I) Fitch Intro
- GG Simpson - Tempo & Mode in Evolution

II) Nancy Mazels

Lynn Margulis wrote - "It's a good book if you only look at the pictures"

- All cells share essentially the same biochemistry

- tRNA & replication

- RNA world
  - catalytic RNA - suggests a solution to chicken & egg problem (Cech Altman Pace)

smallest catalytic unit { UUU / GAAACp } Kazakov & Altman

- possible problem - RNA doesn't show up in Miller-Urey experiments

* - suggests these are conserved because of the # of interactions

- replication
  - problems ① loss of ends ② specificity
  - solution to ① telomeres - circular genome - circularize for replication
    T4 - terminal redundancy - primer protein

like tRNA

phage QB -

- ppp G ——— replicase subunit II CCA-OH

QB Replicase

phage pol - (II)(Tu)
ribosome (S1)(Ts) } Elongation Factors

Suggests that some Viruses are old

- Genomic Tag model
  - 3' terminal tRNA-like structures
    substrates for replicases
  - present in lots of viral genomes

- Suggests that tRNAs have two domains

- Alarming - mt plasmids in N. crassa

dsDNA — CCACCA / GGTGGT — rolling Circle tx

site specific cleavage

Variant Retroplasmid

rev. tcase — CCACCA

pU

rev. tcn

CCACCA

GGTGGT

Retroviruses

RNA — tRNA primer → DNA

Cauliflower mosaic virus

tRNA prmr to make cDNA

Telomerase

- RNA component is a built in template for adding GGGTT to end

- - AACCCCAA
- - TTGGGGTTGGGGTT -
cDNA

reverse trsc

Polymerases

How did tRNA get into protein synthesis

- a charged tRNA (say basic aa with a coat) better interact w/ RNA

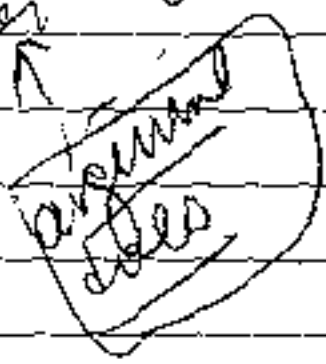
- suggest that early prot. synthesis could have been to better make such changes

- RNAs can recognize aa specifically

Translation
- tons of factors
- suggests it must have arisen stepwise

replication
↓
tRNA
↓
aa tRNA synt.
↓
ribosome

1) modern tRNA synthetases are diverse in size & structure.
2) a.a. does not interact w/ cognate anticodon



Two types of aa-tRNA synthetases
- suggests evolved separately

Summary
① tRNA early in replication

WF Doolittle - Root of Life

Reconstructing Pre-Progenote Evolution
1) molecular fossils
2) cellular fossils (current)
3) comparative molecular biology

cenancestor - the most recent common ancestor of all living things

was the cenancestor a progenote

Progenote Concept

- Woese - suggested cenancestor was Progenote

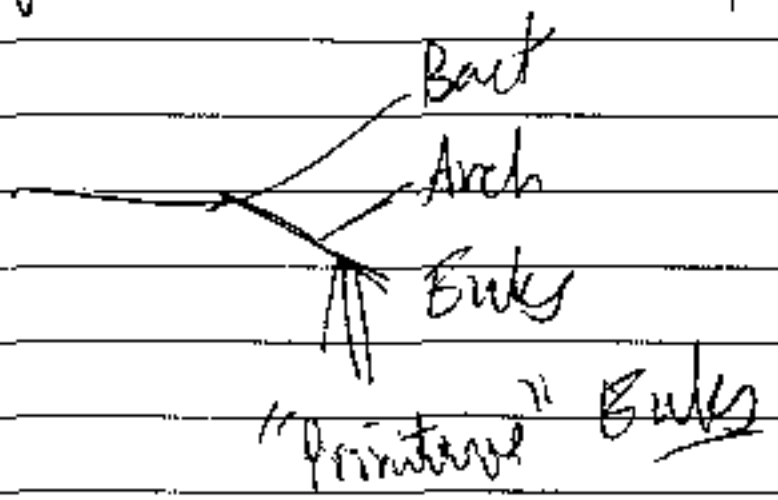
Exon theory of genes apparently still endorsed only by Walter Gilbert

