2 and 3. In linear scale, it appears as if mortality rate is higher in the first year than second. Using a log scale allows us to see that a constant proportion of individuals are dying.

These survivorship curves aren't exactly followed by natural populations — we observe approximations and mixtures.

Methods to estimate patterns of survival —
1. Cohort life table
2. Static life table
3. Age distribution. → Assumes population is stable & there's no migration

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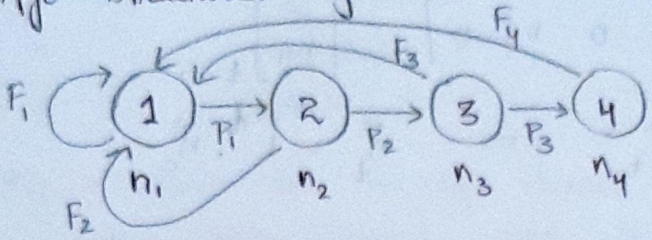
Discussion

- Why is population the fundamental unit – the organism dies but what persists is the population.
  Why not gene? – Genes have no phenotype and selection only happens on phenotypes
- "Setting seeds" – No. of seeds produced.
- In bacteria and yeast it's difficult to track cohorts and/or individuals
- Cohorts are not considered when studying interspecies interaction

Lecture 05 17/7

If we only consider a cohort, the population will only decrease. So, let's include offsprings

Age structured dynamics



n: No. of individuals in each age class
P: Fraction of survivors
F: Reproducibility

$$n_2(t+1) = n_1(t) \cdot P_1 \quad \text{– Similar for } n_3, n_4$$

$$n_1(t+1) = F_1 \cdot n_1(t) + F_2 n_2(t) + F_3 n_3(t) + F_4 n_4(t)$$

$$\begin{bmatrix} n_1(t+1) \\ n_2(t+1) \\ n_3(t+1) \\ n_4(t+1) \end{bmatrix} = \begin{bmatrix} F_1 & F_2 & F_3 & F_4 \\ P_1 & 0 & 0 & 0 \\ 0 & P_2 & 0 & 0 \\ 0 & 0 & P_3 & 0 \end{bmatrix} \times \begin{bmatrix} n_1(t) \\ n_2(t) \\ n_3(t) \\ n_4(t) \end{bmatrix}$$

Task –

Age class	P_x	F_x
1	0.5	0
2	0.2	2
3	0.1	1
4	0	1

Starts with 200 individuals and evolve it 200 time steps

Plot against time –

i) Total population
ii) n_{x+1}/n_x where $x = 1, 2, 3$
iii) N_{t+1}/N_t

Discussion

- In most real cases, $F_i = 0$.
- Using discrete time model here simplifies things. Having F as a function of age and integrating it over time would be a continuous time model – too complicated to use here.
- For sexual reproduction, while modelling it, F is usually just considered for females (assuming they're getting enough matings). It's not considered per capita.

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Lecture 06

Solution to the task –

$$\begin{bmatrix} n_1 \\ n_2 \\ n_3 \\ n_4 \end{bmatrix}_{t+1} = \begin{bmatrix} 0 & 2 & 1 & 1 \\ 0.5 & 0 & 0 & 0 \\ 0 & 0.2 & 0 & 0 \\ 0 & 0 & 0.1 & 0 \end{bmatrix} \times \begin{bmatrix} n_1 \\ n_2 \\ n_3 \\ n_4 \end{bmatrix}_t$$

$$\Rightarrow \vec{n}_{t+1} = \vec{L} \cdot \vec{n}_t \quad \Rightarrow \quad \vec{n}_t = \vec{L}^t \cdot \vec{n}_0$$

* Time series of total population – Initially there are some oscillations, but then it increases exponentially
* Ratio $\frac{n_{x+1}}{n_x}$ – there are some oscillations initially then it dampens and becomes constant

The Stable Age Distribution (SAD) becomes constant. Given certain P_i and F_i, SAD converges to a certain value.

* $\frac{N_{t+1}}{N_t} = \lambda$ – also becomes constant.

This behaviour shown in the example is true for any biologically realistic Leslie matrix – i.e $P_i \in [0, 1]$ and F_i is positive.

Mathematically – λ is the dominant eigenvalue & SAD is the corresponding eigenvector.

(09)

Applications –

Endangered turtle : Loggerhead turtle - Caretta caretta.

Turtles lay eggs away from the waterline – the hatchlings have to walk a long way to the shore to reach the sea.

There are watch-groups which protect the nesting sites by putting a mesh around site and helping them reach the sea.

To see if this is effective, scientists studied a cohort of 100 eggs and followed their life cycle.

Stage	Description	Matrix				
1	Egg/Hatchling	P_1	F_2	F_3	F_4	F_5
2	Small juvenile	G_1	P_2	0	0	0
3	Large juvenile	0	G_2	P_3	0	0
4	Subadult	0	0	G_3	P_4	0
5	Adult	0	0	0	G_4	P_5

Here, there are no age classes but rather life stages – some may survive and remain in the same life stage – the probability is given by P_i.

G_i – probability that it moves to next stage class

P_i – probability that it remains in same stage class

→ So given this distribution of survivorship, what is the stage on which conservation efforts should be directed?

Analysis of sensitivity of r (intrinsic rate of increase) to 10%, 50% or 90% improvement in mortality rates in various stages.

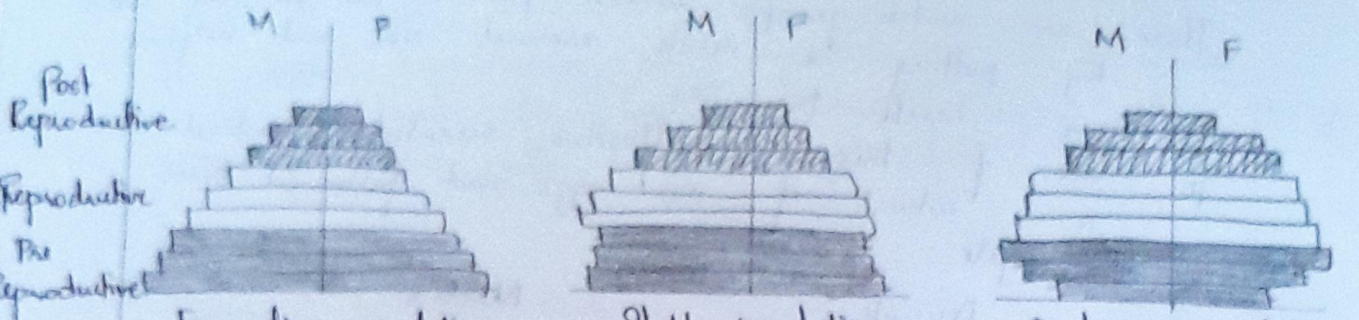
It was found that only intervention in juvenile-adult mortality rates will significantly increase populations.

→ How to protect adult ~~travelers~~ turtles?

Fish & shrimp trawling harms them – they get 5.7 kg of by-catch for every 1 kg of desirable catch. This physically traps and 'tramples' and 'squishes' them.

All fishermen are required to use turtle-excluders in the fishing net, but unless used widely, it won't be very effective. (10)

Population pyramids



Expanding population — India, Nigeria

Stable population — US, Canada

Declining population — Japan

In developing countries – high reproduction rate, high mortality
developed countries – lesser reproduct^{n} rates, lesser mortality
well-developed countries – least reproduct^{n} rates, least mortality.

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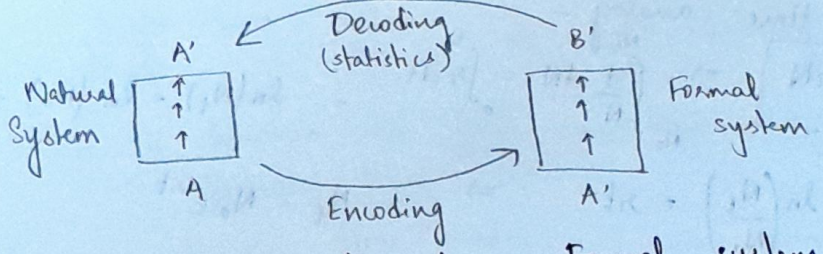
Lecture 07

Desert Locusts – eggs hatch when conditions are viable. Once they've consumed whatever is there, they take wings and fly to find more food – each consumes its weight in food.
They move with wind – least energy consuming. So they move to regions where wind meets rain and there's more vegetation.

First mention of locusts – ox bone 3500 BC in China.

Modelling and Modeling Relationship.

To figure out how natural system is working, we take our best guess and construct a formal system (whose features we know) by encoding or modelling, which can be in form of – mathematical equations
– verbal reasoning.



Then the results from Formal system are compared to the Natural system (primarily by statistics) and the model that is closest to N.S. is accepted.

Robert Rosen – "What is life?"

Based on what we want out of the model, the way we encode will differ –

i) Mechanistic model – to understand the processes of N.S.
Eg: Holdo et. al. – Wildebeast migration model

ii) Phenomenological model – we want the results to agree as closely as possible. So, the encoding will involve feeding in a lot of data to establish some statistical relations. Eg: AI and Machine Learning. Finance models – regression models
Here, prediction is more important.

First modelling was done by Leonardo Bonacci (Fibonacci). He came up with the series to try explain how rat populations would grow.

First Law of Population Dynamics.

$$N_{t+1} = N_t + N_t(b - d + i - e)$$
$$= N_t + N_t \cdot \lambda' = N_t(1 + \lambda')$$

$$\therefore \boxed{N_{t+1} = \lambda N_t}$$

Assuming these rates remain constant –
b - birth
d - death
i - immigration
e - emmigration.

This is a null law.
– its results are more important than the law itself.
Subject develops by studying the departure from assumptions.

$$\Rightarrow N_1 = \lambda N_0 \qquad N_2 = \lambda N_1 = \lambda^2 N_0$$

$$\therefore N_t = \lambda^t N_0$$

Geometric growth $\lambda = \frac{N_{t+1}}{N_t}$

non-overlapping generations

This is the discrete time model.

This law tells us what happens to a population when there are no external factors – it increases/decreases exponentially unless $\lambda = 1$.

Overlapping generations

Continuous time analog –

$$\frac{dN}{dt} = rN \Rightarrow \int_{N_0}^{N_t} \frac{1}{N}\,dN = \int_0^t r\,dt \Rightarrow \ln(N_t) - \ln(N_0) = r(t-0)$$

$$\Rightarrow \ln\left(\frac{N_t}{N_0}\right) = rt \Rightarrow \therefore N_t = N_0 e^{rt}$$

Comparing to the discrete time model,

$$\lambda^t = e^{rt} \Rightarrow \boxed{\lambda = e^r}$$

i.e rate of discrete growth is equal to the exponential of rate of continuous growth

With $\lambda = 1.3$, $N_0 = 10$, the population crosses 25,000 in just 30 generations. But this isn't realistic. i.e. complete exponential growth doesn't happen in nature

③ When variation is added, population will most likely grow acc. to geometric mean and not arithmetic mean

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Lecture 08

As population ~~is~~ increases, the resources available per capita decreases and it can't maintain its growth rate. This is called Density Dependence

In a density independent model – $\frac{1}{N}\frac{dN}{dt} = r$

Intrinsic Growth Rate (r_{max}) → Maximum per capita rate of increase achieved by a species under ideal environmental conditions where b, d, i, e const.

It's the GR when there are no constraints on it – i.e. its the absolute maximum it can reach, when there's no competition.

So its attained when population is 0 – theoretically.

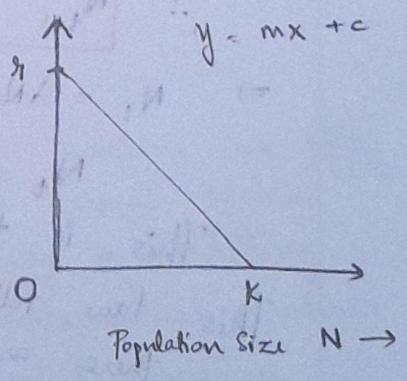
Realized Growth rate $r = r_{max}\left(1 - \frac{N}{K}\right)$

When population increases, GR goes down and stabilises (based on some factors) – this is realised growth rate

The population size (K) at which realised GR is zero is K.

For the graph –

$$\frac{1}{N}\frac{dN}{dt} = r - \frac{r}{K}N$$



Conditions for exp. growth –
- favorable environment
- low population density

Never ending → complete exponential growth

Higher for smaller populations

Probability that a population will go extinct at time t –

$$\rightarrow p_{0,t} = \left(\frac{d}{b}\right)^{N_0}$$ (power) — death, birth

② Geo ≤ Ar

Arithmetic mean = $\frac{\sum_{i=1}^{n} \lambda_i p_i}{\sum_{i=1}^{n} p_i}$

Geometric mean = $\prod_{i=1}^{n} \lambda_i^{p_i}$

① →

$$\frac{1}{N}\frac{dN}{dt} = r_1 - \frac{r_1 N}{K} \Rightarrow \frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right)$$

This is known as Continuous logistic Eqⁿ or Verhulst-Pearl Eqⁿ.

→ For a discrete time model, Y-axis becomes —

per capita growth rate $\frac{N_{t+1} - N_t}{N_t} = r\left(1 - \frac{N_t}{K}\right)$

$$N_{t+1} = N_t + rN_t\left(1 - \frac{N_t}{K}\right) = N_t\left[1 + r\left(1 - \frac{N_t}{K}\right)\right]$$

This is the discrete logistic Equation

→ Considering the population growth rate, $\lambda = \frac{N_{t+1}}{N_t}$

$$\Rightarrow \frac{N_{t+1}}{N_t} = r\left(1 - \frac{N_t}{K}\right) \Rightarrow N_{t+1} = rN_t\left(1 - \frac{N_t}{K}\right)$$

This is also called the Discrete logistic Eqⁿ.

But here, if $N_t = K$, $N_{t+1} = 0$ ⇒ No individuals in next generation

So this form of equation is not useful in biology.

In physics — (while acc to previous eqⁿ, $\frac{dN}{dt} = 0$ not N_{t+1} ! taking $\frac{N_i}{K} = x_i$)

$$\frac{N_{t+1}}{K} = r\frac{N_t}{K}\left(1 - \frac{N_t}{K}\right)$$

$$x_{t+1} = r x_t (1 - x_t)$$ — Also called Discrete logistic functio

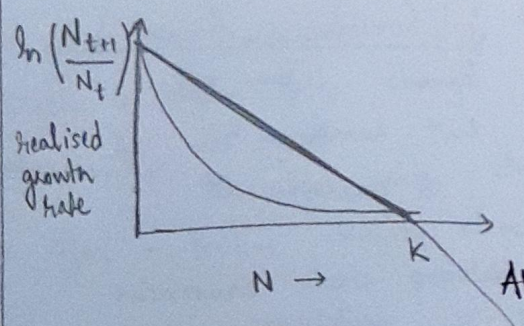
→ Other formulations (λ not $\frac{dN}{dt}$)

Empirically, the growth rate falls exponentially. But if we take logarithmic scale of Y axis, then we'd get a linear curve.

$$\ln\left(\frac{N_{t+1}}{N_t}\right) = r\left(1 - \frac{N_t}{K}\right)$$

$$\Rightarrow \boxed{N_{t+1} = N_t e^{r\left(1 - \frac{N_t}{K}\right)}}$$

This is called the Exponential Logistic equation.



Also called the Rickes model and Discrete logistic Model

K : Carying Capacity
Its the largest no. of individuals that can be sustained by the environment.

Lagged logistic model
When there's a lag between change in growth rate and population size. (In continuous model, there's no lag. The effect is instantaneous).

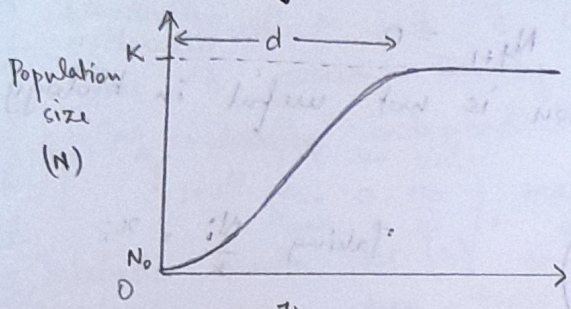
So, equation becomes -

$$\frac{dN}{dt}(t) = r\,N(t)\left(1 - \frac{N(t-\tau)}{K}\right)$$ where τ: lag

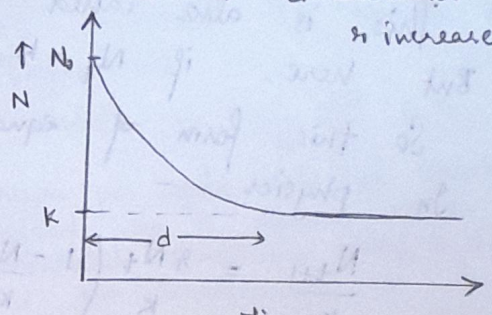
When we introduce lag into the system, we establish cycles - the population won't converge directly to a stable point (typically, not always).

Continous logistic Model - $\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right)$

d decreases as r increases.

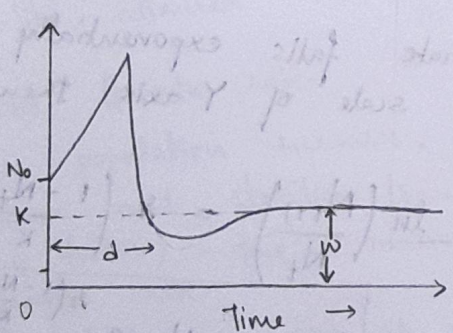


$[N_0 < K]$



$[N_0 \gg K]$

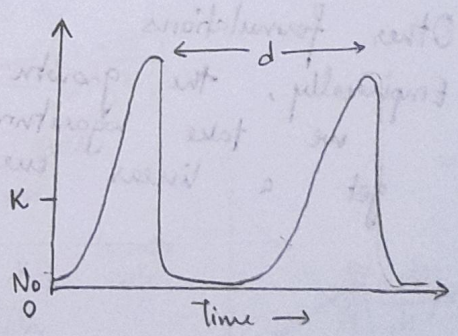
Lagged Logistic Model.



$[N_0 > K]$

w & d decrease as r increases

$w \to 0$ · (why?)



