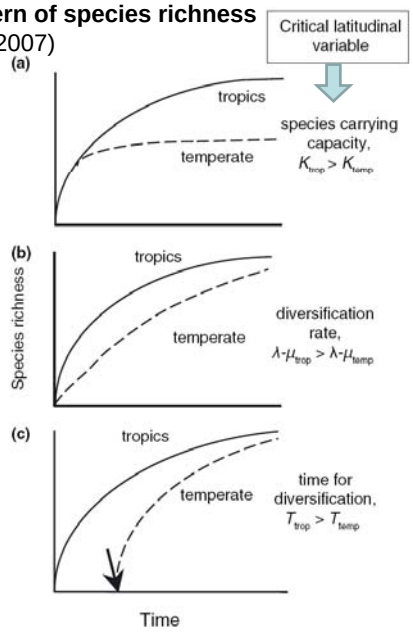
Latitudinal diversity gradient: evolutionary hypotheses

explaining a **pattern of species richness** (Mittelbach et al. 2007)

No difference in diversification rates (**different *K*'s**)

** Higher **diversification rates** in tropics (speciation rate minus extinction rate). Also: refuge hypothesis.

More **time** for diversification in tropics.



Tropics can support more species.

Tropics accumulates species richness faster.

Tropics are older.

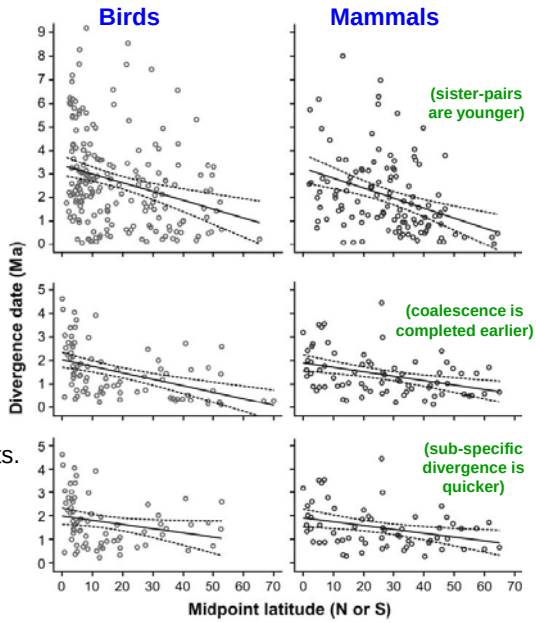
Speciation rates are ***not*** higher in the tropics (Weir & Schluter 2007)

Ages of **sister-species pairs**. (show declining age with latitude)

Max. coalescent dates for within-species haplotype variation. (decreasing lag times with latitude)

Ages of intraspecific splits. (show declining age with latitude)

Higher turnover in temperate zone!



Not to be ignored is **slow** speciation and **long** biological speciation intervals

Nearly identical ***Catalpa*** *ovata* in China and *Catalpa bignoides* in North America: 10 million years.

Similarly, European and North American sycamores (***Platanus***): separated >20 mya, yet they can still cross freely (e.g., the London plane tree).

Searching for *Speciation Genes*

"The real problem of speciation is not how to produce difference but rather how to escape from the cohesion of the gene complex." (Mayr 1963, p. 518)

"Indeed, it is becoming increasingly evident that an approach which merely counts the number of gene differences is meaningless, if not misleading." (Mayr 1963, p. 543)

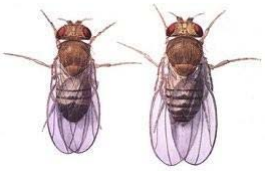
Same idea is suggested by the "Marie Curie Speciation Network" in TREE, 2011 – ***the search for speciation genes is "misdirected."***

Nevertheless, the genetic basis of speciation – its *genetic architecture* or *genomic structure* -- remains of great interest.

Two additional problems not mentioned by Mayr:

A. Lewontin's (1974) "methodological contradiction:" Studying the genetics of speciation is "an attempt to do genetics where it cannot be done – between species" (Coyne & Orr 2004).
   - "The very phenomenon under study, reproductive isolation, precludes most genetic analysis."