Rooting - how?
Iwabe et al
Bacteria
Archaea
Eukarya

Map of genome

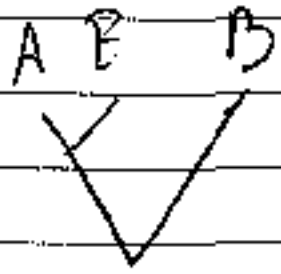
- similar operons

- suggests progenote was sick so simple

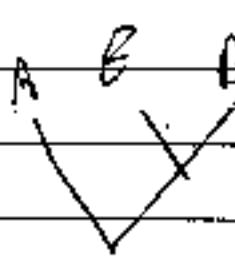


Bact - problems w/ rooting

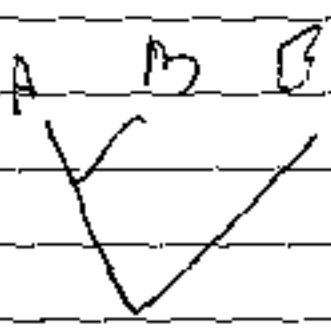
1) EF hard to align



Arginosuc.
ATPase B
2A
Pol II
EF Tu
G
HmgCoA
Ribo L2
L23
RNA pol B



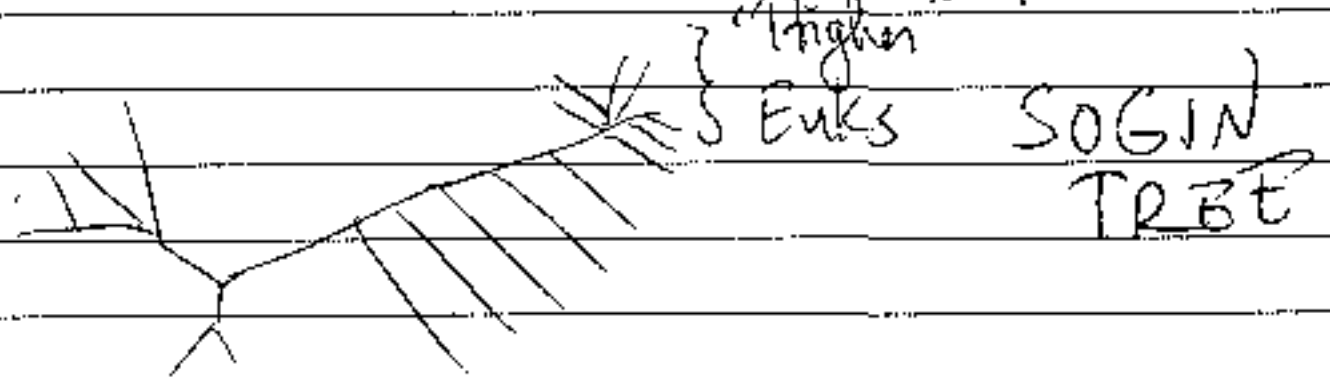
Enolase
FeMn SOD
PGK
GAPDH
MDH
Glut synth
TrpB
C
GDH

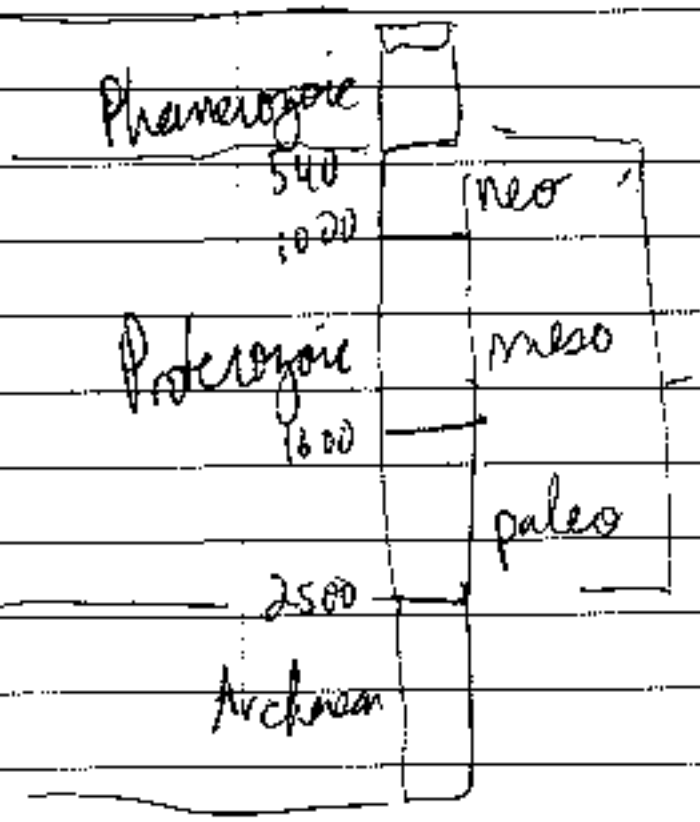


Citrate syn
Glut syn I
Isoleucyl tRNA synthetase
Gyrase B
Hsp 70
TrpA
TrpE

GDH
Aspartate aminotransferase

Andy Knoll - Proterozoic Eukaryotes - Harvard





Fossil Eukaryotes

- 17-1800 mya & before
- 1st 5-600 mya after this - fossil diversity is low
- 900-1000 mya - 1st real morphological diversity of unicellular forms
- 1000-1100 - multicellular show up
- most of these forms disappear ~600 mya

~600 mya - new more morphologically complex forms show up

- most of these are gone by ~560 mya when animals showed up

What about using sliding windows?