$N_0 < K$

d increases as r increases.

Ultimately environment limits growth of population by modifying birth and death rates.

Ratio $\frac{N}{K}$ - "Environmental resistance"

Chapter 2 - Intro to Population Ecology

K : Carrying capacity - In a given environment, its the no. of individuals that can be maintained indefinitely

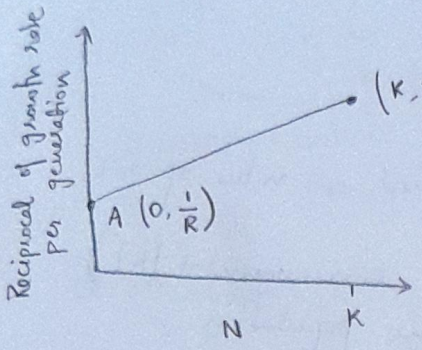
Law of the Minimum, Liebiz (1855) - population size is constrained by whatever resource is in shortest supply

Competition - biological interaction b/w 2 or more individuals for a resource in short supply.

It is a reciprocally negative interaction - decreases fitness in both 2 forms - interference & depletion.

Intraspecific competition manifests through density dependent modification in -

i) birth & death rate
ii) Growth rate
iii) Adult size (in organisms with determinant life cycle)



$$\frac{N_t}{N_{t+1}} = \frac{1}{R} + \left[\frac{1 - 1/R}{K}\right] N_t$$

$$N_{t+1} = \frac{N_t R}{1 + \frac{N_t (R-1)}{K}}$$ Beverton-Holt (1957) model.

Here R is density independent growth rate.

Take $N_{t+1} = N_t R_A$ (actual) (realised)

$$R_A = \frac{R}{\left\{1 + \left(\frac{N_t R - N_t}{K}\right)\right\}^{b^*}} \quad -(1)$$

In (1), if $N = 0 \rightarrow R_A = R$ $\quad N = K \rightarrow R_A = 1$

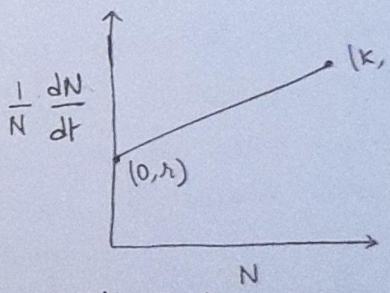
All assuming - its a straight line!

Here $b^* = 1$ If $b^* > 1$ - overcompensation - declines more than necessary

$b^* < 1$ - undercompensation.

Continuous logistic Equation -

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right)$$



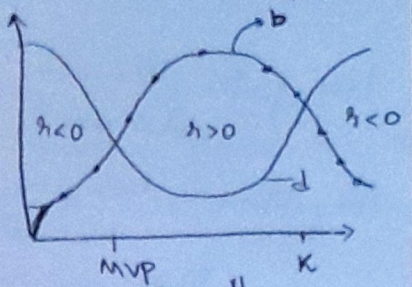
One of the major assumptions of this model -

- Birth & death rates change linearly with population size
  : its also assumed that birth & survivorship rates decrease linearly with density.

Allee effect - Positive density dependence - at very low densities (below the minimum viable population), the growth or fitness of a population is positively correlated with density.

Allee effect – important to study dynamics of small population
Demographic level – reproductive success or survivorship
Genetic level – inbreeding & genetic drift can cause loss of fitness

When $N < MVP$ – extinction ($-ve\ r$)
$MVP < N < K$ – positive growth rate
$N > K$ – negative r



Non-linear modification to cont. logistic eqn –

Generally, $N_{t+1} = N_t e^{r\left(1-\frac{N_t}{K}\right)}$ Introduce θ – $N_{t+1} = N_t e^{r\left[1-\left(\frac{N_t}{K}\right)^{\theta}\right]}$

When $\theta < 1$ – density dependence is strong even when $N \ll K$
$\theta > 1$ – density dependence is weak until $N \sim K$

Continuous lagged logistic model –

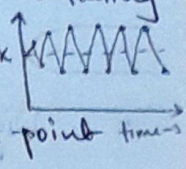
$$N_{t+1} = N_t e^{r\left[1-\frac{N_{t-\tau}}{K}\right]}$$

The lag makes the population oscillate based on value of $r\tau$.

Introducing stochasticity (demographic [c] and/or environmental [K]) vastly changes the dynamics of the population.

(15) ★ Exponential logistic Growth (Discrete logistic in Populus)

- Initially, when $N_0 < K$ and r is small, the curve follows similar path as Continuous logistic growth. ($N_0 = 10$, $K = 500$)
- But when we increase r ($\approx 1.4 - 1.8$) the population goes above K, so it decreases and stabilises.
  When we increase r further, it takes longer to stabilise but it still damping oscillations.
- When increased further ($r \approx 2.05$), the population is oscillating between 2 points (after an initial phase)
- These are known as limit cycles with 2 points
  As we keep increasing ($r \approx 2.55$), it becomes a 4-point limit cycles.



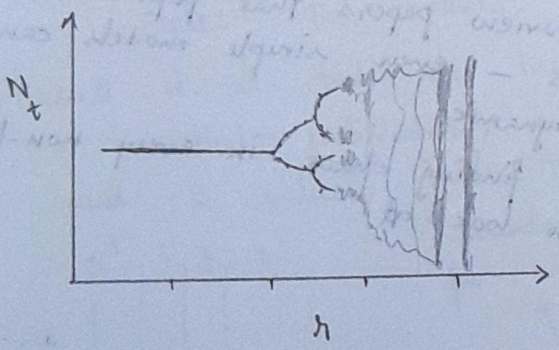
- As we keep increasing r, periodicity goes from $2 > 4 > 8 > 16 \ldots$
- At around r (≈ 2.8), the cycles disappear and the system becomes chaotic.

We're iterating a deterministic equation but getting seemingly random (stochastic) results. This is called Deterministic Chaos.

* Discovered by Ed Lorenz in 1970's

Bifurcation Map.

Its a simple way to represent the trajectory of the system as we keep changing r.
In the Y-axis, we plot the values of N_t (achieved after a long time at some equilibrium point) for different values of r.
The bifurcation or diagram looks like –



For large value of r, when it becomes a chaotic system, the value of N_t can't repeat – if it did, it would enter into a loop.
So, there are infinite points of N_t at each r, so it appears a smears.

Also, this diagram shows a self-similar pattern at smaller and smaller scales – i.e. its a fractal

Lecture 09
Comparing Models to real life.

⇒ Continuous Logistic Growth

The increase in human population in USA followed this curve – but the exponential growth lasted longer because of post WW2 economic boom.
This model is quite useful.

Digression to history of science.

- Pierre Simon Laplace (1814) – absolute determinism : an intellect given all data (precise & accurate) can compute everything that is happened and will happen.
"Laplacian demon."

- Henri Poincare (1908) – Its impossible to measure any value perfectly precisely. Any small change in initial conditions can change the outcome drastically.

- Edward Lorentz & weather patterns.
* In a deterministic system (!), from nearly the same starting point, very soon the patterns diverged and progressed very different.
* Butterfly effect – sensitive dependence on initial conditions.
* He proved that even small imprecisions will make long term climate prediction impossible.
His ideas didn't become popular.

- Edward May – He wrote review papers that popularised the idea of chaos – even simple models can lead to very complicated dynamics
This began the era of finding chaos in every non-linear model one cared to look at.

(17) How do we differentiate real chaos from stable point/oscillation with some noise?

→ Method I

Estimate the Lyapunov exponent from time series data.

Lyapunov exponent (λ) is a quantity that characterizes the rate of seperation of infinitesimally close trajectories.

Two trajectories in phase space with initial seperation vector δZ_0 diverge at rate given by equation —

$$|\delta Z(t)| \approx e^{\lambda t} |\delta Z_0|$$

This requires very long time series (at least few hundred data pts) which are possible to obtain for physical systems, but not biological population (except brain patterns & heart rate pattern)

→ Method II

From parameter estimates by fitting models to time-series data.

Here, you take a time series data and fit a model to it (like continuous logistic); then get the estimates of the parameters.

Pros - easy to do
works for short, noisy data sets

Cons - One must assume that the model being fit is correct
Fitting models is an art.

Hassell et. al —

$$N_{t+1} = \lambda N_t (1 + aN_t)^{-\beta}$$

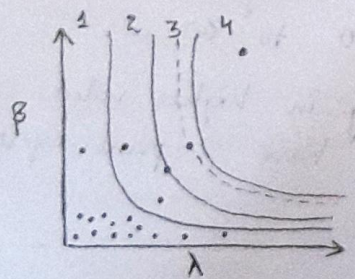
similar to Ricker model

here λ : growth rate parameter

a & β : parameters regulating density-dependent feedback term

The bifurcation map of this is similar to Ricker model (exp logist) and its only affected by λ and β, whereas a just scales the values.

Hassell et. al. took 24 insect populations (because it was good theoretical justification) and fit this model to obtain λ and β values. From the model, they created stability boundaries between λ and β i.e. values for which population shows different kind of growth.



Then they estimated λ and β values of 24 populations and plotted them.

1. Monotonic damping 2. Damped oscillations
3. Stable point oscillation 4. Chaos.

They find out that 22/24 exhibited monotonic damping.
⇒ Most natural populations exhibit relatively simple dynamics. But why?

1/10/20

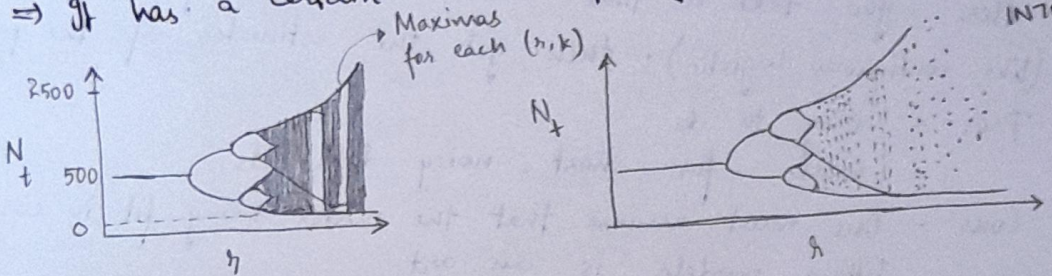
Lecture 10

Even when populations have very high growth rates, they don't exhibit chaotic dynamics. Why not? Henson et al. 2001

1. Real populations are always in integers, whereas most model outputs give a decimal value
But if we force the output to be integer (round it off), then chaos disappears — in a chaotic system, the values never repeat themselves.

Population size is a bounded integer variable. due to finite resources

The equation of the model is quadratic in N_t
⇒ It has a certain maxima for given (r, k) value



The minimum value is 0.
When integerized, the no. of points between 0 & maxima becomes finite and hence, the values have to repeat.
Thus Deterministic Chaos disappears. but stochasticity remains
Also, integerised model doesn't say that the dynamic can't be very complex (n-limit cycle).

This phenomenon is called Lattice effect.

2. Effects of Constant immigration.

$N_{t+1} = f(N_t) + c$ $f()$: Ricker, $K = 500$, $N_0 = 10$

We take out the influx due to immigration outside r.
As we increase c from 0 to 60 —
chaos starts to disappear (starting in higher values of r),
and at $c = 60$, we only have 2-point cycles.

WHY?

The realised growth rate decreases with increase in population but when we take 'c' outside, the no. of immigrants remains constant regardless of size of population. Subtraction i.e. emigration – Soudutta Sinha – PNAS (1993/5)

Model Complexity [Turchin & Taylor, 1992, Ecology]

They wanted to know if Harrell et al got their result (i.e. simple dynamics in real life) because of their relatively simple model of form: $N_{t+1} = f(N_t)$.

They decided to analyse the data directly.

They considered a bunch of time series – original Harrell et al & some vertebrate ones – and fit polynomials to it instead of established models.

With a complex polynomial with high degree, almost any curve can be made to fit perfectly. ##

So they introduced a penalty function: for each increase in complexity of model, the penalised the betterment of the fit by increasing parameters (?)

They used Akaike Information Criteria (AIC) for this

While Harrell et. al. only considered one time lag, they considered multiple time lags – i.e N_{t+1} would be dependent on N_{t-1}, N_{t-2} etc. as well as N_t.

With these conditions, they constructed a suit of complex models and for each time series and fit them to choose the one that would fit the best.

Then they extracted the parameter values from this model to see if it would give monotonic damping, oscillation, limit cycles or chaos – all were observed in natural time series.

To avoid overfitting, they came up with a way to optimize the complexity of the model using AIC.

Conclusion: Use of overly simple models for reconstructing endogenous dynamics from data may be biased in favor of finding stability.

⇒ Harrell et. al. were wrong?

Complex high dimensional objects can have simple low dimension projections – so be careful when inferring the former based on the latter.

Theoretically, refugia, migration, spatial structuring & variation have been shown to stabilise the dynamics of population models.

Lecture 11 — 7/10 — (20)

Stabilising Biological Populations

Controlling the dynamics of real biological population

Most of it has been developed theoretically and not experimentally. The problems with this chaos control models are —

i. Poor knowledge of system
ii. No access to system parameters — i.e. we can't directly control r & k. In fact, we only find out r & k after we get the data, fit a model & estimate them
iii. Short and noisy time series — difficult to distinguish between different dynamics.
iv. Biological systems are inherently noisy & extinction-prone

Theoretical approaches — to fix the drawbacks of models & simulation

* Perturb state variables (like 'c' in pinning), rather than parameters (r, k) → something you have access to during the expt.
* Concentrate on measurable & biologically relevant indices of stability — here, constancy & persistence

Does pinning really work?

Local perturbations doesn't affect the stability i.e pinning doesn't work — because: extinction of subpopulations reduce the impact of pinning → in spatially structured metapopulations

Devising a method to stabilise dynamics of population —
- implementable in real populations
- robust to noise & extinction
- applicable in a biologically meaningful parameter range

Then, they empirically validated the proposed method.

* Adaptive Limit Control (ALC)

Essentially means: don't allow population to go (above)/below a certain threshold level (which is determined based on previous population size). Just in LC → Not in ALC

$$\begin{cases} N_{t+1} = f(N_t) & \text{if } N_t \geq c \cdot N_{t-1} \\ N_{t+1} = f(c \cdot N_{t-1}) & \text{if } N_t < c \cdot N_{t-1} \end{cases}$$

Don't allow population to go below a fraction c of the size of its previous generation

Modeling the dynamics – Ricker Model

$$N_{t+1} = N_t e^{a\left(1 - \frac{N_t}{k}\right)}$$

Why this – • Simple generic model with no species specific parameters, ⇒ it can be generalizable

• Derived analytically from first principles based on biological assumptions – * All individuals are spread randomly
* Includes scramble competition

• Empirically shown to be widely applicable – better to fishes when biomass is grown as discrete population

Quantifying Stability

→ Constancy
Reduction in amplitude of fluctuation in time series.

Fluctuation Index : $FI = \frac{1}{\langle N \rangle} \cdot \frac{1}{T} \sum_{t=0}^{T-1} abs\left(N_{t+1} - N_t\right)$

Makes it dimensionless — Average

→ Persistence
Reducing the frequency of extinctions within a given time

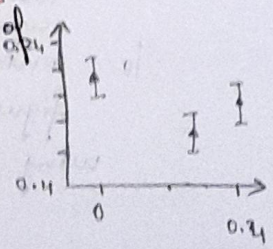
⇒ Experiment on spatially unstructured (homogeneous) population
Using Drosophila melanogaster. Consider 3 values of c

$c = 0$ (control), $c = 0.25$, $c = 0.4$

↳ if $N_t < c.N_{t-1}$, they bring it up to $c.N_{t-1}$, but
if $N_t \geq c.N_{t-1}$, it's not disturbed

8 replicate populations per treatment – census over 13 generations

Observation: ✓ ALC enhances constancy stability of experimental populations
Even a low value of ALC enhances persistance of experimental populations, although difference is not significant at 5% level. (error bar)

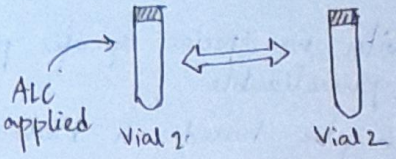


∴ ALC can stabilize dynamics of spatially unstructured population.

Metapopulation – subpopulations connected by migration
Migration rates affect metapopulation constancy

So this has to considered as a population

⟹ Experiment on two-patch metapopulation
- 8 replicate metapopulations, 15 generations
- Rate of migration : 10% (stabilising) & 30% (not)
- c = 0, 0.25, 0.4



Observation -

* ALC reduces metapopulation FI under high migration rate (ie when its not stable) but not low migration rate (when its already stable).
* ALC promotes persistence under both migration rates but not much difference b/w c = 0.25 and c = 0.4

Summary

- ALC is a potent mechanism to increase stability in real populations. (First method empirically shown to work)
- Since empirical results are supported by many models, its likely to be broadly applicable
- How generalizable are these methods ? Does it work at other rates of migration, k values, extinction and higher no. of subpopulation.
- Mathematically establish why ALC attains stability in single population

Comparing Efficiency of control methods -

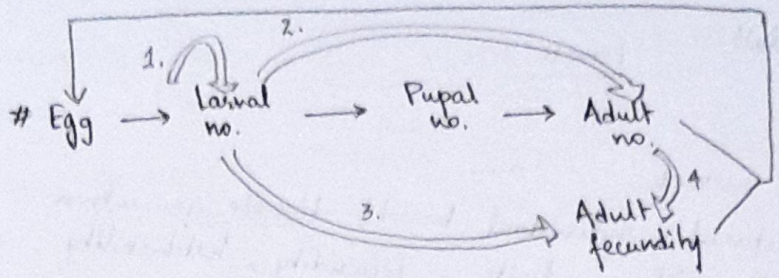
For large enough control parameter values, most control methods can induce (almost) any level of stability.

To ~~control~~ compare different models, we've to fix a level of stability, figure out some parameters & compare the efficiencies using some kind of composite index.

Eng Lecture 12

One-parameter control – this method can't attain 3 kinds of stability, only 2 at a time
i.e limiting from above or from below
While two-parameter control can achieve that.