B. A second, practical problem: Our genetic workhorse, *D. melanogaster* is useless.
   - It forms only sterile or inviable hybrids when crossed with any other species.
   - **Haldane's rule**.



C. **A third problem:** The genetics of a critical isolating factor is not equivalent to the genetics of speciation (Woodruff & Thompson 2002)

1. Inviability of hybrids:
   - Determining genetics of such postzygotic isolation may be easy.
   - But hybrid inviability may be unimportant in speciation, because:
   - …ethological or habitat isolation may prevent hybridization in nature.
2. For species that evolved long ago:
   - Genetic analysis may overestimate the number of genes involved.
   - Most of the changes may have *followed* the speciation event.

The analogy of the abandoned pickup truck:
   - flat tires, dead battery, frozen engine, hole in fuel tank
   - were any of these the causes of its abandonment?
   - no – it ran out of gas and got left there.

Common questions about speciation genes

1. *How many* gene loci are involved?
   - many genes of small effect (Type I* architecture)?
   - few genes of major effect (Type II* architecture)?
2. *Where* are those genes located?
   - autosomal chromosomes?
   - sex chromosomes (X or Y)?
3. *What types of genes are they* – i.e., "ordinary" or unusual (e.g. repetitive DNA sequences or transposable elements)?
4. Do they *share common characteristics* across species, families, orders?
   - particular functional classes (transcription factors; regulatory genes)?
   - dominant or recessive?
   - particular rates of change (e.g. fast evolving)?
5. What *effects* do those genes have on reproductive barriers?
   - postzygotic reproductive isolation?
   - prezygotic reproductive isolation?

*(Templeton 1981)

Methods for exploring the genetics of speciation

1. *Mendelian methods*: F_1 and F_2 crosses and backcrosses.
2. *Quantitative genetic studies* using data as gathered above.
3. *Traditional crossing studies*: identifying hybrid sterility and hybrid inviability.
4. *Chromosomal change* accompanying speciation: duplication, fusion, fission.
5. *Genetic differences* accompanying speciation (i): *phylogenetic* approaches.
   - allozyme studies.
   - other molecular markers: nuclear, cpDNA, and mtDNA nucleotide sequences, microsatellite markers, RFLPs, AFLPs, SNPs, etc.
   - calculate genetic distances and percent sequence divergences.
6. *Genetic differences* accompanying speciation (ii): *finding associations* of phenotypic traits with chromosomes or subsections of the genome.
   - genome-wide association studies (GWAS) using NextGen methods.
   - linkage mapping, QTLs, & candidate genes.
   - success depends on ability to hybridize sister-species!
7. *Gene expression* (transcriptomics) and proliferating evo-devo methods.

1. How many gene loci are involved?

1. **Type I genetic architecture:** Multiple loci of small effect (polygenic)

- *Drosophila melanogaster* & *D. simulans,* hybrid sterility: >9 loci (Pontecoriso 1943)
- Other *Drosophila* studies (reviewed by Wu & Hollocher 1998)

  - hybrid sterility between species: >120 loci
  - sexual isolation between races: >10 loci

- *Laupala* crickets of Hawaii, sexual isolation: 8 "loci" (K. Shaw lab)

2. **Type II genetic architecture:** Few loci of major effect

- Swordtail x platyfish, hybrid inviability*: 2 loci.
- *Mimulus guttatus* "species" (monkeyflower), hybrid inviability: 2 loci (Bradshaw *et al.* 1995)
- Lake Malawi cichlids, sexual isolation: 1-4 loci (O'Quin et al. 2012)

- *Chrysoperla carnea*-group, sexual isolation: 1-3 loci.
- *Ostrinia nubilalis* (European corn borer), sexual isolation: 1-3 autosomal loci.

(Cardé 1978, Löfstedt *et al*. 1990)

*(tumors)

2. Where are those genes located?

Hybrid sterility in *Drosophila* (another survey by Coyne & Orr 1989)

- loci responsible are **concentrated on the X-chromosome**
- reminiscent of Haldane's Rule: In interspecific crosses, if only one sex of hybrids is sterile, it's usually the heterogametic sex.
  - males in most species (XY or XO)
  - females in birds and butterflies (XZ)

- **Expectations**, if first changes of speciation are on the X:

  - difficulties would arise first in heterogametic hybrids, because of the mismatch between the X-chromosome and one set of autosomes
  - for recessive genes, evolution on the X-chromosome is faster: they're rarely expressed on autosomes, but only need to be present in one dose on the X.
- Conclusion: **changes in recessive genes on the X-chromosome come first**, followed by changes on autosomes.