Suggests part of explosion at ~600 mya may be related to the origin of meiosis

also suggests that explosion of very distinct phyla simultaneously may have an ecosystem explanation

Bill Schopf - UCLA

Emphasizes importance of extinction

Claims two separate "regions" of evolution of life

Tachytelic - Fast Morphological Change

Bradytelic - Slow "

Hypobradytelic - "

Cyanobacteria

- suggests they have been Hypobradytelic

- Problems
  1. morphology may not reflect internal
  2. not many fossils
  3. not many morphological characters
  4. not always match between rRNA & morphology

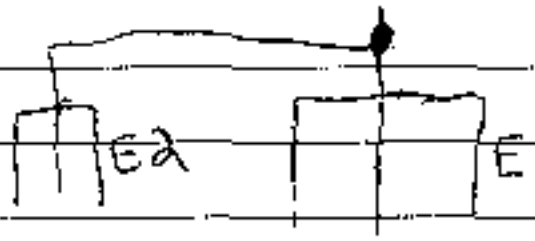
- Results up to 3.5 bya
  - fossils of certain types virtually identical to modern ones

How explain - why so slow

① strictly asexual
② ecological generalists - but will groups not species
③ difficult to exterminate

No mutation
Don't evolve 'cause they can't evolve

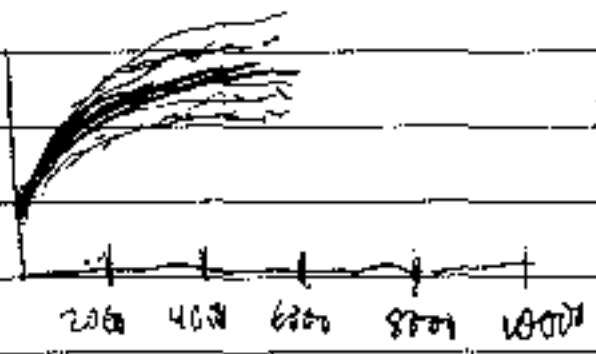
Richard Lenski - [redacted] - Mich. St.
Dynamics of Adaptation and Divergence
in Bacterial Populations



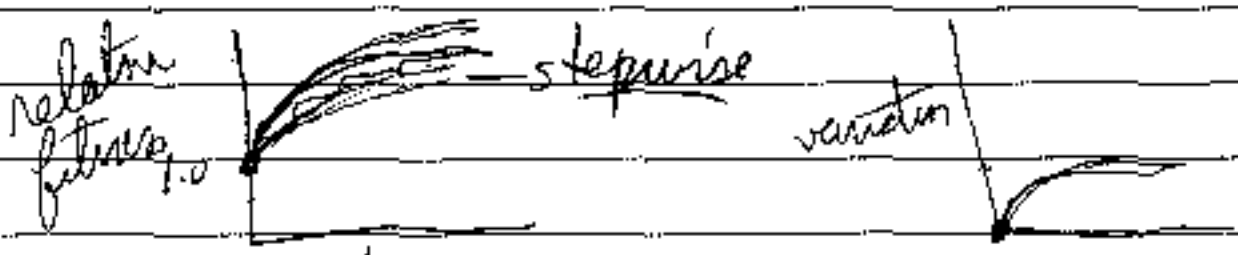
- same environment
- multiple replicates

Ecoli
- strain they use is rec⁻
- all beneficial mutations are new
- serial dilution