Individual based model for Drosophila dynamics
Interaction of life-history traits with nutrition ability to shape population dynamics & stability.
This model is species specific.
There 3 Feedback loops govern dynamics of Drosophila populations

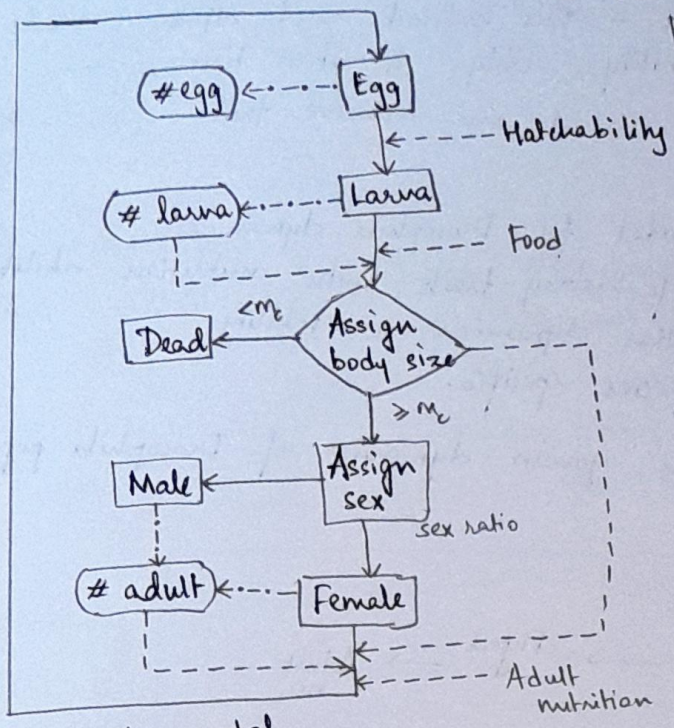


There are also cases of egg cannibalism, but essentially ignorable

- A larva needs to have a certain critical mass to survive the pupal stage. This attainment of mass is dependent on each individual# and the nutrition available.
So (1.) represents the density dependence of larval survivorship.
- (2.) is obvious – greater the no. of larva, greater the adult population
- The adult fecundity is also dependent on the larval mass – because it means they have that much more/less resources to lay eggs : (3.)
- (4.) When no. of adults increases, the per capita adult fecundity decreases because – * limited food
  * increasing waste accumulation.
  * also, because of jostling & the mechanical hassle increases when numbers increase and space remains same – this reduces fecundity

i.e. even with unlimited food supply, there's a spread of the mass attained.

Schematic



This works for any holometabolous population ie. life cycle of -
Eggs → Larva → Pupa → Adult

Features of model

- * Stage structured, individual based, discrete generation
- Includes life history traits - fecundity, hatchability, critical mass etc which is typical of many holometabolous insects.

There are two density dependent criteria —
* Larval food - high / low
* Adult nutrition (yeast) - high / low

From this we get 4 regimes —
H H , H L , L H , L L
larval Adult - in this order

Over 49 generations with 8 replicate populations
In the 4 regimes, the population size distribution & fluctuation index (FI) was observed

HH & LL were used as training datasets - the data that's used to parametrize the model.
Once the parameters are obtained, then they check if the rest of the data (Test data set) fits well to the model or not.
↓
LH, HL.

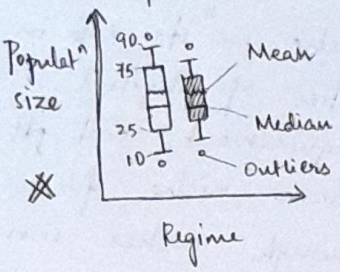
★ Population Distribution

Refer to the box plot – 30:00 mins in Lec. 3.6.

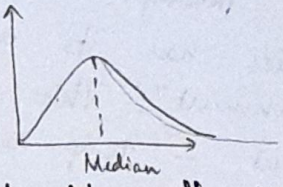
Box plot – plots the values between 25th & 75th percentile called, Inter-Quartile-Range (IQR)

⇒ Median ~~Mean~~ : 50th percentile.

Expt, simulation



⇒ Skewed data



when median & mean intersect like this, ⇒ normal distribution

The model fit pretty well.

★ Fluctuation Index (FI)

Decending order of FI : LH > HH > LL > HL

∴ They showed that model captures the dynamical features of empirical data both qualitatively & quantitatively in all four regimes

Once we know that the model works –

Model exploration – mechanistic understanding

What happens to FI when the parameter values (critical mass, hatchability, adult-density & adult size) are varied

sensitivity to

Observations –

i) Life-history parameters differentially affect population stability

ii) Nutritional regimes interact with life history traits to influence stability

iii) Prediction – FI increases monotonically (in all 4 regimes) with increase in m_c

✓ Experimental results validate model prediction

→ simulation

≠ White boxes – expt data Gray box – model data

LL, HH would match (∵ trained sets) But LH and HL also match pretty well (data set)

Lecture 13 - Chapter 04

Three kinds of species interaction

Competition Predation Symbiosis.

The established ecosystems which are self-sustaining are created through a long process called Succession.

Each species has its own unique niche — "its role in environment". This has characteristics of physical environment — temp range, water requirement, soil pH etc.

No two species can occupy the same niche for a sustained period of time. Although niches can overlap and this leads to competition.

Two species with identical niches cannot coexist indefinitely. This leads to the prediction that coexisting species have different niches

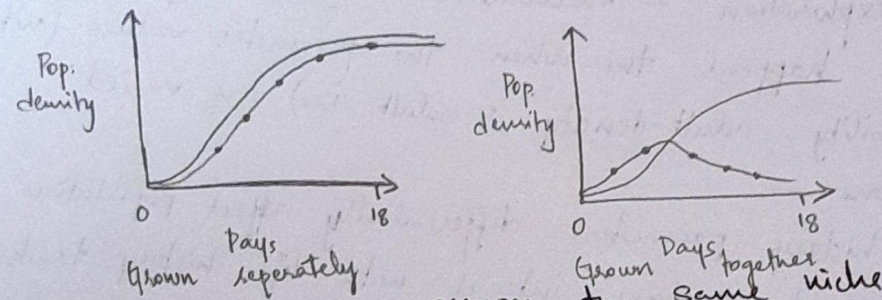
Competition.

It played a big role in Darwin's Theory.

Early experiments —

⇒ Gause's Principle of Competetive Exclusion.

Different species of *Paramecium*



No two species can occupy the same niche.

⇒ Later experiments : Tilman et. al (1981)

He worked with diatoms which have cell walls of silica.

Eg: Synedra & Asterionella - Synedra could survive on lower conc of silica & hence, it always drove Asterionella to extinction in 25 - 50 days.

R* rule in exploitative competition (Tilman)

When 2 species are limited by the same resource, the species that can maintain a positive per capita growth rate at lowest resource level will exclude other species

(27)

What is competition?

* Limited resource is not ~~note~~ a necessary factor for competition. The no. need not be limited – a few individuals can corner most of it, leaving none for others.

"Any use or defence of a resource by one individual that reduces the ability availability of that resource to other individual"

Two groups use the same resource, or seek that resource, to the detriment of one or both.

• Interference competition – involves direct interactions b/w individuals
May occur in absence of obvious resource limitation

Two kinds of competitions –

1. Intraspecific competition
   i) Density dependent growth rate – resource competitions → self-thinning rule
   ii) Competition for mates

Plants decrease in population density as total biomass of population increases

Interspecific competition
Two populations of different species compete for same resource

Features –
- nontrophic interaction
- results in reduction of growth rate (r) or equilibrium number (K) in one or both populations.

L-V Model predicts coexistence when, for both species, interspecific competition is weaker than intraspecific i.e. $\alpha_{12}, \alpha_{21} < 1$

Modelling competition.
Consider two species with logistic growth model (continuous)

$$\frac{dN_1}{dt} = r_1 N_1 \left(\frac{K_1 - N_1}{K_1}\right)$$

Now, we see what happens when we add 1 individual of species 2 into the population.

α_{12} : Interspecific competition coefficient. Measure of how much competition one individual of N_2 will induce in terms of N_1 individuals

So, the equations become –

$$\frac{dN_1}{dt} = r_1 N_1 \left(\frac{K_1 - N_1 - \alpha_{12} N_2}{K_1}\right) \qquad \frac{dN_2}{dt} = r_2 N_2 \left(\frac{K_2 - N_2 - \alpha_{21} N_1}{K_2}\right)$$

LOTKA - VOLTERRA COMPETITION EQUATIONS

Relation between α_{12} and α_{21} ? — Not just dependant on resource consumption but other things like toxins produced.

Analysis:

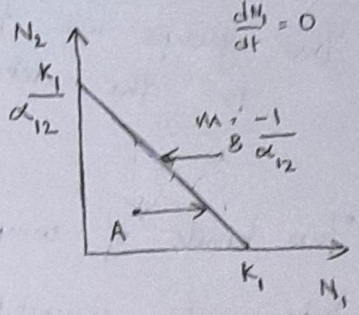
$$\frac{dN_1}{dt} = r_1 N_1 \left(\frac{K_1 - N_1 - \alpha_{12} N_2}{K_1} \right)$$

To find the equilibrium value, we find solution of $\frac{dN_1}{dt} = 0$

$r_1 = 0$ & $N_1 = 0$ are trivial solutions

$$\Rightarrow \frac{K_1 - N_1 - \alpha_{12} N_2}{K_1} = 0$$

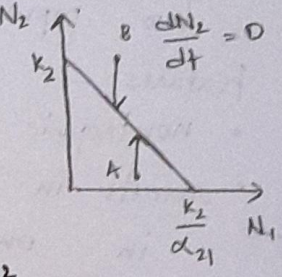
$$N_2 = \frac{K_1}{\alpha_{12}} - \frac{1}{\alpha_{12}} N_1$$



If we decrease N_1 (move to point A), N_2 or both, then dN_1/dt increases [this is what density dependent growth is all about] ⟹ Population of N_1 increases

So the line $\frac{dN_1}{dt}$ contains all the stable points and if the system were anywhere else, it would try to come to that point.

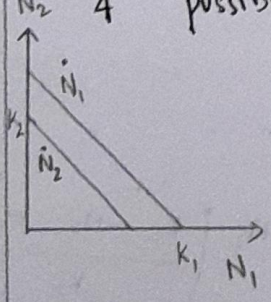
So if system were at A, N_1 would increase & at B, N_1 would decrease

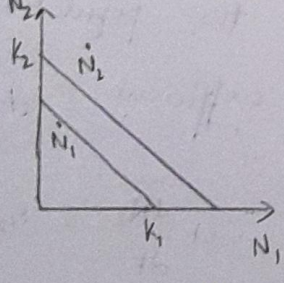


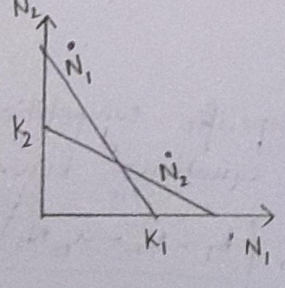
For species 2, $\frac{dN_2}{dt} = 0 \Rightarrow N_1 = \frac{K_2}{\alpha_{21}} - \frac{1}{\alpha_{21}} N_2$

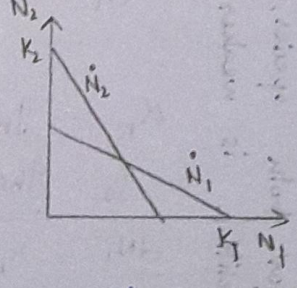
These lines on N_1-N_2 plane are called Zero-growth isoclines.

If both lines were plotted together, we have 4 possibilities —









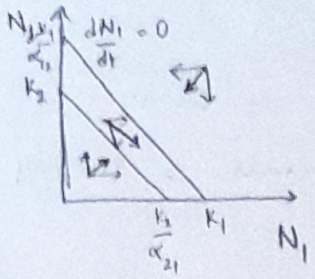
Everywhere along the lines, population growth is stopped

Population growth for 2 species will stop when : $N_1 = K_1 - \alpha_{12} N_2$ $N_2 = K_2 - \alpha_{21} N_1$

Lecture 14

Isoclines

Case I

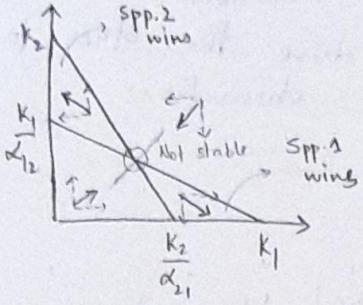


In the middle region, the population dynamics goes down to N_1 diagonally, and hits X-axis i.e. species 2 is driven to extinction.

" Species 1 wins"

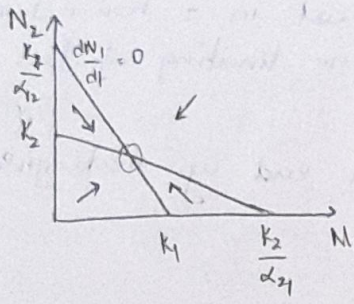
Case II : Similarly, " Species 2 wins"

Case III



In the intersection of two lines, at that point, the two species will co-exist but in unstable equilibrium because even with small perturbations, the system will drive to extinction of one of the species.

Case IV



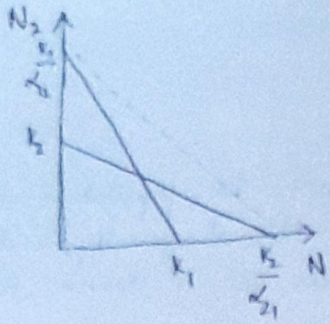
Here, the intersection point is a stable point. The dynamics will converge here and two species will exist stably.

When two isoclines overlap, the two species behave as one — as long as it satisfies : $K_1 = \frac{K_2}{\alpha_{21}}$ $\quad K_2 = \frac{K_1}{\alpha_{12}}$

As a species is driven to extinction, the dynamics accelerate but as the two species go towards stable points, the dynamics decelerate

This acceleration & deceleration is a function of r. But in this model, we are only considering the 'result' - the asymptotic value of population dynamics.

IV : Each species is limited more by its conspecifics than interspecific competition.



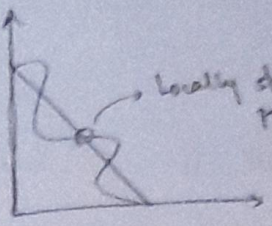
For species 1 to drive up 2 to extinction, the direct way is to increase value of K

⇒ K-selection - increasing competitiveness

α - selection

(1) Increasing α_{21} - another way of increasing competetiveness is having non-linear, curved iso clines.

Another way of increasing



In L-V model,

We see that in 3 out of 4 conditions, model predicts that one species would drive the other to extinction

This agrees with the experimental observations.

5/10/20

Lecture 15

How does the model compare to real life systems?

Invasive species

They are species that are introduced in a non-native environment where they have no limiting factors

Eg. Lantana, Congress grass, Apaima & Alligator gas - Kerala

These species out-compete natives and end up destroying the flora / fauna divessity.

Controlling invasive species.

Instead of trying to eradicate such a species, another approach is to use it and integrate it.

Eg: Eichhornia (Water hyacinth) - 300 women in Paramus (KIDS) employed to make mats & crafts

(self study) - Asian; carps, Caulerpa Texifolia.

(21)

In nature, lots of species with similar niche seem to co-exist.

What is a niche? Thomas Schoener, (2009) Review

→ Ecological niche : n-dimensional hypervolume

Consider an n-dimensional space where each axis represents various conditions like, temperature, pH, humidity, rainfall etc. For each of them, there's a range within which the species can exist — this gives us a specific hypervolume

Hutchinson : Ecological Niche is a quantitative description of the range of environmental conditions that allow a population to persist (positive growth rate) in some location

→ Resource-Utilization niche

MacArthur & Levins : Focuses on what the species actually do — especially how they utilize resources

Niche axes — Habitat (microhabitat, macrohabitat)
Food type (food size, hardness)
Time (daily activity, seasonal activity)

Niche is a very divisive topic — some ecologists believe its just heuristic, others believe its useful in models.

When is co-existence possible?

If there is seperation/partitioning of niche in terms of resource partitioning. This reduces the interspecific competition.

01. SPATIAL PARTITIONING

▲ Gause's expt. with P. caudatum and P. bursaria

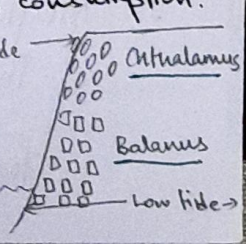
They co-exist as P. caudatum feeds on top layers of culture while latter feeds on the bottom.

≠ Whereas both P. caudatum & P. alt... are both surface feeders.

▲ Spatial niche seperation #2 : Connell's Barnacles

Barnacles — marine, sessile arthropods. — they attach to the hull of a ship and cause a drag, increasing fuel consumption.

The larvae of two species of barnacles are motile. When Barnacle settle, they always grow in a certain distribution.



Experiment —
- When Chthamalus is removed, Balanus doesn't grow above its level
- When Balanus is removed, Chthalamus grows downward and covers the whole cliffside

This is because : Balanus outcompetes Chthamalus and covers the cliffside till a certain level.
Above that level, Balanus can't grow, but Chthamalus can because its more resistant to dessication, which allows it to survive at a level of high tide.
So, Fundamental niche : theoretical niche
Realised niche : Niche that is constrained by interaction with other species

Balanus is bigger and more muscled. So it can grow (develop, not a value) faster & better than outcompeting Chthamalus.

(?) This is an example of α - selection.

02. Temporal partitioning
The common spiny mouse and golden spiny mouse ~~are~~ co-exist in deserts of Israel
The overlap in - habitat use (rocky use)
food items (arthropods)
reproductive seasons

But they are active during different times - common one is nocturnal while golden one is diurnal.

They removed the common spiny mouse from regions and observed that golden spiny mouse became nocturnal in 6 months.
People observe that some species share resources. Then they try to determine if its due to competition.

16/10

Lecture 16

03. Morphological seperation : Character displacement
Phenomenon by which character traits of two or more closely related species differ more when they occur together than when they occur in different geographical areas.
Eg: Darwin's finches in Galapagos # islands.

Conditional Niche Seperation - Thomas Park (1954)

He worked on two species of flour beetles whose growth depended on Temperature & humidity.

Two species : Tribolium confusum (cool, dry)
Tribolium castaneum (warm, humid)

∴ Physical conditions can be critical for outcome of competition.