3. What types of genes are "speciation genes?" e.g., *Drosophila*

Postzygotic reproductive isolation caused by change at a single locus on the X-chromosome (Ting et al. 1998; Sun et al. 2004; Phadnis & Malik 2013):

- ***Odysseus*** gene (*OdsH*) affects sperm production
  - *Drosophila simulans* x *D. mauritiana:*
  - F_1 hybrid males are sterile (Haldane's Rule).
  - But inserting **just *Odysseus*** from *mauritiana* into *simulans* males causes Haldane-type sterility in *simulans.*
  - *Odysseus* contains a homeobox (a sequence that regulates development).
  - Rate of evolution of *Odysseus* in these fruit flies is 1000-fold faster than in other animals.

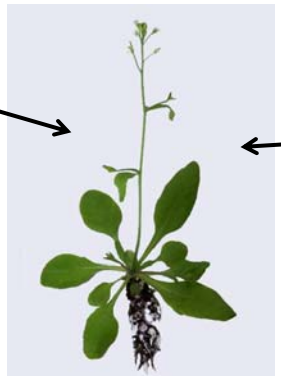
Postzygotic isolation caused by the **nucleoporin** proteins *Nup96*, *98*, & *160* (Presgraves et al. 2001; Tang & Presgraves 2009; Maehara et al. 2012):

- Gatekeepers of a cell's nucleus – in an arms race with viruses
  - *Drosophila simulans* (chromo. #3) x *D. melanogaster* (X-chromosome interaction factor)
  - Evidence of **selection** as the cause of rapid change, with sterility between species resulting as an incidental byproduct of divergence.

3, continued: What types of genes are "speciation genes?"

e.g., *Arabidopsis thaliana* (Bomblies et al. 2007; Rieseberg & Blackman 2010)

- Offspring of crosses between strains of *A. thaliana* are often tiny, with twisted & warped leaves and massive cell die-off – can't even flower.
- Caused by loci (& alleles) for **disease resistance** – analogous to function of *nucleoporins* in *Drosophila*.
- So: rapid evolution of disease resistance in different strains may be the start of reproductive isolation between them.



5. What are the effects of those "speciation genes" on reproductive barriers?

Postzygotic isolation:

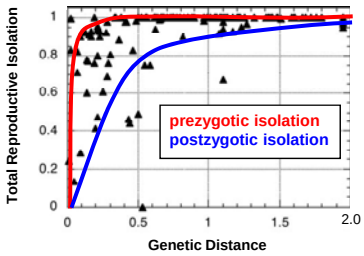
- Hybrid sterility (*Drosophila*)
- Hybrid inviability (swordtails x platyfish, monkeyflowers, *Arabidopsis*)

Prezygotic isolation:

- Behavioral barriers (*Drosophila*, green lacewings, *Laupala* crickets, European corn borer)

Evidence from Coyne & Orr's *Drosophila* survey (1997):

- Prezygotic isolation evolves much faster (at much lower values of genetic distance) than does postzygotic isolation



…so speciation genes are more likely to cause **prezygotic** reproductive isolation.

Tentative answers to questions about speciation genes (Orr 2005)

-- The factors that cause postzygotic reproductive isolation – at least in *Drosophila* – correspond to **ordinary** genes, having normal functions within species.

-- Speciation genes do not fall into any particular functional class:

- some do transcriptional *regulation* (*Odysseus* and 1 or 2 others)
- others code for *structural proteins* (*Nup96*)

-- Speciation genes may (*Odysseus*) or may not (*Nup96, 160*) be members of *duplicate gene families*.

-- Speciation genes are *often recessive* (*Nup96* and others) but don't have to be (X-chromosome interaction factor).

-- Speciation genes may (*Odysseus*) or may not (*Nup96, Nup160*) reside on the *X-chromosome*.