Sing fl

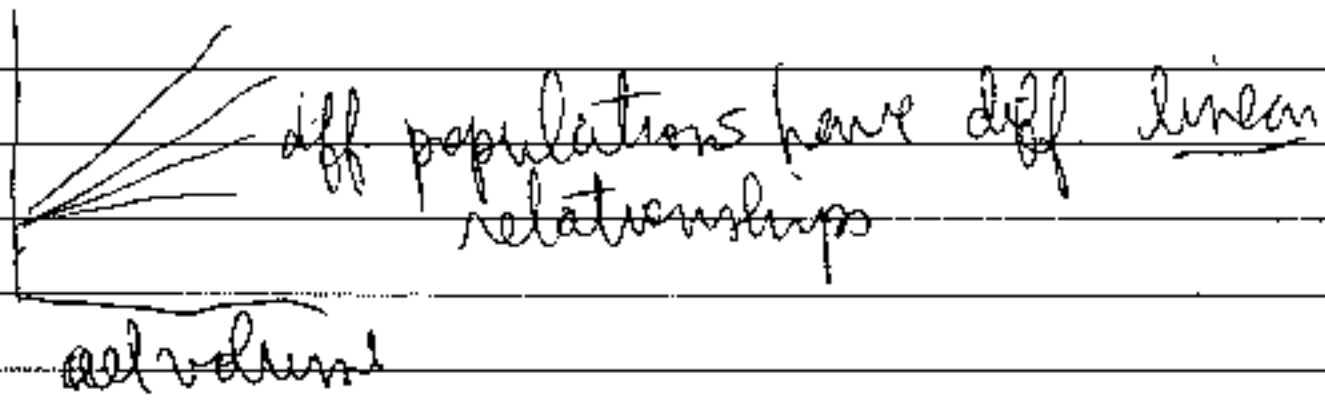


← all 12 generations do similar thing

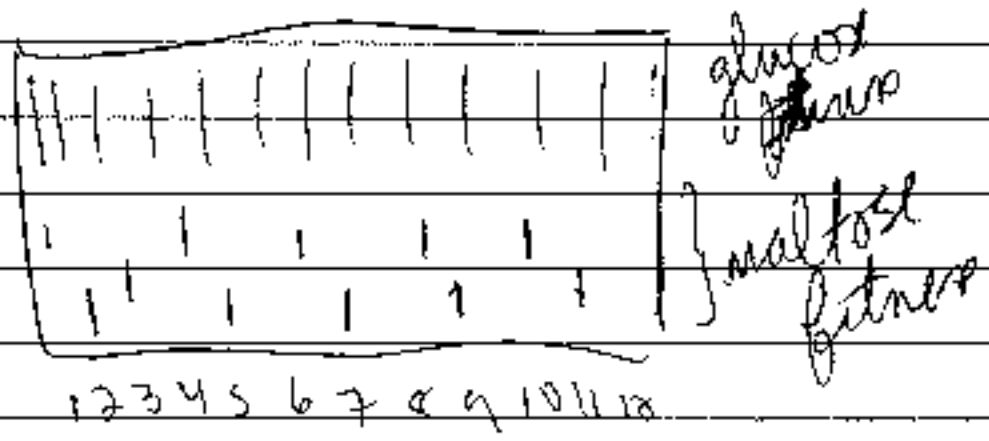


Adaptive landscape

Covariance of Fitness & Morphology

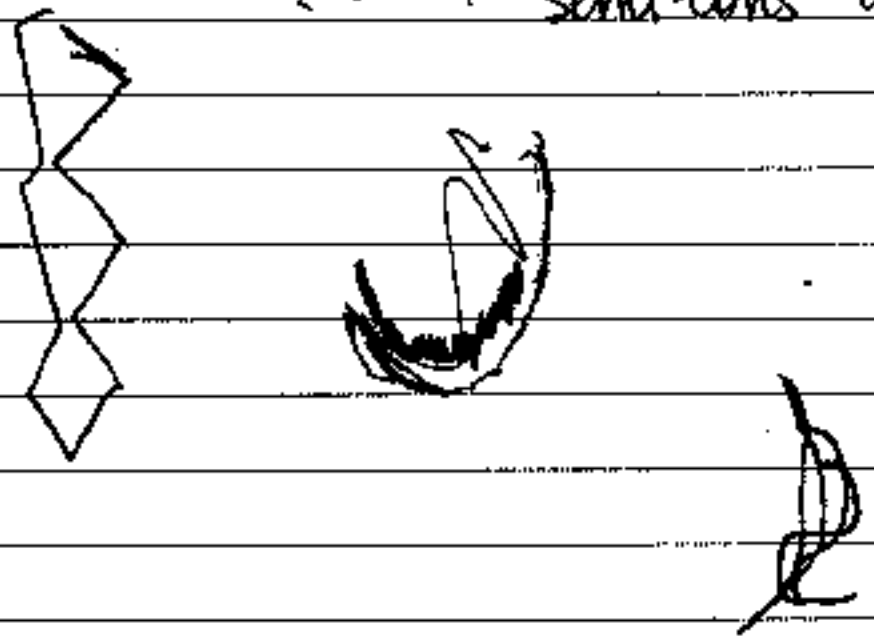


Thinks diff. orders of mutations are important



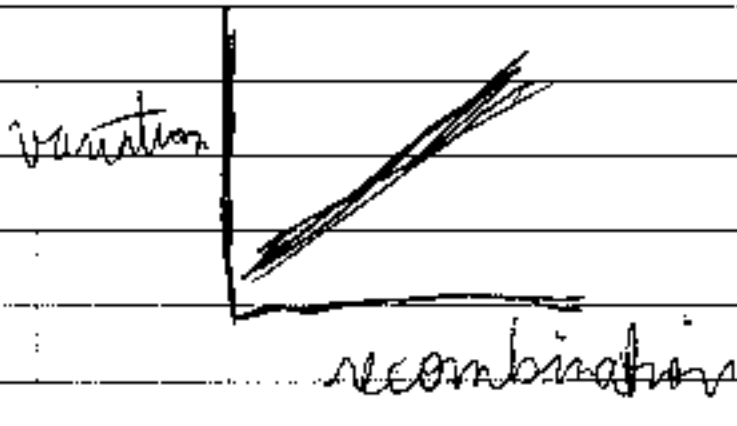
Dan Hartl - Genome Organization - Harvard

Polytene Chromosomes
- patterns conserved w/in species
- " " semi-cons among closely related species



Hudson - UC Irvine

Recombination



Explanation

① mutation rate & recomb. associated - says no rec. betw sites are not high variation

② hitchhiking

- variation deep in regions of low recomb. because area of "sweep" is higher

③ background selection

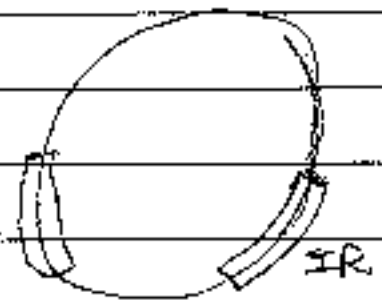
- region of genome w/ tight linkage in which deleterious mutation rate is high should have low variation.