Highes equilibrium size (No. of adults, larvae & pupae) doesnot guarantee winning the competition.

- Priority effect: When these beetles were grown in intermediate environmental conditions, the species establishing itself in greater numbers first generally wins out in competition (i.e outcome not always predictable) 24/10

Lecture 17

Predation

Process by which an organism eats all or part of another organism.

Why study ? - Pest management, pisciculture
Conservation
Evolutionary role

Types of Predation (Exploitative interactions)

Herbivory — Carnivory — Cannibalism.
Parasitoidism — Parasitism

Parasitoid - an insect whose larva consumes its host and kills it in the process. - functionally equivalent to predators.

Hudson's Bay Company - kept records of the pelts they bought of Snowshoe Hare and Lynx.

Elton (1924) noticed that there are regular oscillations in prey and predator densities.

The population of prey occurs first and then predator population spikes.

Theories -

- External factors : Sunspot cycles, climate changes
- Intrinsic / indegenous factors.

Population cycle in snowshoe hare is the result of interaction among 3 trophic levels - hares, their food supply (elevated conc of defensive chemicals) and their predators.

Parasites can alter the behavior of their hosts

Modelling predation – Lotka-Volterra worked on this

Host → H Predator → P.

$$\frac{dH}{dt} = rH - aHP$$

1 2

• a: rate of predation

1. Assumed that in absence of P, the host grows exponentially
2. This growth is reduced when H and P come across one another and a fraction of them (a) result in a kill.

$$\frac{dP}{dt} = c.aHP - mP$$

1 2

• c: conversion factor

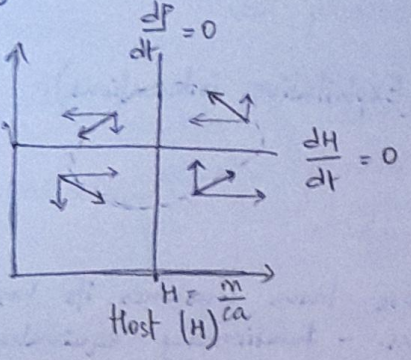
1. c: efficiency of P to convert the kill to energy to make babies.
2. m: density independent death rate

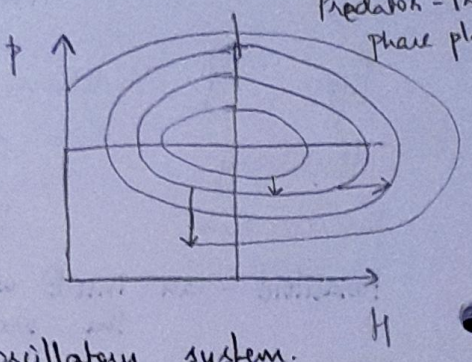
To draw isoclines, we consider –

$$\frac{dH}{dt} = 0 \Rightarrow rH = aHP \Rightarrow P = \frac{r}{a}$$

$$\frac{dP}{dt} = 0 \Rightarrow c.a.HP = mP \Rightarrow H = \frac{m}{ca}$$

} constant





This model ~~et~~ gives us an oscillatory system.

It also exhibits Neutral stability: when the system is perturbed, it won't go back to the initial point, but it'll attain a new trajectory which includes that point.

A large perturbation can take a trajectory that leads to extinction of one or both species.

Eg: largely reducing no. of predators leads to their extinction.

Assumptions –

- Growth of Host is only regulated by predation & nothing else.
- Predator is a specialist that can persist only if victim population is present.
- No change in environment
- Predator and host encounter each other randomly in a homogenous environment.

To make it more realistic –

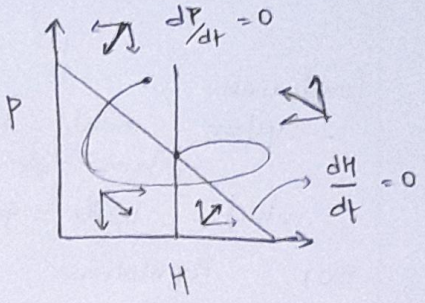
- Density dependent growth rate of host
- Competition b/w host & predator for resources like water,
- Temporal delay / inhomogeneity. ie mating season.
- Overtime, the prey can learn to avoid predators

Consider density dependent growth –

$$\rightarrow \frac{dH}{dt} = rH\left(1-\frac{H}{k}\right) - aHP$$

$$\frac{dH}{dt} = 0 \Rightarrow P = \frac{r}{a}\left(1-\frac{H}{k}\right)$$

$$\rightarrow \frac{dP}{dt} = c.aHP - mP.$$

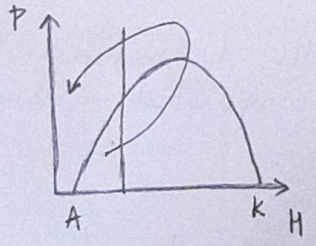


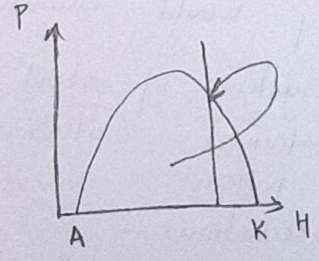
We see that there are dampening oscillations ie the populations will converge to a point.

Arditi-Ginzburg model of Ratio-dependent functional response – Pg. 324 Molles & Sher

Rosenzweig-MacArthur Model

R-M doesn't assume that the isoclines are linear. Depending on where where predator isocline intersects the prey isocline, this system shows different behaviours.





A: Allee Point (?)

Lecture 18 — 22/10/20

Net-logo Prey-Predator Model

The wolf-sheep simulation is very close to the Lotka Volterra model, except it is finite (no. of population).

Thus, their interactions are random so we get different kinds of dynamics — if wolf going extinct or both going extinct

=> Realistic L-V model doesn't give coexistence or oscillations, like it does in real life.

Gause's experiment

Paramecium : Prey (refugia) Didinium : predator

a) No predator : led to extinction of both

b) With predator : leads to extinction only (refugia) Refugia : glass wool Paramecium could enter it

c) With constant immigration of prey (& refugia) and predator : led to sustained oscillations.

Conclusion:

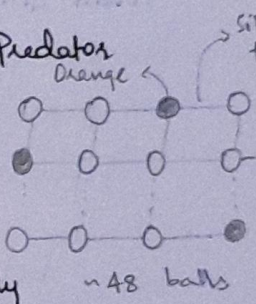
System would be driven if left to itself. But its an open system => there are constant interferences from outside which leads to sustained oscillations

BUT : Populations can be closed systems and oscillations are still observed

Huffaker's Expt.

He experimented on two species of mites on spatially distributed oranges

E. sexmaculatus - Prey T. occidentalis - Predator



He created a heterogenous system with oranges and plastic balls. Mites could only grow on oranges and this system ensured they would encounter each other randomly

In a system of about ~48 balls, he manged to attain sustained oscillations with complicated arrangement of patterns.

His observations -

- Distributions and relative abundance of mites changed over time
- Prey needs some advantage to survive, or it'll be driven to extinction [In form of refugia or patchy land => so it can stay ahead]

Refuges — 1. Space (burrow, trees, air)
2. Safety in numbers (predator satiation)

- Metapopulations promote prey-survivorship by extinction-recolonization
- If predator is efficient, prey can only survive in patchy environment.

In nature -

Predators can reduce population size of prey, even more effectively than pesticides

Many kinds of dynamics are introduced because -
- Multiple prey - multiple predators
- Spatial heterogeneity (refugia)
- Predator efficiency changes (ie it won't hunt if its not hungry)
- Evolutionary changes in prey & predator

Video:

→ Prey defense strategy
- Run / fly away from predators
- Use armour, quills or thorns for protection
- Camouflage : blend in to its surroundings
  # Even predators use camouflage to attract prey.
  Eg: Orchid mantis
- Chemical warfare : Skunks & Bombadeer beetle
  Poisonous animals - show warning coloration
  - Batesian mimicry : Harmless animal mimics a dangerous one
    Eg: Beetles & flies mimic the yellow-black band of bees, wasps
  - Mullerian mimicry : Two or more unpalatable animals resemble each other to reinforce avoidance by predators by increasing frequency of unfavourable encounters.
    Eg: Monarch & Viceroy Butterflies.

→ Predator offense strategy
- Run / fly faster to catch the prey
- & Use venom to kill or stun - snake, wasp, spider
- Mimicry : Alligator turtle - tongue = worm

→ Coevolution : Two species evolve in a coordinated fashion by adapting to changes in each other

Example of coevolution –

- Heliconius butterflies lay eggs on the leaves of Passion flower vines which are poisonous to other herbivores.
- But caterpillars of this butterfly eat the leaves & thrive on it.
  In order to avoid crowding, these butterfly don't lay eggs on leaves that already has a 'yellow mass' i.e eggs on it.
- Hence, the plant grows a bright yellow structures on some of the leaves, preventing ~~their~~ butterflies from laying their eggs there.
- Moreover, these structures act as nectaries which attract ants and other insects which eat heliconius eggs.

18/11

Lecture 19

Aral Sea – Lake in Central Asia (Khazakhstan). 4th largest lake (1960) → 142x Pune
The water from 2 rivers that fed this lake was diverted for cotton agriculture (Soviet initiative).
Its been drying up ever since – now $\frac{1}{10}$th of its size.
- This is a man-made ecological catastrophe – it left a lot of small fishing posts destitute
- Health toll : Rising illness, falling life span
- Then the Govt. & World Bank built a dam to increase level of the Aral Sea, so it has gotten a little better
- When supply was cut off, a lot of water evaporated (bcoz bowl not wide) leaving the water salty.
  This was exacerbated by increase in use of fertilisers and chemicals left behind by weapons testing in Soviet era.
- Ecological demise :
  Fish 32 → 6
  Birds 319 → 160
  Mammals 70 → 32

So, human intervention, basically destroyed the lake.
But, even left to themself, many lakes suffer a similar fate due to Succession.
This is due to Eutrophication.

Predator Avoidance
Lecture 5.3

21/12

Refer video – pg. 37

One of the major strategies –

⟹ Coloration

- Aposematic coloration – warning coloration markings that make a dangerous, poisonous or foul-tasting animal particularly conspicuous and recognizable to a predator.

Eg: Yellow-winged darter.

- Crypsis – blending with the background in such a way that predators can't recognise you.

Eg: Dead-leaf mantis, tawny frogmouth (bird), leaf-mimicking mantis, lantern fly.

Its important to get the color as well as the pattern right.

- Mimicry – attempts by one species to resemble another species.

* Batesian mimicry

• Here, the model has an attribute that makes it unpalatable to predators. The mimic sends the signals similar to the model but doesn't share the unpalatable attribute

Eg: Drone fly mimicing honey bee

• Producing the ~~up~~ unpalatable attribute (sting, poicon etc.) is considered costly. Moreover, the mimic might not be able to produce this attribute. So if mimics a species which is actually dangerous.

• Also, the increased frequency of encounters with the ~~up~~ unpalatable prey increases the rate of the predator's learning.

So, the no. of models & mimics need to be critically maintained in an ecosystem.

i.e if the predator encounters more mimics than models, then it won't learn that coloration is harmful.

* Mullerian mimicry

Here, two or more species have similar warning signals and both share genuine unpalatable attributes. If a predator confused them with each other, individuals in both species

are more likely to survive because the frequency in of encounters increases. which means the predator will learn faster.

Eg: Heliconis butterflies

⇒ Behavioral

1. Catelepsis - prey playing dead so predator ignores the prey
   Eg: Opossums, Costa Rican katydids

2. Intimidation display - attempt to avoid predation by startling the predator long enough to get away or to convince it that prey will be too costly to attack

   Eg: moths

3. Group living - helps in watchfulness, mobbing and confusing. Alarm calls, alarm pheromones in aphids.

⇒ Chemical strategies

They make the prey too toxic or smelly or too distasteful to eat.

Eg: Badger and skunk.

Bombardier beetle - stores two chemicals in seperate compartments near the anus. - when threatened it mixes them and creates an exothermic reaction i.e. scalds the predator with hot, smelly jet of liquid.

Toxins can poison the predator, but it doesn't save the prey
Needs to be associated with aposematic coloration.

Winnogradsky's Column

Eutrophication

→ Classical view of lake types –

Oligotrophy	little productivity & clear waters
Eutrophy	high productivity & greenish waters (phytoplankton)
Dystrophy	low conductivity & brown waters (dissolved humic substances)
Extinction	lake becomes a marsh/swamp.

→ Modern View of lake types

The state of lake is considered as a continuum in which 'oligotrophic' is a region. The productivity of a lake is mainly dependent on amount of Phosphates.

Condition	$[P_{tot}]$ $\mu g\ l^{-1}$
Ultraoligotrophic	< 5
Oligotrophic	5 – 10
Mesotrophic	10 – 30
Eutrophic	30 – 100
Hypereutrophic	> 100

⇒ Factors determining 'trophic' condition of lake

1. Size of basin – mean depth
2. Nature of drainage basin – Erosion rates
   Types of rocks / soil composition
3. Climate – Rain & snowfall
   Mean temperature (which affects rate of evaporation and pO_2, pCO_2 etc.)
4. Human activity – Fertilisers (N, P)
   Human waste product (domestic, industrial)

   Cultural Eutrophication

⇒ How does trophic condition change?

01. Nutrient enrichment (mainly Phosphorus) through natural & man-made activities (agricultural, industrial).

02. Eutrophication
⇒ Increased organic matter production through algal blooms, mainly blue-green algae (Anabaena, microcystis etc.)
- They form a layer/mat of organic matter, reducing availability of light to lower layers.
- This causes death of rooted aquatic plants, resulting in reduction of dissolved O_2, particularly hypolimnion.
- This anoxic reaction releases even more phosphorus from the soil (This leads to positive feedback cycle :: algae keep growing, not limited by P)
- This leads to increase in pH of water

03. Death of fish and other aquatic life forms.

Eutrophication is not just a problem of small lakes — its also been observed in northern part of Caspian Sea.

19/11

Lecture 20
How do we reverse Eutrophication?

Lake Washington, Seattle
1940s - Oligotrophic lake
1950-1960s - sewage disposal in the lake → Lake Stinko
1970 - W.T Edmondson : studies to understand the cause - sewage was implicated.

Because of his work and initiatives, sewage was diverted to sea

Transparency of lake is measured in terms of depth
- Once sewage was redirected, there was reduction in P-level and lake condition improved
- Further improvement happened when Daphnia (crustacean) came back due to disappearance of cyanobacteria.
- The main implication was on detergent manufacturers — they were compelled to switch from phosphate salts to non-phosphate salts.

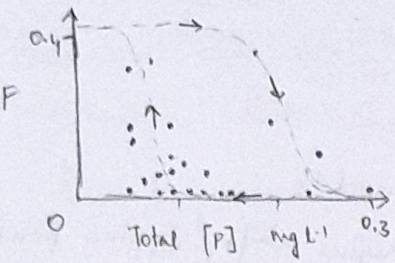
Video: Lake Eyrie - shallowest of 5 great lakes
recent (~2010) eutrophication due to fertilisers runoff

The rainfall pattern affects the fertilisers runoff.
The nutrient distribution in a feild is not uniform - so its better if you locate deficit areas and apply fertilisers as required.

In both cases, phosphate conc. seems to be the major problem.
Its not possible to reduce P in fertilisers because its also used by plants

But P levels are not the only controlling factor

Scheffer et al 1



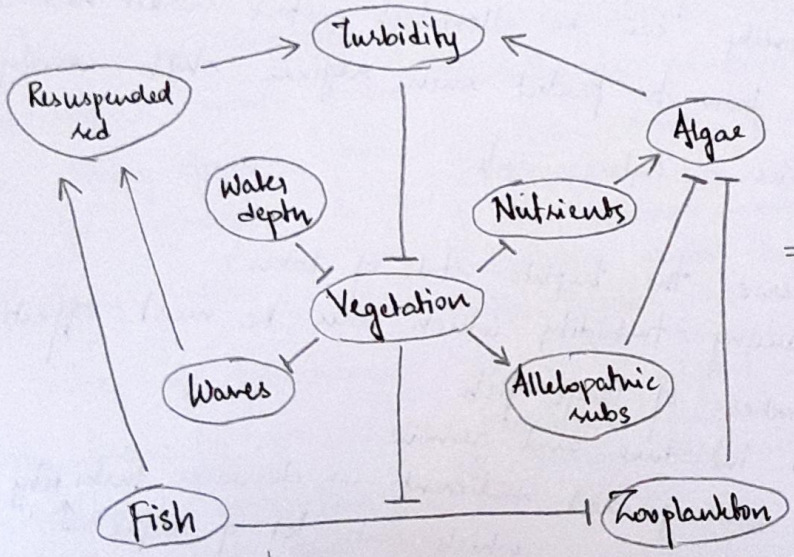
Lake Veluwe, Netherlands

F: Fraction of lake surface covered by charophytes
Charophyts - submerged vegetation - more the better
Good proxy of the health of the lake

* As P level increased, F decreased to near zero
But when they started reducing P levels, F didn't increase right away - it started increasing once [P] was sufficiently reduced
* It follows a hysteresis curve

Interaction in Shallow lakes

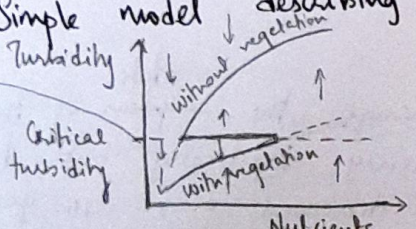
→ ⊕ → ⊖



* Multiply the signs to get interaction effect of two components

⇒ Vegetation & turbidity have negative effects on each other

Simple model describing Turbidity vs. Nutrients - Alternative Stable States



Catastrophic regime shift - TIPPING POINT

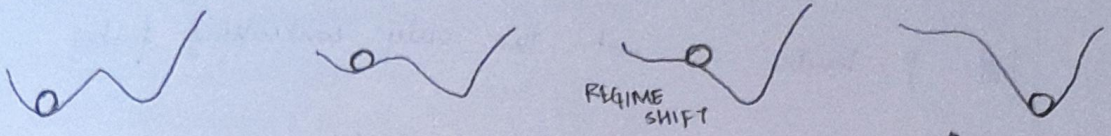
Turbidity is kept in check by the vegetation i.e as you increase nutrients turbidity increases slowly in presence of vegetation, but after a point it jumps ∴ vegetation is killed off.