Michael Clegg - Chloroplast DNA - UC Riverside

Plants
nuclear
mitochondrial
Chloroplast - origin ~1 bya
land plants 500 mya
flowering plants 200 mya

Chloroplast Genome
- ~80 crt
- all tRNAs & rRNAs

Liverwort: Tobacco complete sequence
Rice
Black pine
- operons
- similar order to bacteria
- some group II introns
- IR

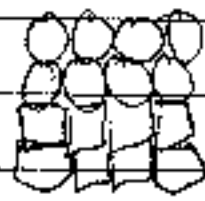


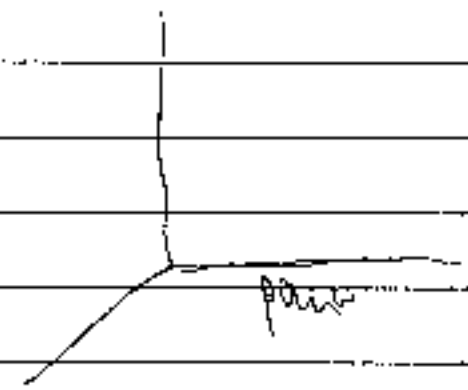
	Liverwort	Tobacco	Rice
tRNA	32	30	30
rRNA	4	4	4
ORFs	83	~80	~80

∴ General chloroplast genetic evolution appears to be conservative

what about
w/ species or individual
variation

Rubisco





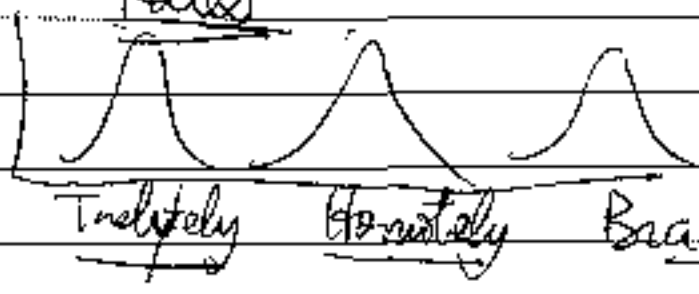
Gould - Harvard

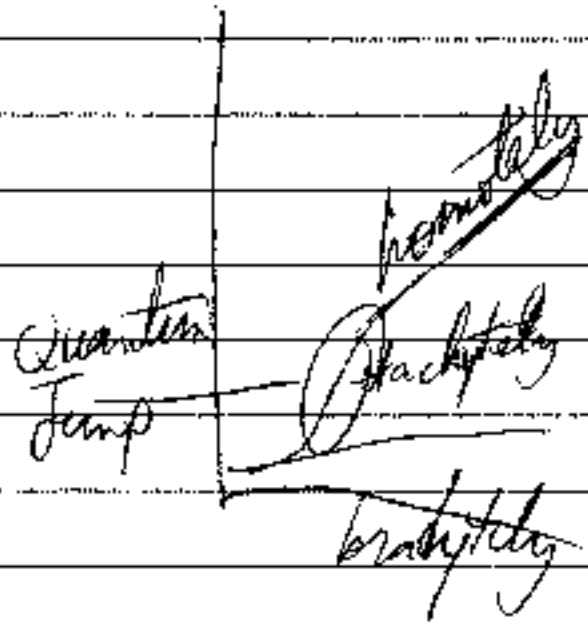
"Many Darwinisms"
- e.g. uniformitarianism
- paleontologists are there for documentary evolutionary history

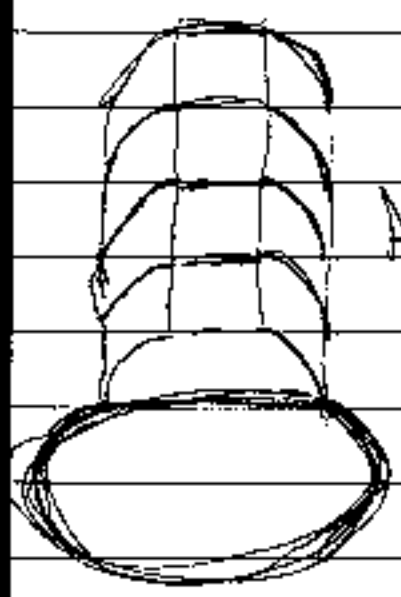
Simpson's T & M in E 1944
- says 1st book to address process & mechanisms not just evolution as an entity
- only one picture of an organism
- study of Tempo quantitatively
- can use Tempo to infer mode

3 Modes
1. speciation
2. quantum evolution
3. phyletic evolution

"Rates"