But as you reduce nutrients, the turbidity takes much longer to come below CT (in absence of vegetation)
So, even when you start reducing [P] levels, you've to reduce it to much lower levels before vegetation can grow again.
This is what is responsible for the thyskrosis curve
Making the model more realistic doesn't change the qualitative conclusions

20/11/20

Lecture 21

Another way to visualize alternative stable states –



Clear water shallow lakes

P accumulation in soil & lake mud

Flooding, warming, overexploitation of predators.

Turbid-water lakes

- These notions have solid mathematical models
- They belong to catastrophe theory & regime shift / tipping point which studies questions like –
  - Why Sahara became a desert
  - Why coral reefs collapse
  - How poverty can be alleviated, stock market rise & fall etc
- Research on how to predict such regime shifts – early warning signs
  # Cybernetics – Taylor's rule

How to reverse the trophic state of lakes?
By reducing turbidity which can be most effectively done by –
Reducing number of large fish
- Easy to introduce and remove
- Decreases resuspended sediment → decreases turbidity
- Increases zooplankton which eats lot of algae →

Biomanipulation
Manipulating ecosystems to change the state of the system
Decreasing fish increased transparency drastically but it didn't last – transparency decreased over ~10 years.

Global phosphorous Pollution

About 1.5×10^9 kg of phosphorus were dumped into freshwater systems

(China contributes the most (30%), then India (8%))

In many areas around the world, either there's not enough water to assimilate the P, or the pollution load is so huge that water can't assimilate everything

Methods of restoring a lake (~ 16 min)

Physical	Chemical	Biological
• Sediment dredging	• Hypolimnetic oxygenation	• Removal of zooplankton eating fish
• Dilution / flushing	• Phosphorous inactivatn	• Stocking predatory fish
• Water level manipulation		• Introduction of mussels
• Hypolimnetic withdrawal		

Refer to video 6.3 for more - pros & cons

Lecture 22

Chapter 8 - Evolution

James Hutton & Charles Lyell - Uniformitarianism (Geologists)

Explanandum (that which needs to be explained) of evolutionary biology

- Tremendous diversity of living organisms, which is not random

In natural world, the diversity is random.

This living diversity has a pattern such that organisms can be grouped in ne

Self-study videos

Brief history of Evolutionary

▲ Early Greeks (4th-5th cent BC

Typological explanation — by God, and the with humans on top — "Scala Naturae"

Each entity on the level has a "Type" or an "essence".

Individuals deviate from this essence & hence we see diversity (# variations in expts are considered "errors") Essentialism — Variation is accidental imperfection

Evidences * direct observation

1. Drug resistance - evolution at small scale
2. Evolution occurs by artificial selection
3. Nat. Selection on variation leads to speciation
   Proof: Ensatina salamanders - Ring species
   * Speciation by artificial select": Hybridisation → Polyploidy
4. Homologous similarity (Pentadactyl limb). Vestigiality
   * Homologies are correlated & heirachically classified
5. Fossil evidence

1. Evolution 2. Transformism (multiple short pts) 3 Creationism

- Jean-Baptiste Lamarck (18th cent) Transformational theory
  - Inheritance of acquired characters
- Charles Darwin (19th cent) Variational theory
  - Evolution through natural selection.

Evolutionary theories — through this, he changes the level of focus/study

Population thinking

- Every species is composed of numerous local population
- Each individual in a population is uniquely different (variation)

Darwin's argument –

1. More offsprings are produced than environment can sustain (Malthus)
2. Individuals vary (Direct observation)
3. Individuals compete for limited resources (Smith) → This isn't needed for evolution
4. Individuals with favorable variation have behind more offsprings (Nat. Sel)
5. Variations are inherited : Descent with modification

Differences in reproductive output could lead to subtle changes in traits which, if accumulated over long periods of time (Lyell), could lead to new species.

Darwin's conceptualisation of species (Morphogenetic species)
For him, the traits of species all exist in a continuum, and its upto a human taxonomist to draw boundaries and create species.

Ernst Mayr : Biological defn : Species is a group of individuals that can viably exchange genes.
Darwin's concept can't explain the origin of these species.

Nutshell : Variation, Heritability, Fitness → Evolution

Natural selection explains the adaptiveness of individuals and the biological diversity.

Requirement : Differential reproduction and heritability. NOT SURVIVAL OF THE FITTEST

* Read up on: • Motoo Kimura - Neutral theory of molecular evolution.
• Wheywell vs. Mill

Synapomorphy - derived form of a trait that's shared by a group of related species *

Consequences —

1. Non-constancy of species (basic theory of evolution)
2. Descent of organisms from common ancestors (branching evolution)
3. Gradualness of evolution (no discontinuities) — Not always true
4. Multiplication of species (origin of diversity)
5. Natural Selection as a mechanism — Not the only one
6. Historical nature of evolution

Other theories —
1. Neo-Lamarckism
2. Orthogenesis (st. line evolution toward fixed goal)
3. Mutationist

Criticism of Darwin's Theory

He didn't give any mechanism for inheritance

Darwin proposed the 'blending theory of inheritance', which was immediately objected to — because then, all traits would converge to mean and no variation would exist.

This was solved by the re-discovery of Mendel's work. (particulate inheritance)

26/11

Lecture 23

Biometrician vs. Mendelian controversy

Mendelian traits were discrete whereas most traits studied exhibited a continuous distribution.

Biometricians — Weldon & Pearson

Mendelian mode of inheritance couldn't account for evolution of such continuous traits.

Mendelian — Hugo de Vries

Continuous traits have less variation, insignificant to evolution.

Resolution — Ronald Aylmer Fisher (After 15-18 yrs)

"Continuous traits can be thought of as the combined effect of many genes"

Modern Synthesis (1950 -1955) RA Fisher, JBS Haldane, Sewall Wright
↳ mathematical model

Major tenets of evolutionary synthesis —

1. Populations contain genetic variation that arises by mutation and recombination (+ horizontal gene transfer)

2. Populations evolve by changes in gene frequency brought about by genetic drift, gene flow & natural selection
⇒ one-to-one relation b/w gene frequency and phenotypic distribution. but this is not true.

3. Most adaptive genetic variants have individually slight (Not necessarily) phenotypic effects so that phenotypic changes are gradual

4. Diversification comes about by speciation which normally entails the gradual evolution of reproductive isolation among populations. → not logical, more faith.

5. These processes, continued for sufficiently long, give rise to changes of such great magnitude as to warrant the designation of higher taxonomic levels. – not necessarily

Fukuyama

Since then, we've made great leaps in understanding mol bio, developmental biology, environment etc

Rethinking the Theory of Evolution – Extended Evolutionary Synthesis

Combining the insights from last 150 years and editing the tenets, while maintaining the structure

No paradigm shift, at least not yet

1. The Selfish Gene by Richard Dawkins
* Genes are the fundamental entity upon which evolution acts – genes use the body & phenotypic features as vehicles to pass from one generation to next.

MES 1

* Concept : Features of an organism are entirely determined by its genes.

Implication : Environment acts just as an agent of natural selection.

LECTURE 24 27/11

Epigenome : Shapes the physical structure of the genome
Condenses inactive gene
Its affected by signals from the external environment

Its the complete description of chemical modifications to DNA & histone that regulates gene expression without actually changing the ATGC sequence

Example trait

Maternal rat care — mothers lick and groom their pups

If the pup is raised by an anxious, low-nurturing mother, it becomes an anxious adult.

But when raised by relaxed, nurturing mothers, they become relaxed adults (normal behaviour when left in an arena & when drowned).

→ How do we know this is not genetic?

	Biological Mothers	
	R	A
Foster Mothers R	R	R
A	A	A

So, the personality is not determined by genes

→ How does it work?

The Hippocampus has Glucocorticoid Receptors (GR) on the surface of the brain

When mammals are anxious, adrenaline - noradrenaline regulates the fight-flight response. But if they're not removed from the bloodstream, the animal will be perpetually anxious.

The GR take down these hormones & keeps it inside hippocampus

⇒ More GR → Less anxious

At birth, the promoter of hippocampal GR gene is highly methylated. The nurturing (in first 7-10 days) highly reduces the methylation, so the pups are less anxious

→ This applies to humans as well

They studied 3 groups of people -

Control | Suicide abused | Suicide non-abused

* Humans with abused childhoods show less GR mRNA & GR

They also show more methylation of promoter for GR gene.

→ Romanian babies

In 1980s, Romania was ruled by a dictator who banned abortions to build a large workshops. So, many babies were abandoned in orphanages where they weren't well cared for.

In 1989, when the dictator was shot, many people adopted these abandoned babies.

Almost without exception, when they grew up, they had personality disorders and couldn't form emotional bonds.

EES ①

So, in Extended Evolutionary Synthesis –

Concept: At least some features of an organism are determined by an interaction of the gene and the epigenome

Implications: Environment is not a mere agent of selection, but can also directly alter the epigenome & hence the features of the organism

But there's a problem with this –

We know that acquired characters are not inherited, as proved by August Weismann (19th generation of mice still had a tail).

↳ Change in thinking of Niche Construction

MES ②

Concept: Acquired traits cannot be inherited

Implication: Adaptive evolution occurs through the differential survival of competing genes

Transgenerational Epigenetic Inheritance

Generally, during gametogenesis, the eg. epigenome is stripped. This is called epigenetic reprogramming. But its not infallible

Example –

1. When a pregnant female rat is zapped with a drug called Vinclozolin, altered methylation pattern is observed.
The rat pups born have testes abnormality, prostate disease, kidney disease, immune abnormality & tumor development.
All the males in future generation (at least 4) also show the same diseased phenotype

2. Toadflax (Linaria vulgaris)

- The flowers of this plant was known to have 2 kinds of phenotypes - forma peloria (pentagonal radial symmetry) & forma typica (bilateral symmetry)
- It was assumed that this was due to 2 kinds of alleles, peloria was thought to be a mutant.
- But after sequencing, we know: no difference at genetic level Lcyc gene is heavily methylated in peloric form, & it is stably inheritable.
- Also, it was discovered that increasing methylation gradually on the Lcyc gene will form semi-peloric flowers with extra spurs of increasing length

LES (2)

Concept: Some acquired traits can be inherited
Implication: Adaptive evolution can potentially occur at the level of the 4 systems of inheritance - genome and the epigenome
1. Genetic one
2. Epigenetic one
3. Behavioral one
4. Symbolic one (limited to humans)

MES (3)

Concept: All genetic information is passed within the same species
Implication: Descent (with modification) forming a lineage - Tree of Life"

But - 3 Horizontal Gene Transfers

Elysia Chlorotica sea

* It is a kind of slug that is the only photosynthetic animal. They're not born this way - the juveniles eat a kind of algae called Vaucheria litorea.
* It doesn't digest the whole thing but retains the chloroplast One it has accumulated enough, it stops feeding & subsists on carbohydrates synthesized.
* The plastids require several nuclear proteins to function and it was shown that 52 genes of Vaucheria had been incorporated into the genome of the slug.

Update: Recent evidence suggests HGT hasn't ~~happen~~ happened and that Vaucheria plastids are autonomous i.e. they don't require nuclear proteins.

Bdelloid rotifers

They have been reproducing parthenogenetically for the last ~80 million years.

This should have significantly decreased their fitness.

~8-9% of their genes have come from other species, accounting for 39% of enzymes in identified pathways

Update: Cross contamination explains the results the group got. so its not entirely... true.

HGT is very relevant when it comes to microbes, especially in the context of antibiotic resistance

EES (2)

Concept: Genetic information can be passed across species

Implication: Descent (with modification) but lineage is not well defined.

Tree of life is now a Reticulate pattern of life

Implications for process of evolution

Features	Genetic changes	Extra-chromosomal change
Pace of occurance	Slow	Can be fast
Stability	Relatively static	Much more dynamic
Multiplicity	Low probability of multiple occurrences	Can occur in multiple individuals simultaneously

Epigenetic changes and HGT —

- greatly increase the amount of variation
- complicate patterns of inheritance
- makes evolution a lot more dynamic than portrayed by MES
- potential practical implication in drug resistant bacteria, invasive species. etc

But ultimately, they don't impact the existing theory of evolution in any ground-breaking way.

* Evolutionary stasis - non-random origination of evolutionary novelties in time & space

* Macroevolutionary role of phenotypic plasticity via - genetic accumulation & phenotypic accomodation

Emergent properties of biological system provides additional push to generate potentially-adaptive, complex phenotypes

Lecture 23A. 12/12/2020

Chapter 9 - Selection

Natural Selection is the differential survival and reproduction of individuals due to differences in phenotype

Selection is a very strong force of microevolution that explains adaptation.

Distinction b/w artificial and natural selection just persists in literature to classify selection due to nature vs. humans. There's no difference in the mode they operate.

"# The Beak of the Finch" by Jonathan Weiner *

Video 1 - Action of selection on short & long time scales
The Galapagos finches (~ 3:51 min)

Peter and Rosemary Grant - since 1973 - studied Galapagos Finches on Daphne Major (relatively small)

* Galapagos - volcanic islands ~ <5 million years old.
13 species of finches

* Islands are different in size, topography and height - trees grow at higher elevation while lower islands are covered by cactus, shrubs and grass.

* Warbler finch - needle-like beak - insects
Woodpecker finch - beetle and termite larvae
Cactus finch - probes into cactus flowers
Ground finches - small, medium & large

* Through DNA sequencing, we can say that all these finches have come from the same ancestor species.

* They tracked over a thousand birds by tagging and documenting them.

* In 1977, a drought occured - no rain for 18 months - only cacti & leafless trees survived - no small seeds, only big hard ones remained ⇒ 80% of medium ground finches died

The birds with larger beaks survived more - in a single generation, the average beak depth had increased

* In 1983 a very strong El Niño brought 10 times more rain - and island was overrun with vines (with small seed) that covered even cactus. When drought struck 2 years later, large seeds were scarce and small ones abundant ⇒ Selection favoured smaller beak.

* Speciation occurs through isolation (geographic or sexual)
What kept these finches from mating?
The songs and appearance of finches differs significantly and males of a certain species recognize females of same species through this.
So changes in these traits set stage for speciation.

⇒ Video 2 - Origin of a species
A new species arose through hybridisation between an immigrant large male of Conirostris from Española island (~100 km away) and the resident Fortis females.
The offsprings learnt their father's unusual song and had a relatively large beak wrt their body (⇒ ate a variety of foods). So, they became sexually isolated, bred within themselves and started following becoming their own species.

In a nutshell - Theory of evolution by Natural Selection -

IF:
1. ∃ variation within populations
2. Part of observed variation is heritable
3. Some individuals are more successful in reproduction (fitness) than others
4. Variation influences fitness

THEN: Those with most favorable phenotypes are "naturally" selected

Each of the 4 postulates & their consequences can be verified independently.

Case Study: Darwin's finches
Galapagos islands - natural laboratory
- Replica populations relatively isolated on different islands
- Smaller islands ⇒ easy to census
- Survival, fitness, morphology can be measured on site

50 C

Galapagos – 800 -1100 km west of Ecuador,
45 islands, islets and rocks
0.7 ~ 5 mya old

14 species of birds descended from a single flock that migrated 2-3 mya from mainland. Great diversity exists.

Prerequisites for selection

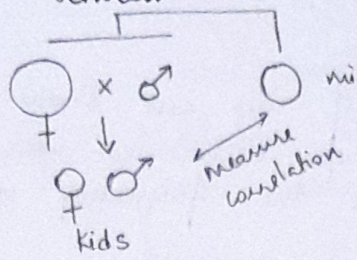
Beak size is important to finches in foraging

Phenotypic variation in G. fortis – Beak depth

In 1976 (with 751 individuals), there was a good distribution of beak size.

Is this variation heritable? How to measure it?

Similarity between relatives –

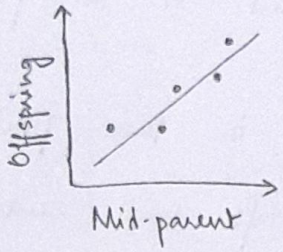


Variation in beak size could be due to –

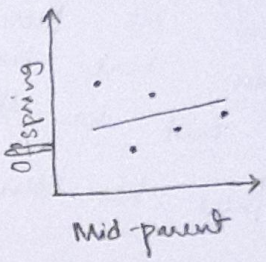
- Amt. of food received as chicks
- injuries or abrasion against hard seeds or rocks could also affect adult beak size and shape

The relation (measured as slope of regression line) between parents and offspring is a measure of heritability.

STRONG HERITABILITY



WEAK HERITABILITY



Greater the heritability, stronger the inheritance component

Thumb rule: If heritability (h^2) < 0.2, then it doesn't respond strongly to selection, but if its $h^2 > 0.2$, then it responds strongly

When measured and plotted, beak depth in finches is heritable.

3. Is there variation in terms of survival or reproduction?

Study on Daphne Mejos in 1977 – big drought where < 20% of rainfall ⇒ plants didn't flower ⇒ no seeds ⇒ birds died en masse

HERITABILITY – fraction of population that is due to differences in genes

Complications – 1. Misidentified paternity
2. Conspecific nest parasitism
3. Shared environment
4. Maternal effects.

In 1975-76, $N = 1400$, but in 1977, $N = 200$ 50 P
This is due to seed abundance in those years —
$S = 10\ g\ m^{-2}$ $S = 3\ g\ m^{-2}$

⇒ Agent of selection: Seed availability
The characteristic of available also changed — just before the drought, most seeds were soft & small.
But as the drought progressed, they were all eaten and most available seeds were large and hard.

The survivorship of finches in 1978 was related to resource availability — birds with larger beak survived & those with smaller beak perished.

∴ All 4 postulates of Darwin are met.

Did the population evolve?
The answer is affirmative if we can show that the selected trait i.e large beak size was passed on to the next generation ⇒ the frequency of the trait changed.
If we look at the distribution of beak size in the generation born 1976 (before) and 1978 (after), then we can see there's a distinct shift in distribution — the mean beak size has increased & the frequency of the trait has shifted

∴ There has been microevolution in these finches

Also, moist years produce more soft, small seeds ⇒ selection favours smaller beaks.