Levels of selection

Agrees w/ - DH
- individual
- group
- species
- Dodd

Henry McHenry UCDavis

Simpson & Human Evolution

Human Paleospecies

5 Australopithecus species

- including A. aethiopicus from 1 skull

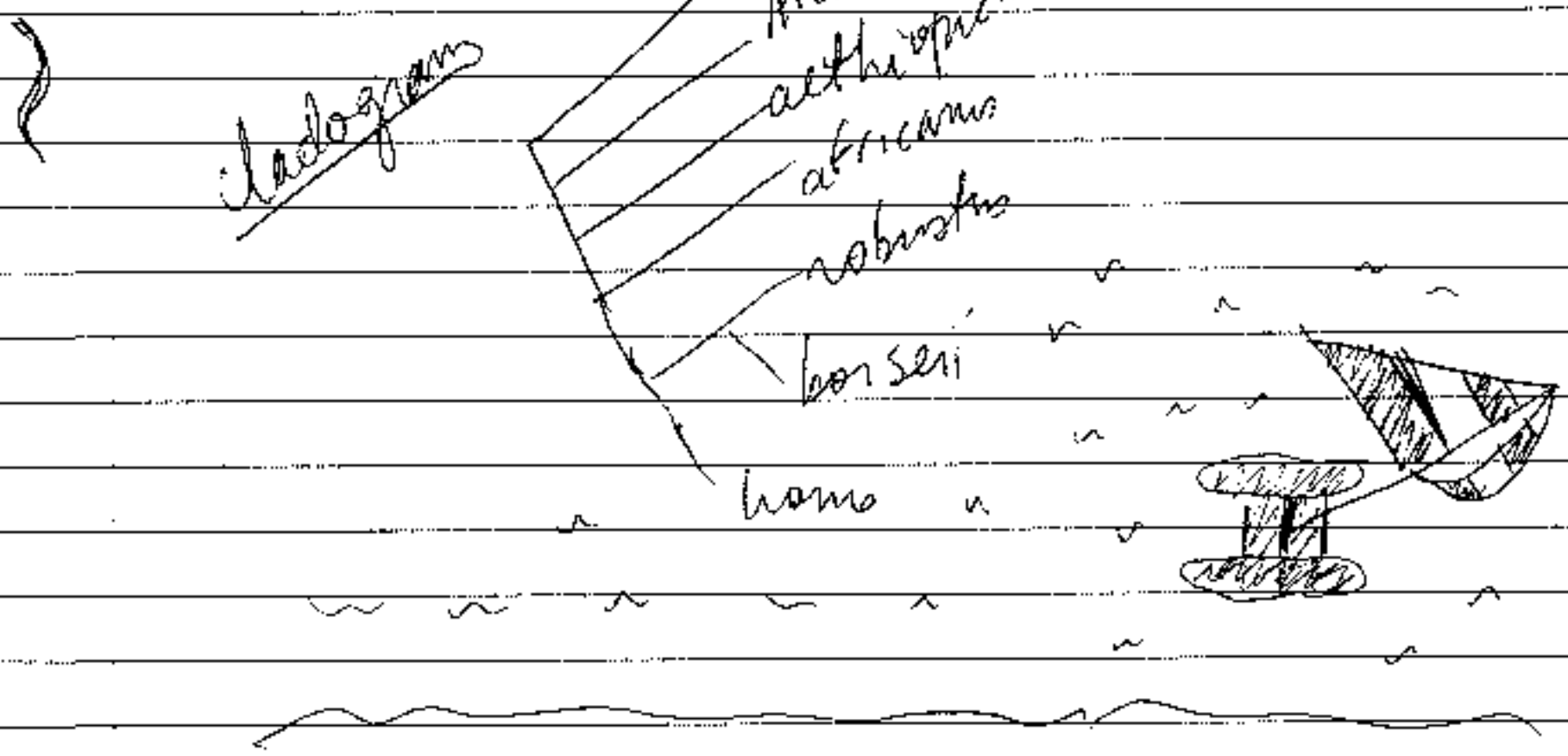
Homo

Homo habilis

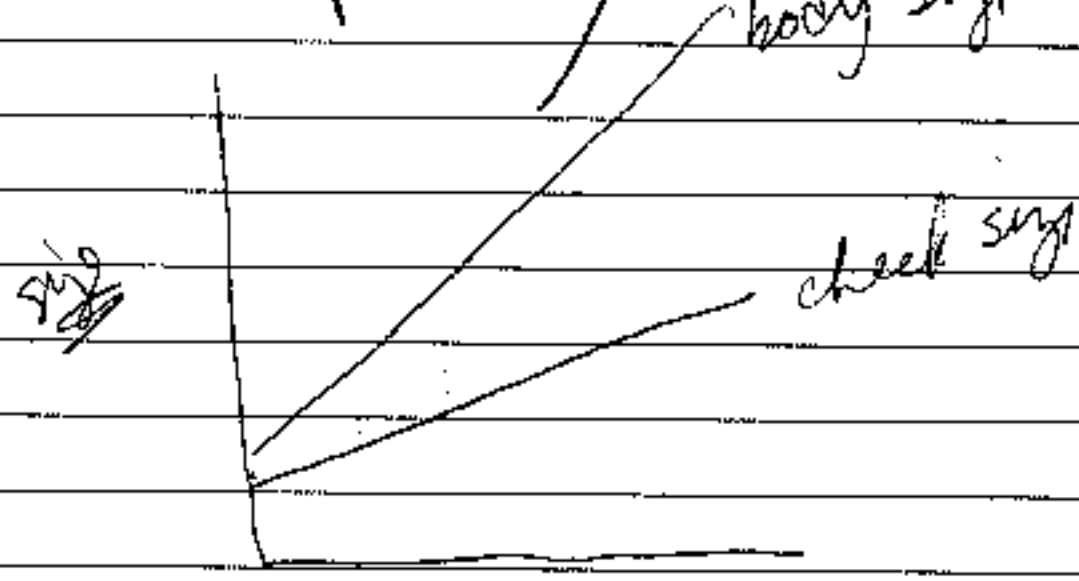
Homo erectus - 1st ~ 1.8 mya - 0.25 mya

H. sapiens - 0.4 mya