Bigger isn't always better
- Maybe poorer at manipulating smaller objects
- Energetically expensive to produce
- Affects vocal communication, other aspects of mate choice
- Genetically correlated with other traits also under selection?

50 E

The beak size over 3 decades has varied considerably depending on resource availability, with amazing resolution due to Grants' work.

Since this resolution of trait distribution is rarely available, the MES describes evolution as a gradual change that occurs over long period of time.

This needn't be true. Microevolution has occured in birds in 1-2 years, as seen, and in microbes it can occur in 15 - 30 days!

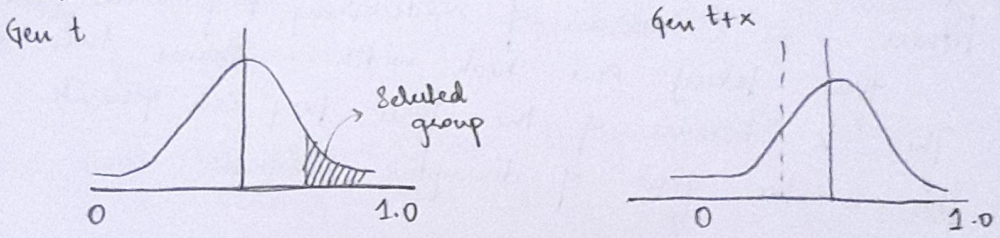
But when people think of macroevolutionary changes, their intuition based on fossil data, then they tend to think of change over very long, coarser time scales.

Insights -

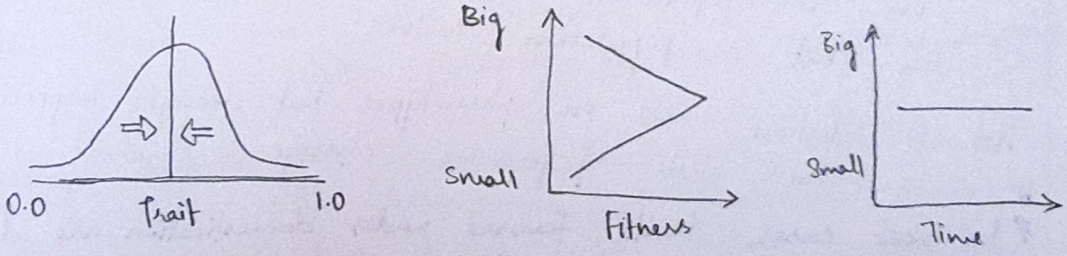
* Microevolution can occur rapidly and dramatically
* Sources / agents of selection often shift, so evolution also reverses itself or takes new directions
* The see-saw of selection in changing environments may give the impression of stately gradual change

Nature of Selection

1. Directional selection



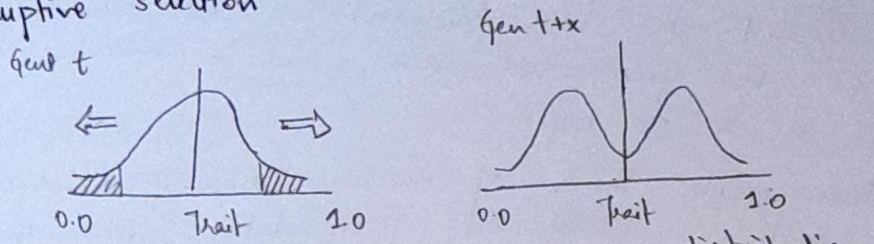
2. Stabilising selection



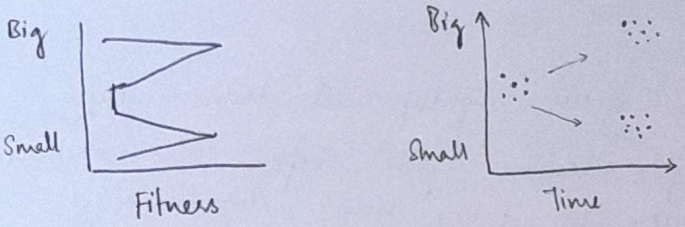
Eg: Traits related to physiology like body weight

In stabilising selection, the extremes are rejected and the moderate trait is selected. So, although selection is operating, we don't see trait value changing in any direction.
This is the most common kind of selection.

3. Disruptive selection



Unimodal distribution → Bimodal distribution



This kind of selection is quite rare
Eg: Black-bellied seedcrackers - African finch
The polymorphism observed - large beak & small beak is an example of disruptive selection
Their beak size is the only trait to have bimodal distribution instead of a bell curve one
Fitness - as a measure of seedcracking performance [inverse of time taken] and beak width - shows twin peaks.
This is because of the force they can generate
This is the agent of disruptive selection

→ * Natural selection acts on individuals, but its effects are seen on the population - an individual either survives or dies but a population evolves.

* Natural selection acts on phenotypes but changes happen only when allele frequencies change [i.e. evolution]

In most cases, traits favored under domestication are deletirious / disastrous in the wild.

* Natural Selection is always behind
- NS changes gene frequencies in population after exposure to selection
- Selection can't anticipate future changes in environment
  i.e evolution is not forward-thinking; new mutations donot arise to solve problems.

* Selection is not "progressive"
  It improves fitness along certain axis but it doesn't mean it improves the organism

Although organisms have become more specialised & complex, this doesn't imply some ultimate goal.

Organisms 'devolve' as often as they evolve — eg: Parasitism

Adaptation occurs through character gain, loss & modification.
↳ Eg: Panda's "thumb"

* Refer next page *

* Selection acts on pre-existing variation
  So, often tools at hand are less than perfect solutions to the problem.

Evolution leads to wherever it has to — no grand design, no concept of "perfection"

Eg: Males of Gambusia affinis sport gonopodia to attract females — but it hinders escape.
If the population is in a no-predation area, the males focus on getting the gonopodia bigger. But in a high predation population, the size of gonopodia has to balanced b/w attracting mates & avoiding predators

* New traits can evolve, even though natural selection acts on existing traits
  Eg: 1. % of oil content in corn kernels — the mean trait value went up by 4 times in ~100 generations.
  2. Insectivorous plants

Exaptation - a trait that's used in a novel way
Herbivory deterrant → trap capture
↳ happenstance

a) New traits can evolve through mutation & if there's a positive selection pressure on them, they are selected

b) Through recombination — during this process, genes get switched b/w sister chromosomes i.e. genes are coming in combinations that didn't exist before — Epistatic Variation

Along with selection, this ensures that more variants are formed that can be selected for.

* Panda's thumb

Panda is one of the non-primate mammals that can hold and manipulate items in their hands.
This is because all 5 of their fingers face in one direction and a wrist bone has modified into a 'thumb'.
Characters that are co-opted for new purposes are called preadaptations.

Chapter 7.1 Conservation Biology

99% of all species that ever existed are now extinct. But why should we worry?
Because human beings have a disproportionate impact on nature — contemporary rates of extinction are vastly greater (100-1000 times) than they are typical of geological past, & projected to get worse

In western world, John Muir - American naturalist (1838-1914) gave - "Romantic-Transcendental Preservation Ethic"
Assumes that communion with nature brings one closer to god, so its a moral duty to preserve wilderness.
Petitioned US Congress & instrumental in passing National Parks Bill (1899) which led to Yosemite and Sequia National Parks.

In contrast - Gifford Pinchot (1865-1946) - 1st chief of US Forest Service
- he was a protégé of Dietrich Brandis - 1st inspector general of forests in India.
- for him, conservation = art of producing from forest whatever it can yeild for the service of man.
- called for wise, judicious use of resources so future generation wouldn't be deprived

Aldo Leopold (1887-1948), American naturalist
He takes a more neutral stance: preservation of biotic communities is important. At same time, humans play an important role in earth, so we can utilise resource as long as we respect the community & its members.

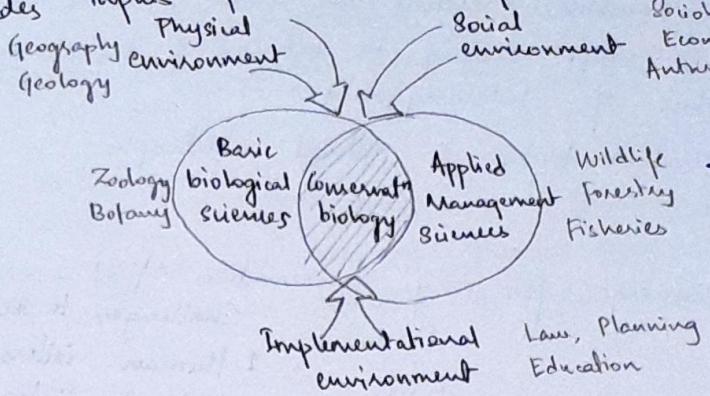
Resource Conservation Ethic
Evolutionary Ecological Land Ethic

Logistical considerations - degree of threat, current conditions, feasibility.

Reserve selection - to protect largest no. of representative species. Then gap analysis.

Conservation biologists study small and/or declining populations & their environment & try to suggest measures to protect them in nature. but through a coarse filter approach. i.e preserving ecosystems will preserve most species.

Includes inputs from other disciplines —



Geographic Information System
- assimilates many layers of information into composite maps.

Vulnerability of species to extinction : Seven forms of Rarity.

L: large pop. size
S: small pop size

Tolerance	Range of distribution: Extensive	Range of distribution: Restricted
Broad	L - Sparrow, Dandelion S - Tiger	L - Galapagos medium ground finch S - Tasmanian devil
Narrow	L - California Gray whale S - Northern spotted owl	L - Fish crow S - Gorilla

Examples

⇑ Rabinowitz rarity classification

Examples —

1. Homing pigeons : Extensive range, ~~bro~~ large population, narrow tole
- It was a very abundant species — the settlers hunted it for food initially
- They were hunted in hundreds of thousands to feed th growing population in Eastern cities
- By 1860s, their numbers began to dwindle. In 1880s, they were only found abundantly in Michigan — but they were hunted ceaselessly there too
- Last female died in Cincinatti zoo in 1914.

Tigers - Extensive range, broad tolerance, small population [50]
Small populations are more vulnerable from 3 kinds of stochasticities -

- Demographic : Random variation in birth & death rate, sex ratio etc
- Genetic : Genetic drift leading to inbreeding depression and loss of heterozygosity - reduces fecundity & ability to cope with stress.
- Environmental - stochasticity & natural catastrophe

Four things converge for any conservation effort -
Science
Common sense
Economics
Politics

Challenges to reserve management
1. Human visitors
2. Natural disturbances
3. Water regimes
4. Invasive exotics & overabundant natives

21/12

Lecture 7.2
Case study : Project Tiger
Estimated no. of tigers at beginning of 20th cent : 40-45 thousand
In India, in 1972, N = 1827

Reasons :
- Hunting
- Poaching
- Competition with local villagers & domestic animals.
- Habitat destruction
- Loss of prey

On 1st April 1973, GOI launched Project Tiger. Its aims -
- To ensure maintainance of a viable population of tigers in India for scientific, economic -- ecological values
- To preserve areas of such biological importance as a national heritage for everyone

BBC Video
- 28 reserves were created for tigers, patrolled by ~4,000 guards

Selected areas' criteria -
- little disturbance
- high development potential in terms of no. of prey & predators
- no drilling, mining or timber-harvesting
- were in different states so they wouldn't be a burden

50K

First 9 tiger reserves covered ~ 13,000 km²

Activities – establishment of core/buffer area
connecting habitats through corridors
halting cattle activities & harvesting of forest products
regular patrolling to discourage poachers

Problems with Project Tiger

Video 2

The numbers doubled in ~~20~~ 11 years. The numbers were counted through pugmarks – tracing & plaster casting them

Sariska reported no loss of tigers, despite no sightings
In 2004, census reported 8 tigers missing. No help.

Basic weakness in counting methods
Sariska claimed to have 18 tigers, as did state govt. and Project Tiger. But Wildlife Institute of India couldn't find a single tiger. They had been poached from a forest reserve with ~300 forest staff.

In Litang Horse Festival in Sichuan, Tibetans wore tiger skin capes, which upset the Dalai Llama. He preached against and managed to create a cultural change i.e. they gave up tigerskin capes.

N > 4000 in 11 years (this figure is questioned) but there was definitely an increase in no. of tigers & many other species.

Major reversals –
- Lack of govt will in face of pressure from farmers
- Ensoachments in buffer zone & access routes
- Reduction in prey animals due to local hunters
- Accelerated poaching
- Lack of funds.

So, even though no. of reserves increased, they still faced these problems & no. of tigers declined again.

Video 3

Govt. team's report said that poachers were killing 3 out of 5 tigers. Counting pugmarks was definitely not a foolproof.

In Sept 2006, Project Tiger was replaced by National Tiger Conservation Authority (NTCA) which was weaker

50 L

In 2018, estimated tigers in India – 2967.

Man-animal conflict

To reduce this, forest department (Sunderban, WB)

- has put up fences around the reserve, which adjoin villages
- has set up Forest Protection Comittees (FPC) in the villages so that they don't harm the forest animals & they're promised alternate means of living

In Corbett –

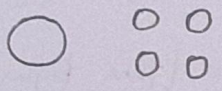
~ 227 tigers in 2010. But due to overcrowding, there's increased man-animal conflict. The govt. is not yes declaring a corridor connecting Corbett & Rajaji National Park as protected area.

~ 90 villages are 2-3 km away from the reserve and people and cattle get killed, so villagers have become less tolerant.

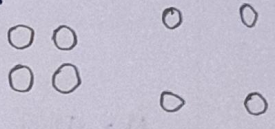
Reserve Design – Jared Diamond (1975)

1. Large > small

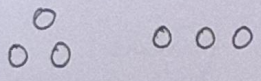
2. 1 L > nS (= Area)



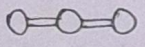
3. If nS, doses > far



4. Clusters > linear



5. Connect with corridor



6. Make reserves as circular as possible (lesser edge)

Case for large reserves – have large population unlikely to become extinct
relatively shorter edge ⇒ less susceptible to invasive species & poachers
less vulnerable to catastrophic events

Reserves should be buffered from most harmful human activities by semi-natural ecosystems Eg. forests ranged for production of trees

Connectivity to ensure – 1. Daily movements 2. Seasonal/Annual migration
3. Dispersal movement 4. Range shifts in response to CC.

Corridors – particularly vulnerable to external disturbances & may facilitate the spread of diseases & exotic species.

Lecture 24 9/12/20

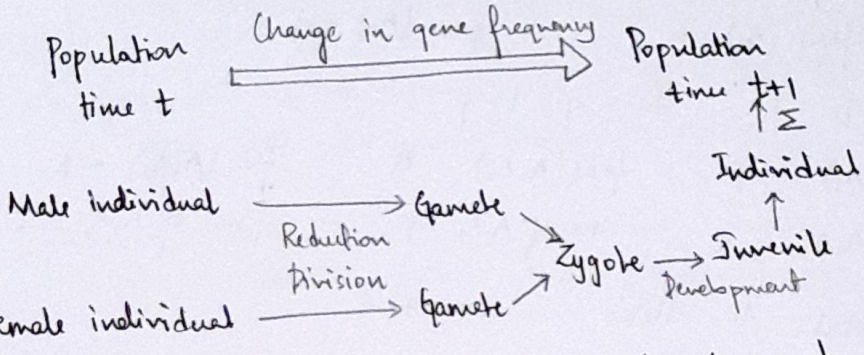
Population genetics : HW Equilibrium.

⇒ Revisiting Mendel –

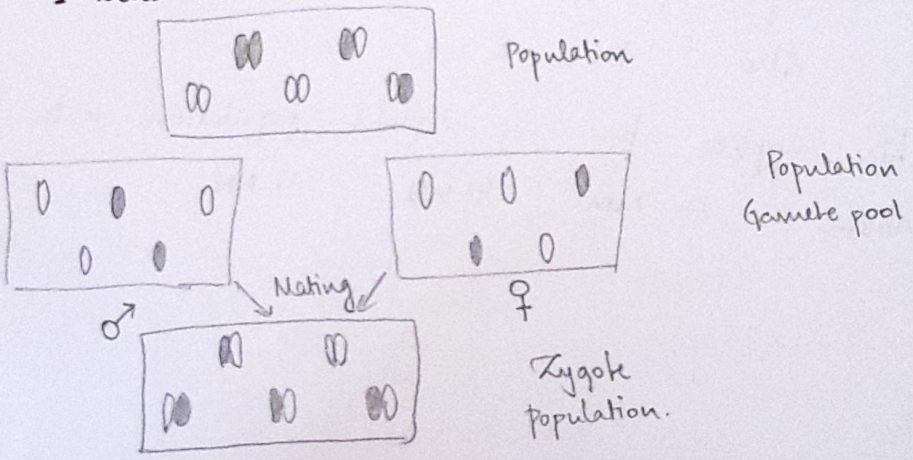
1. Law of segregation
During gametogenesis, each gamete receives only one copy of a gene

2. Law of independent assortment
During gametogenesis, alleles of different genes assort independently
≠ This breaks down when we consider linkage.

Mendel worked with a family, but evolution occurs at the level of population.



Population genetics – extension of Mendel's laws to population
Here, an individual is conceptualised as a pair of alleles of a given gene locus.
i.e 1-locus 2-allele system



With this schematic, we're just tracking the alleles, disregarding many other aspects – morphology, development, environment, development etc.

Mating represented by coming together of gametes
Next generation is formed by sampling from this pool of gametes.

∴ Entire process of transmission of genes over a large no. of mating pairs is abstracted into sampling from a bagful of gametes ("beanbag gametes").