90+ cranial traits out



if a trait is variable and a new mutation shows up and spreads —



$y_1 = 2x$

$y_2 = 3x$

body = 3x

teeth = 2x

body = ax

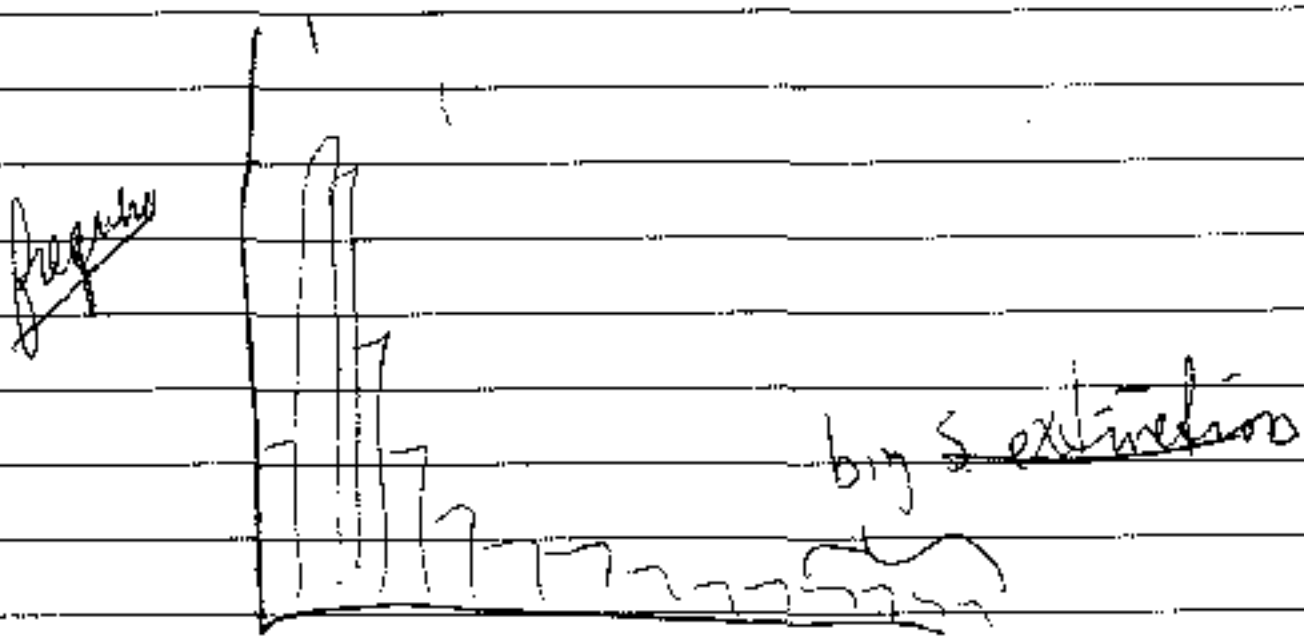
teeth = bx

teeth/body

b/a

y =

David Kamp (being read by

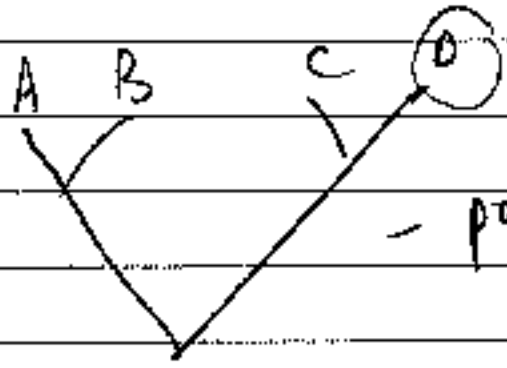


- says clustering cannot be explained by random phenomena

(- what about many height phenomena)

John Avise

Speciation
- species themselves are paraphyletic



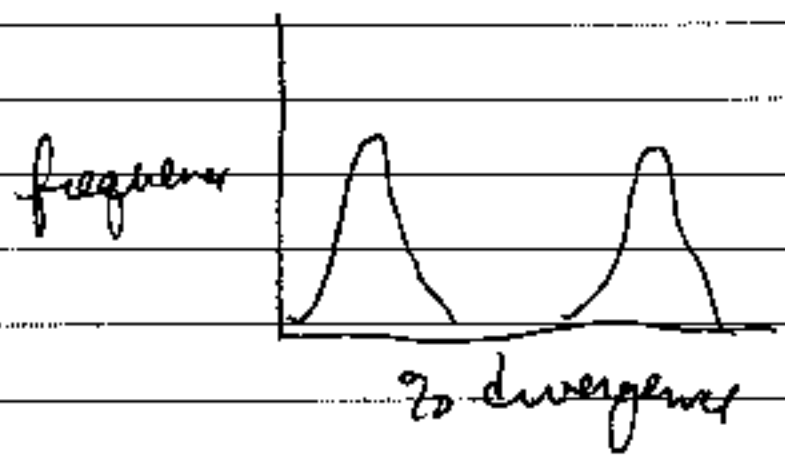
Intraspecific evolution

Coalescence

~~Phylogeography~~ Pocket Gopher = 1st study

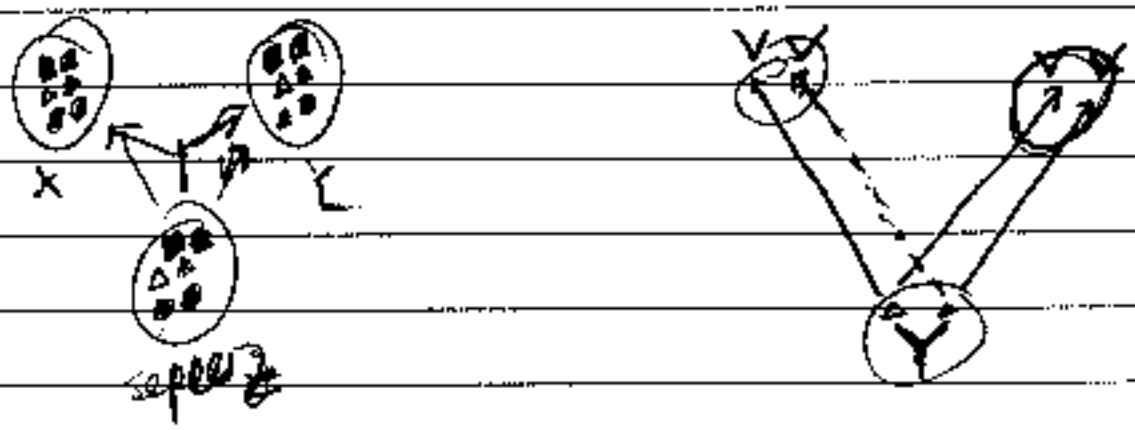
~~Mt~~
- overlay phylogenies on geographic map
- relatively physically proximal

Snow Geese
- ♀ return to where born
- ♂ meet ♀ in winter in US & return w/ ♀



1) current biology doesn't tell much about genetic relatedness
2) current genetics don't tell much about biology

Francisco Ayala - Molecular Genetics of Speciation



with gene duplication prior to divergence - gene phylogenetic split predates species split

- e.g MHC

what affects transmission of polymorphism
t = time to fixation of new allele
N - effective pop. size
S - selection effect

IF allele is effectively neutral ($NS < 1$)
$t = 4N$

- other non-species alleles

Bottleneck?

Fitch: Molecular Clocks

Definition:
~~base replacement~~
- for a molecular clock the events (letter replacements or substitutions) must be regular

Problem - don't observe replacements just comparisons

Solution

→ ZKP 1962 ① assume replacements = differences
② but not true

→ Margoliash & Smith 1965
- $r = -n \ln(1 - d/n)$
- assumes infinite # of sites

invariable - cannot w/o loss of f(x)
vs
unvaried - does not

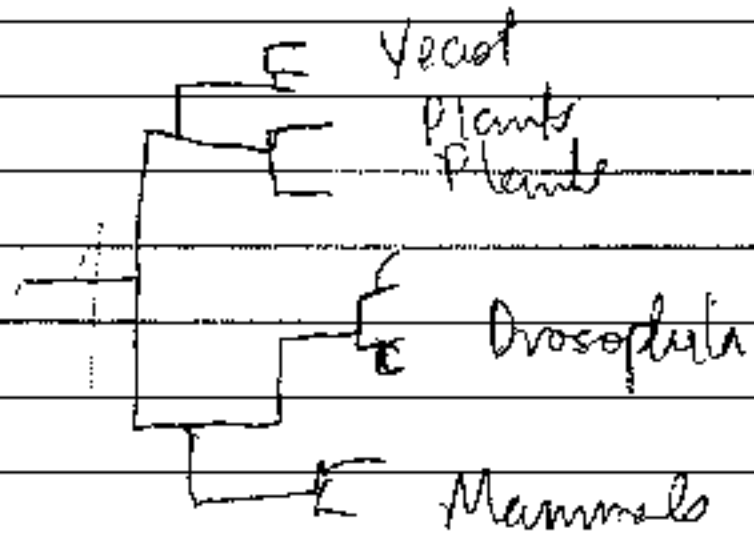
Jukes & Cantor 1969
- how many sites possible
- $r = -19/20\, n \ln\left[1 - (20/19)(d/n)\right]$ a.a's
- $r = -3/4\, n \ln\left[1 - (4/3)(d/n)\right]$ nucleotides

- but what about invariable sites

Fitch & Margoliash 1967

-- may be many unvariable positions

SOD



Conditionally unvariable positions
Sequence dependent invariable

SOP
Ayala - # aa subs not linear w/ time

Simulation of Covariation

Wyatt Anderson

Inversions

- Have a parsimony network of inversions

- Amyloid - on inversion