1-locus 2-allele

No. of individuals — N

Alleles — A_1, A_2

Phenotype — A_1A_2, A_1A_1, A_2A_2

freq $(A_1) = p$ freq $(A_2) = q$

$p + q = 1 \Rightarrow q = 1 - p$

freq $(A_1A_1) = P$ freq $(A_2A_2) = Q$ freq $(A_1A_2) = R$

no. of $A_1A_1 = X$ no. of $A_2A_2 = Y$ no. of $A_1A_2 = Z$

Total no. of alleles — $2N$

Total A_1 alleles $= 2X + Z$

$$\text{freq}(A_1) = \frac{2X + Z}{2N} = \frac{X}{N} + \frac{1}{2}\frac{Z}{N} = P + \frac{1}{2}R$$

$$\therefore p = P + \frac{1}{2}R$$

Similarly, $q = Q + \frac{1}{2}R$

Also, $P + Q + R = 1$

This applies for any diploid population with 1 locus, 2 allele

Problem: 83 MM 46 MN 11 NN

$N = 140$

$X = 83$ $Y = 11$ $Z = 46$

$P = \frac{83}{140} = 0.59$ $R = \frac{11}{140} = 0.078$ $Q = 0.328$

$p = P + \frac{1}{2}Q = 0.757$

$q = R + \frac{1}{2}Q = 0.243$

Deriving Hardy Weinberg law

One locus, two alleles – A_1, A_2. $freq(A_1) = p$

Freq (A_1) in parents — p

⇓ No segregation, No mutation, migration, Equal fertility

Freq (A_1) in all gametes — p

⇓ No sampling errors so that the chosen gametes are representative of all gametes ⇒ Large population, equal fertilizing capacity

Freq(A_1) in gametes forming zygotes — p

⇓ Random mating, Equal frequency in males & females

Genotypic frequency among zygotes — $p^2 \cdot 2pq \cdot q^2$

|| Equal viability (No viability selection)

When forming zygotes, alleles are randomly chosen from both male & female gamete pools – independently –

$A_1A_1 - p^2$ $\quad A_1A_2 - 2pq$ $\quad A_2A_2 - q^2$

This is the same as its frequency in the population

⇓

Genotypic frequency among progeny — p^2 $2pq$ q^2

⇓ Equal viability

Frequency of A_1 in progeny — $p' = p$

$p' = p + \frac{1}{2}R$

$= p^2 + \frac{1}{2}(2pq)$

$= p(p+q)$

$\therefore p' = p$

Hardy-Weinberg law – Godfrey Hardy & Wilhelm Weinberg.

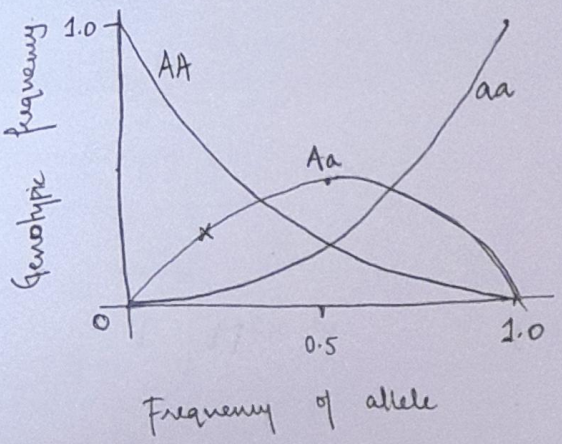
In a large random mating population with no selection, mutation or migration, the gene frequencies and the genotypic frequencies are constant from generation to generation, and there's a simple relationship between gene frequencies and genotypic frequencies.

This is a null law – an ideal case where what's more interesting is to study what happens when & how this law is violated.

Lecture 25

Weinberg published his work in German in 1908.
Pearson's student Udney Yule asked why the dominant phenotype doesn't wipe out the recessive to R.C. Punnet.
Punnet mentioned this to Hardy who figured out the solution and published in Science in 1909.
In 1943, Weinberg's work was rediscovered and "Hardy's Law" was renamed ↓ more thorough.

H-W law is used to determine if the population is in equilibrium. by -
computing gene frequencies
calculate expected genotype frequencies
compare observed no. of homozygotes with expected no.



* Heterozygotes are maximum when both alleles are present equally.
* When genotypic frequency of A/a is less, most copies of A/a are present in heterozygotes so that they're not visible in them
⇒ Eugenics is doomed to fail

* Frequency of heterozygotes cannot be greater than 0.5. in HWE
* Gene & genotype frequencies don't change from generation to generation
* Progeny genotypic frequencies depends only on parental gene frequency. and not genotypic frequency
* Relationship between gene and genotypic frequencies is reached in a single generation (consider gene freq is p in population at $t=0$. ($\approx p + \frac{q}{2}$)
* Neutrally stable equilibrium In $(t+1)$ generation itself, the cond^n $p^2 + 2pq + q^2$ is attained, given all conditions are satisfied

17 min
(?)

One-locus multi-allelic case : $(p+q+\dots n)^2$

H-W law is easily extendable to this case

Problem : $a = 9.6 + \frac{48.3}{2} + \frac{2.8}{2} = 35.15\%$

$b = 34.3 + \frac{48.3+5}{2} = 60.95\%$

$c = 3.9\%$

No cc individuals because, # $(c^2) = 0.0016 \times \underbrace{178}_{N} \approx 0$ people

Why is H-W important?

- Biology proceeds by studying departures from the law
- Asserts that, all being constant, allelic diversity is maintained
  - ~~Stores~~ preserves maintains the variation necessary for evolution
- ⇒ Provided strong ~~theory~~ support for Darwin's theory.
- Expands easily to multiple allele case & higher ploidy level.
- Helps in calculating gene freq. of recessive allele & frequency of carriers in a population

Eg: Phenylketonuria — $1/11,000$ — autosomal recessive gene

$q^2 = \frac{1}{11,000} = 9.09 \times 10^{-5} = 9.5 \times 10^{-3} = 0.0095$ ⇒ $p = 0.9905$

$2pq = 0.0188$ ⇒ ≈207 in 11,000 people are carriers

- Also important in conservation biology — while dealing with inbreeding population

14/12

Happened on 10/12

Lecture 26

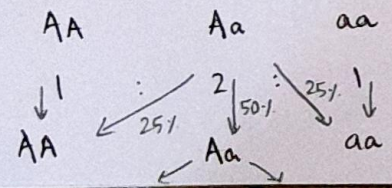
Population Genetics Selection

Counter-example

Consider a population & a trait (1 locus, 2 allele) where the individuals reproduce by selfing.

Let's say we start out with all heterozygotes —

Aa $p = 0.5$ $q = 0.5$



Ultimately, we'll just have AA & aa individuals ⇒ it appears that the populat^n has evolved, but gene freq. is still the same — $p = 0.5$, $q = 0.5$

So the population hasn't evolved throughout this, there's been no selection or mutation. Due to selfing & underlying genetics of the trait, the trait frequency changes.

Effects of Selection — Refer Evolutionary Analysis by Hall

1 locus 2-allele

Viability Selection model — effect of survivorship

	A_1	A_2	
In parental generation —	p	q	$1-q=p$

Genotypic freq before selection	p^2	$2pq$	q^2	Assume that they don't survive equally
Genotypic fitness	w_{11}	w_{12}	w_{22}	let w_i be the rate of survivorship

Post selection $\quad p^2w_{11} \quad 2pqw_{12} \quad q^2w_{22}$ } Sum of these is not necessarily equal to 1

Avg. fitness — $\bar{w} = p^2w_{11} + 2pqw_{12} + q^2w_{22}$

Frequencies after scaling $\quad \frac{p^2w_{11}}{\bar{w}} \quad \frac{2pqw_{12}}{\bar{w}} \quad \frac{q^2w_{22}}{\bar{w}}$ } New frequencies sum to 1

Gene freq in next generation after selection

$$p' = \frac{p^2w_{11} + pqw_{12}}{\bar{w}} \qquad q' = \frac{q^2w_{22} + pqw_{12}}{\bar{w}}$$

$$\boxed{p' = \frac{p}{\bar{w}}\left(pw_{11} + qw_{12}\right)} \qquad q' = \frac{q}{\bar{w}}\left(qw_{22} + pw_{12}\right)$$

Here gene freq (p' & q') are in terms genotype freq. This is because fitness can only be defined on genotype and not directly on the allele

To tackle this, we calculate Marginal allelic fitness — average fitness of every individual who has that particular fitness This will be scaled by the corresponding genotype frequency.

Marginal allelic fitness —

	A_1A_1	A_1A_2
A_1		
w_1^*	pw_{11}	qw_{12}

This is scaled by probability of picking A_1 in the 2nd allele, keeping first allele fixed

$$\Rightarrow \boxed{\begin{aligned} w_1^* &= pw_{11} + qw_{12} \\ w_2^* &= qw_{22} + pw_{12} \end{aligned}}$$

(87)

From this, $\bar{w} = p^2 w_{11} + 2p(1-p) w_{12} + (1-p)^2 w_{22}$

$\Rightarrow \bar{w} = p^2 w_{11} + 2pw_{12} - 2p^2 w_{12} + w_{22} + p^2 w_{22} - 2pw_{22}$ — (*)

$\bar{w} = p^2 w_{11} + 2pq w_{12} + q^2 w_{22}$

$\bar{w} = p(pw_{11} + qw_{12}) + q(pw_{12} + qw_{22})$

$$\boxed{\bar{w} = p w_1^* + q w_2^*}$$

Avg fitness (genotypic) is the scaled sum of marginal allelic fitnesses — scaled by corresponding allelic frequency

So, going back

$p' = \frac{p}{\bar{w}}(pw_{11} + qw_{12})$ $\qquad$ $q' = \frac{q}{\bar{w}}(qw_{22} + pw_{12})$

$q' = \frac{q}{\bar{w}} w_2^*$

$$\boxed{p' = \frac{p}{\bar{w}} w_1^*}$$

$\Delta p = p' - p = \frac{p}{\bar{w}} w_1^* - p\frac{\bar{w}}{\bar{w}} = \frac{p}{\bar{w}}\left(w_1^* - [pw_1^* + qw_2^*]\right)$

$= \frac{p}{\bar{w}}\left(qw_1^* - qw_2^*\right)$

$\boxed{[pw_{11} + qw_{12} - \bar{w}]}$: Avg excess of A_1. If this value is positive (i.e. A_1 carrying individual has higher than avg fitness) then frequency of A_1 will rise

$$\therefore \boxed{\Delta p = \frac{pq}{\bar{w}}\left(w_1^* - w_2^*\right)} \quad — (1)$$

$\Rightarrow$ Change in gene frequency depends on product of gene frequencies and the difference of marginal allelic fitness, which keep changing along with p and q.

Consider Eqn (*). We consider that genotypic fitnesses (w_{11}, w_{12}, w_{22}) are independent of frequency. So —

$\frac{d\bar{w}}{dp} = 2pw_{11} + 2w_{12} - 4pw_{12} + 2pw_{22} - 2w_{22}$

$= 2\left[pw_{11} + \underbrace{w_{12} - pw_{12}}_{(1-p)w_{12}} - pw_{12} + \underbrace{pw_{22} - w_{22}}\right]$

$= 2\left[pw_{11} + qw_{12} - (pw_{12} + qw_{22})\right]$

$= 2\left(w_1^* - w_2^*\right)$

For 1-locus, 2-allele
df = 3 − 2
df = 1

$$\therefore \boxed{w_1^* - w_2^* = \frac{1}{2}\frac{d\bar{w}}{dp}}$$

$$\Delta p = \frac{pq}{2\bar{w}}\cdot\frac{d\bar{w}}{dp} = \frac{pq}{2}\frac{d(\ln\bar{w})}{dp}$$

} Wright's equation for fitness landscape — Sewall Wright.

Critical value of χ^2 for df = 1 is 3.841.
df = 2 : 5.991

χ^2 Test: $\chi^2 = \sum \frac{(Obs - Exp)^2}{Exp}$

df = No. of classes − No. of independent values calculated

$df = k - 1 - m$ → No. of ind. allele freq estimated

$$\Delta p = \frac{pq}{2\bar{w}} \frac{d\bar{w}}{dp}$$ $\Rightarrow$ the sign of Δp is determined by $d\bar{w}/dp$.

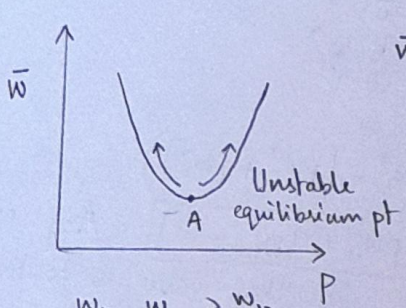
Also, $$\frac{d\bar{w}}{dp} = 2\left[pw_{11} + w_{12} - pw_{12} - pw_{12} - w_{22} + pw_{22}\right]$$

$$\Rightarrow \frac{d^2\bar{w}}{dp^2} = 2\left(w_{11} - 2w_{12} + w_{22}\right)$$

The minima / maxima in $\bar{w}$ vs. p graph is determined by the sign of $d^2\bar{w}/dp^2$.

If $w_{11}, w_{22} > w_{12}$ — Minima

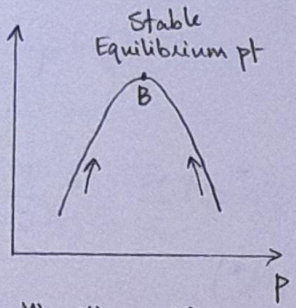
$w_{11}, w_{22} < w_{12}$ — Maxima



$w_{11}, w_{22} > w_{12}$

Underdominance

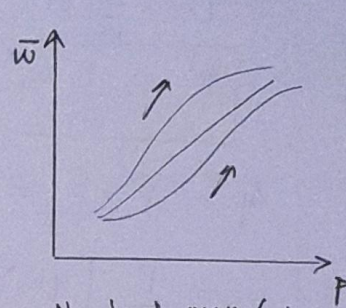
Homozygotes have better fitness than hetero



$w_{11}, w_{22} < w_{12}$

Overdominance

Heterozygotes have better fitness than homozygotes



No local max/min

If the system is at A, B or case 3, $\bar{w}$ always increases, there's nothing else it can do.

This kind of analysis leads to the intuitive understanding that selection always increases population fitness.

16/12

Lecture 27

Earlier, we assumed that genotypic fitnesses are frequency independent. In the frequency dependent case —

$$\frac{d\bar{w}}{dp} = 2\left(w_1^* - w_2^*\right) + p^2\frac{dw_{11}}{dp} + 2pq\frac{dw_{12}}{dp} + q^2\frac{dw_{22}}{dp}$$

Each term is weighted by genotypic frequency $\Rightarrow$ its the expectation of dw/dp

$$\Rightarrow \frac{d\bar{w}}{dp} = 2\left(w_1^* - w_2^*\right) + E\left[\frac{dw_i}{dp}\right]$$

BI 2113 – Extra computations on HWE and selection

1. Combining probabilities
2. Selection

	AA	Aa	aa
Genotypic freq.	p^2	$2pq$	q^2
fitness	w_{11}	w_{12}	w_{22}
Scaled, post select^n frequencies	$\frac{p^2 w_{11}}{\bar{w}}$	$\frac{2pq w_{12}}{\bar{w}}$	$\frac{q^2 w_{22}}{\bar{w}}$

where $\bar{w} = p^2 w_{11} + 2pq w_{12} + q^2 w_{22}$

Gene frequency in next gen

$$p' = \frac{p^2 w_{11} + pq w_{12}}{\bar{w}} \qquad q' = \frac{q^2 w_{22} + pq w_{12}}{\bar{w}}$$

$$\Delta p = \frac{p}{\bar{w}}\left(p w_{11} + q w_{12} - \bar{w}\right) \qquad \Delta q = \frac{q}{\bar{w}}\left(q w_{22} + p w_{12} - \bar{w}\right)$$

Average Excess.

3. χ^2 – Test

$$\chi^2 = \sum \frac{(\text{Observed} - \text{Expected})^2}{\text{Expected}}$$

df = No. of classes – No. of independent values calculated.

* Classes – 3: the genotypes
* Ind. values – Total no. of individuals & value of 'q' (z)

For df = 1, critical value = 3.841
df = 2, critical value = 5.991

4. Selection on recessive allele.

w_{AA}	w_{Aa}	w_{aa}
1	1	$1-s$

s: selection coefficient.

If selection is positive, we can substitute -ve value for s.

$$q' = \frac{q^2 w_{aa} + pq w_{Aa}}{p^2 w_{AA} + 2pq w_{Aa} + q^2 w_{aa}} \Rightarrow q' = \frac{q(1-sq)}{1-sq^2}$$

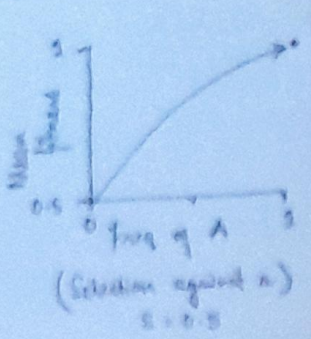
For a lethal recessive a, $s = 1 \Rightarrow q' = \frac{q}{1+q}$

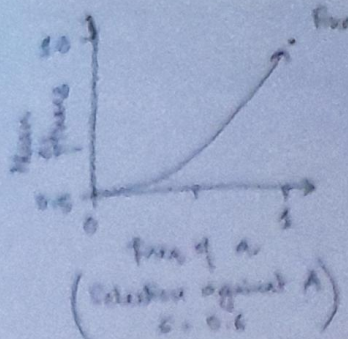
* Selection on dominant allele

w_{AA}	w_{Aa}	w_{aa}
$1-s$	$1-s$	1

$$\Rightarrow p' = \frac{p(1-s)}{1-2sp+sp^2}$$

Selection against a recessive allele is selection in favor of dominant allele & vice-versa.





5. Stable equilibria with heterozygote overdominance (superiority) and unstable equilibria with heterozygote inferiority (underdominance)

→ $w_{12} > w_{11}, w_{22}$: Heterozygotes have increased fitness

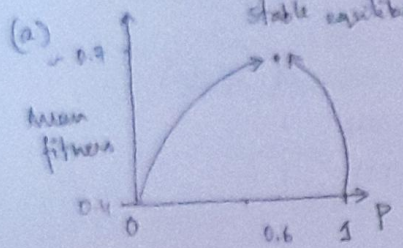
$$\Delta p = \frac{pq}{\bar{w}}\left(w_1^* - w_2^*\right) = \frac{pq}{\bar{w}}\left(w_{11}p + w_{12}q - pqw_{11} - q^2w_{22} - 2pqw_{12}\right)$$

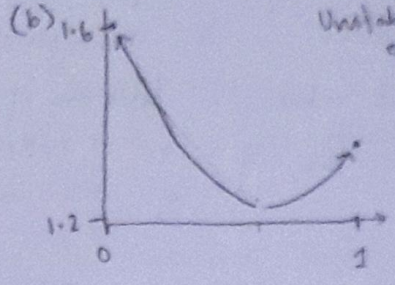
At equilibrium, $\Delta p = 0$.

$$\Rightarrow \hat{p} = \frac{w_{22} - w_{12}}{w_{11} - 2w_{12} + w_{22}}$$

w_{11}	w_{12}	w_{22}
$1-s$	1	$1-t$

$\therefore \hat{p} = \frac{t}{s+t}$ if t, s: positive → overdominance (a)
negative → underdominance (b)





6. Mutation

μ: Mutation rate from A → a (negligent back-mutation)

$p' = p - \mu p$ $\quad q' = q + \mu p$

$\therefore \Delta p = -\mu p \Rightarrow p_n = p_0 e^{-\mu n}$: After n generations

7. Mutation - selection balance for (i) deleterious - recessive

Consider a is deleterious $\Rightarrow w_{11} = 1 \quad w_{12} = 1 \quad w_{22} = 1-s$

After selection : $p^* = \frac{p^2w_{11} + pqw_{12}}{\bar{w}} = \frac{p}{1 - s(1-p)^2}$

After mutation : $p' = (1-\mu)p^* = \frac{(1-\mu)p}{1-s(1-p)^2} = p$ → when select^n & mutat^n balance $\Rightarrow \therefore q = \sqrt{\frac{\mu}{s}}$

(ii) Lethal dominant allele

w_{11}	w_{12}	w_{22}
1	0	0

$\Rightarrow p^* = 1$ ∵ (selection removes all q) $\Rightarrow p' = 1 - \mu = p_2$ (Equilibrium cond^n) $\Rightarrow \therefore q = \mu$

(49)

$$\Rightarrow w_1^* - w_2^* = \frac{1}{2}\left(\frac{d\bar{w}}{dp} - E\left[\frac{dw}{dp}\right]\right)$$

$$\Delta p = \frac{pq}{2\bar{w}}\left(\frac{d\bar{w}}{dp} - E\left[\frac{dw}{dp}\right]\right) \quad \text{from } \text{Eq}^n (1) \text{ in pg 54}$$

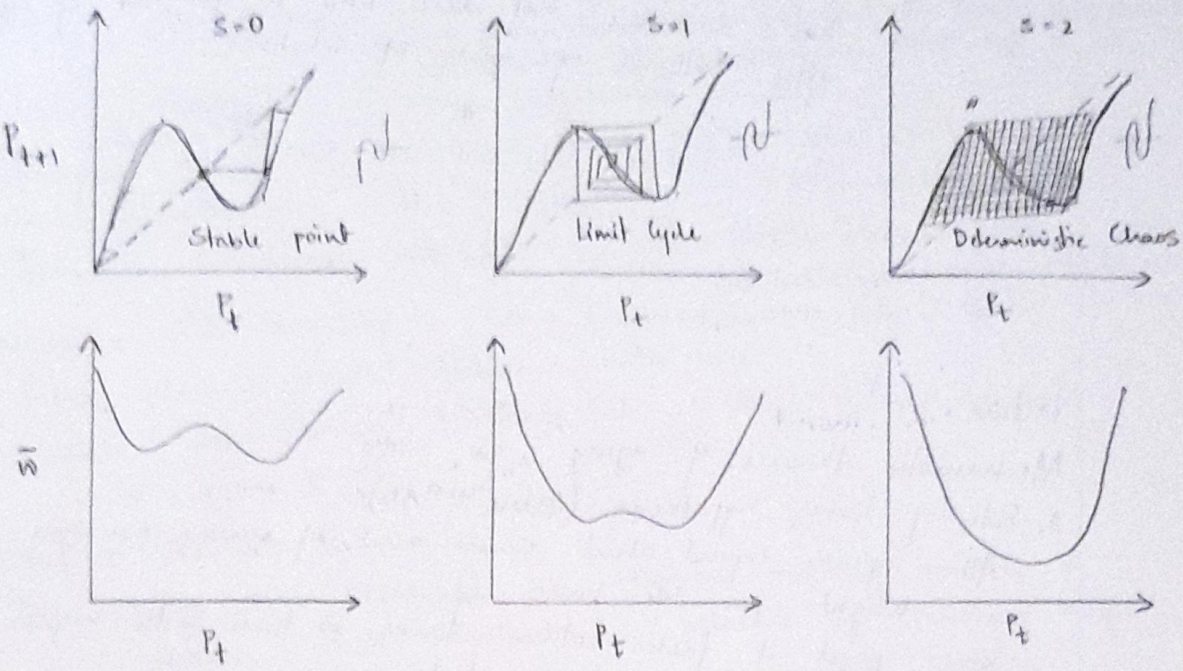
$$\Delta p = \frac{pq}{\bar{w}}(w_1^* - w_2^*)$$

In the independent case, $\Delta p = \frac{pq}{2\bar{w}}\frac{d\bar{w}}{dp}$, where sign of Δp was determined by $d\bar{w}/dp$ & $\bar{w}$ was always increasing

But here, for $\Delta p = 0$, $\frac{d\bar{w}}{dp} = E\left[\frac{dw}{dp}\right]$, which doesn't always happen.

⇒ We won't get an equilibrium, we'll get some bizarre behaviours -

* Not Equal peaks



Cobweb diagrams - commonly used in non-linear dynamics, in situations where one iteration is plotted against previous one

The digonal is the equilibrium line ($P_{t+1} = P_t$)

P_{t+1} vs. P_t is called the First return map.

Tracing the lines of cobweb diagram gives us the time evolution of the system

We can figure out the dynamics directly, without iterating it.

Here, selection doesn't always increase fitness; it may lead to decrease in fitness and even extinction, called Darwinian extinction.

Darwinian selection only works for a narrow set of conditions

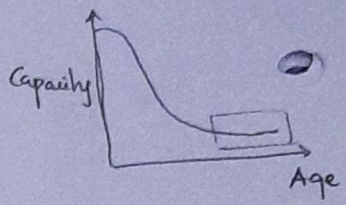
All else being equal, aging decreases an individual's fitness

Aging

There is an age-dependent decline* of many anatomical and physiological factors (like nerve conduction, renal flow etc) cardiac index, work capacity, accommodat"
Including importantly — • fertility
• survivorship

Aging is a big deal psychologically, medical & economically. But scientifically—

Why do organisms age?
Natural selection should increase the fitness of organisms but aging definitely reduces fitness
So why has evolution not been able to get rid of aging after millions of years of selection?



* All factors don't decrease steadily, but after certain years, the ability plateaus.
This is called late-life plateau

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Lecture

Mechanistic theories of aging

1. Rate of living hypothesis (Galen 2nd AD)
All species expend about same amt of energy per gm body weight per life time
Some spend it faster, others slower ⇒ those with higher metabolic should die faster

Testing: Austad & Fischer, 1991
- Amt of energy spent per gram of 164 species of 14 mammalian orders — bats have highest metabolic rate but also live 3 times longer and marsupials have lowest metabolism but also lowest longevity
- Also: birds live longer than mammals of same size, both are endotherms
ectotherms don't live longer than endotherms.

2. Wear and tear theory
Life is a "machine" and over time, it breaks down after some long time

Here, aging is caused by accumulation of damages to cells and tissues in form of -

- Errors during replication, transcription & translation
- poisonous metabolic by-products like Reactive Oxygen Species (ROS)

Most organisms are capable of resisting or repairing such damages (Eg: Proof reading DNA, superoxide dismutase etc)

But its suggested that due to selection, organisms have reached the maximum biological limit of error correction (ie no genetic variation) & this is the residual error even after the correction processes

One of the examples are — oxidative damage, sensor kinase enzymes activate p53 — a Tx factor that put the cell into a permanently non-dividing state, known as senescence — or induce programmed cell death.

Telomere shortening

Telomeres are long sequences of repetetive DNA at the ends of chromosomes. At every replication, part of DNA is lost.

This is associated with aging p53 — too little → cancer risk, stem too much → adversely affects cells

Either way, early — death.

Telomeres and longevity in Zebra finches

Mean telomere length declines with age

Finches with longer telomeres at 25 days of age tended to live longer

Joeng et al. 2004

This group over expressed at telomere-binding protein in C. elegans that resulted in increase in telomere length.

This increased their longevity, than corresponding controls

But this doesn't seem to hold true over all species

Longer-lived mammals have shorter telomeres — controlling for shared evolutionary history, mammals show a significant inverse relation (Gomes et al 2011) ⇒ long-lived mammals tend to have shorter telomeres

No correlation b/w telomere length and longevity in other strains of C. elegans.

In mice, wild derived strains with shorter telomeres show similar longevity as compared to laboratory strains

Relationship between aging & telomere shortening is complex at best and non-existant at worst.

Evolution of endothermy in mammals would have elevated mutation rate - suppressed telomerase activity counters this

→ What if wear and tear is happening through some other mechanism and we have maximised the error correction rate through selection?

⇒ Longevity of each group is at its highest and cannot increase

Then organisms shouldn't respond to selection for longer lifespan. We can check this hypothesis by doing an experiment to select for longevity and see if lifespan increases -

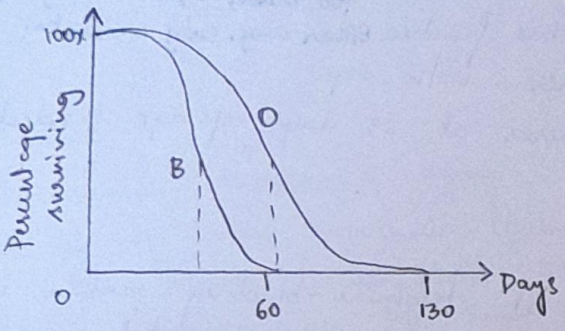
• Rose, 1984, Luckinbill 1984

Artificial selection for evolution of longevity through selection on late-life fecundity -

Two kinds of drosophila populations -

"B" population - reproduces at 4 days of age ⇒ early fertility selection

"O" population - reproduces at later stages ~ 70 days ⇒ late fertility selection



⇒ Populations can respond to selection for increased longevity indicating that longevity hasn't been maximised and genetic variation for the trait still exists

The amount of selection we can do is also astounding - increase the mean significantly

Wear and tear theory doesn't look generalizable

Some believe that its all in the genes.

Hundreds of genes have been implicated in aging but no single set of genes implicated for aging across data.

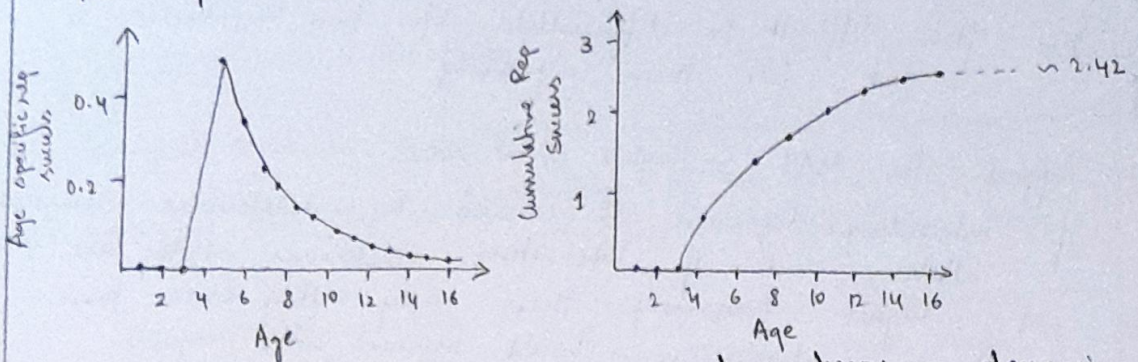
Lecture

Evolutionary Theories of Aging

They say that aging is an inevitable outcome of the way natural selection operates.

Reproduction is contingent upon survivorship
Only those individuals that survive to a certain

Consider a stable cohort table with 15 age groups and equal fraction (0.8) survives each generation. After certain age, reproductive success is also the same till the end
i.e. no aging wrt fecundity.
The age specific reproductive success keeps declining ($l_x \cdot m_x$)



Relative contribution of given age class keeps on decreasing with age ⇒ effect of natural selection goes down with age
i.e. the effect of Age 8 dying would be much greater than that of Age 16

If there is a late-acting deleterious mutation —
The surviving fraction is 0 at age 14 & 15 ⇒ expected life time reproductive success goes to 2.34 from 2.42.

01. Mutation accumulation theory (Peter Medawar, 1952)
In a population mutations which express later in life face a weaker selection ⇒ they accumulate more easily.
i.e mutations with strictly age-specific expression accumulate more readily in the population as force of natural selection declines
The effect of these late-acting deleterious mutations is what shows up as aging.

Example ← Hereditary nonpolyposis colon cancer - caused by mutation in germ line that codes for enzymes that repair DNA mismatch

02. Antagonistic Pleiotropy (GC Williams)
Genes have impact at multiple ages. As one grows, some mutations have negative effects at late-life i.e antagonistic genes.
→ an individual
but good in early life.

Eg: mutation that causes less energy to be allocated to repair and more to reproduction in early life ⇒

Mutations that increase early fitness are selected even if they negatively affect late-life fitness

Trade off between early fertility and later fertility / survival

In the cohort table, consider that Age 12-15 die but reproduction begins at age 2. — Expected lifetime RS increases from 2.42 to 2.86.

It's often difficult to differentiate b/w two hypotheis & test for them seperately

Support for MAT — Hughes et al 2002

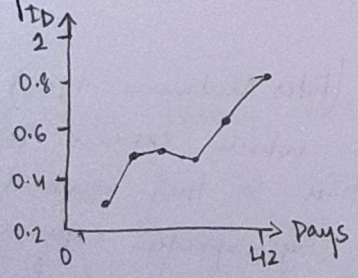
If inbreeding depresion is caused by deleterious recessive alleles, and if late-acting deleterious alleles are maintained at higher frequency than early acting ones, then inbreeding depression should increase with age.

They took 10 inbred populations & made all possible crosses i.e 90 outbred and 10 inbred progeny population

Inbreeding depression is computed through age-specific fecundity

ie. $I.D = \frac{fec(outbred) - fec(inbred)}{fec(outbred)}$

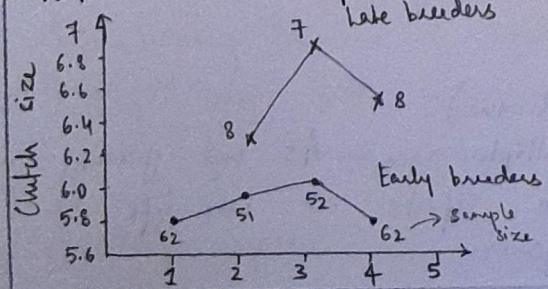
They're trying to see if fecundity of inbred population is going down at faster rate with age



Inbreeding depression increases with age thus supporting MA Theory

⇒ Support for Antagonistic Pleiotropy

a) With collared flycatchers — Gustaffson & Pärt (1990)



Early breeders pay a cost of having a lower clutch size (no. of eggs) by about 1.2 eggs on avg.

In spite of that, early breeders have a greater life-time RS due to their numbers.

(65) b) Back to O & B populations — fig 6.

As the O population is selected for longevity, they have two-three times the lifespan.

Prediction: trade off b/w early life & late life fecundity

Data is consistent with hypothesis - B has greater fecundity in early life and O has greater fecundity in late life

Effects of Extrinsic mortality

↓ Ecological mortality ⇒ ↑ selection on deleterious, late-acting mutations

↑ Ecological mortality ⇒ ↓ selection of late-acting antagonistic action of late-acting pleiotropic genes

→ These populations live longer → spread out reproduction over their life so, if a gene causes early death, it will be eliminated faster ⇒ increased selection on late acting mutations

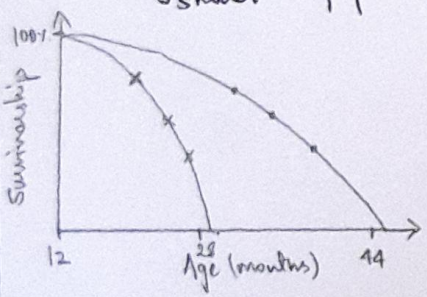
→ They need to reproduce fast ∵ they die young. What happens at old age has lesser effect

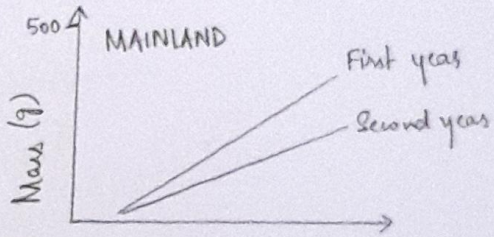
Hypothesis: Populations with low ecological mortality should show delayed senescence

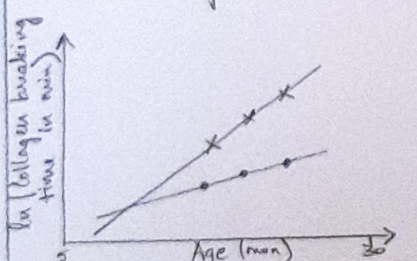
Austad (1993)

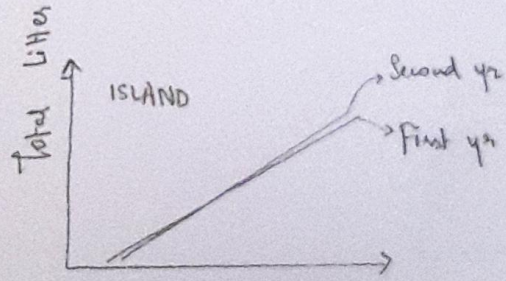
Worked with 2 populations of Virginia opossum (marsupials)

- Mainland population (high rate of predation)
- Island population (low rate of predation)









- Island population had higher survivorship percentage, their fecundity remained constant (proxy litter mass) while mainland populations decreased in both cases.
- Collagen hardens when an individual ages i.e. one of the characteristics. Greater breaking time ⇒ mainland populations age faster than island ones

Conclusion

Organisms age because selection largely overlooks those who live to late life (mutation accumulation) and/or, because it has favoured individuals that seek early benefits even at ~~cost~~ expense of late-life costs (antagonistic pleiotropy).

Two hypotheses are not mutually exclusive. We can find support for both, across wide-spread taxa (than for mechanistic theories), but these theories are much harder to check for