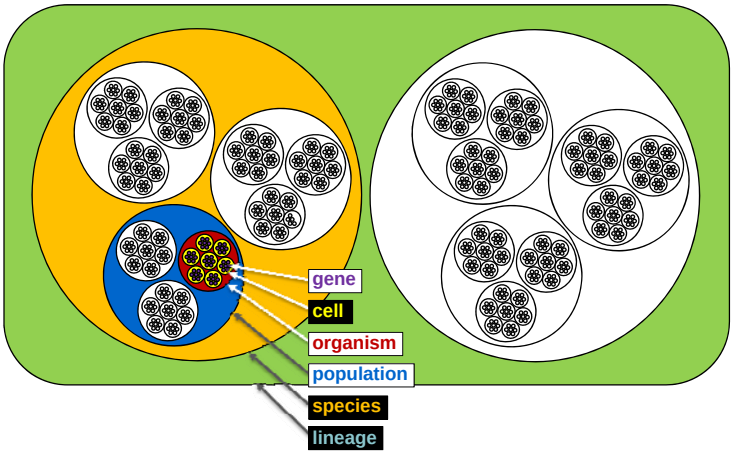
-- Speciation genes are *rapidly* evolving.

-- Speciation genes often evolve by ***positive Darwinian selection***.

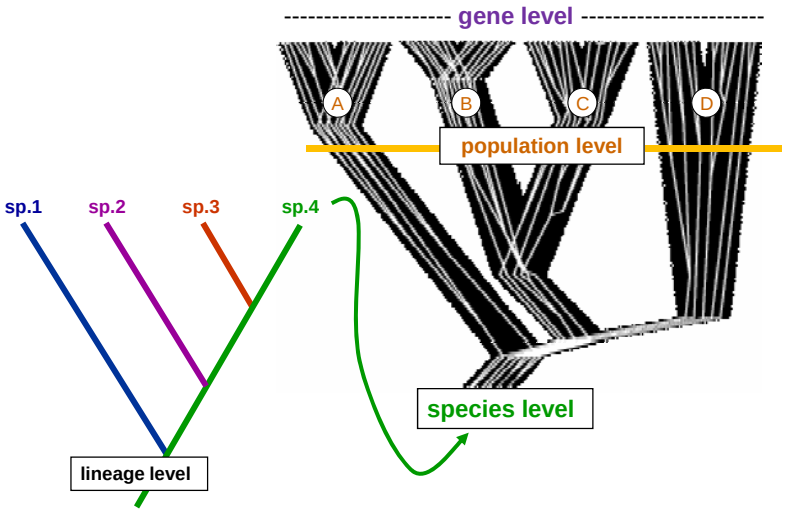
Our knowledge of speciation genes is in its infancy.

(More about speciation after we have discussed sexual selection.)

Our 3rd major topic:
Levels of Selection – or, "Multilevel selection theory"
(see Wilson & Wilson 2007, 2008)



Hierarchical organization of levels:



† David Hull

Individuality, replicators, and interactors

(Hull, Ghiselin, Williams, Dawkins, Michod, Buss, Sober, Brandon, Hurst, Gould, Lloyd, Godfrey-Smith, and others)

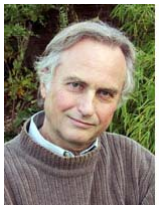
† George C. Williams

† Stephen J. Gould

Michael Ghiselin

Leo Buss

Richard Dawkins



Lawrence Hurst

Individuality, replicators, and interactors

- Natural selection acts on individuals, leading to adaptation.
- But **individuality** is a property not just of the individual organism, but of other levels of organization that are characterized by:
  - *variation, inheritability* (heredity), *reproduction,* & *continuity*

More explicitly: (Dawkins; Williams; Hull)

1. The **interactor** (Hull): An entity that directly interacts as a cohesive whole with its environment, such that this interaction causes replication to be differential (the **vehicle** of some authors).
   - It has the properties of variation and inheritability.
   - ***Organisms*** are the most common interactors/vehicles.
2. The **replicator** (Dawkins): An entity that passes on its structure largely intact in successive replications.
   - It has reproduction.
   - It has continuity (immortality): longevity, fecundity, & fidelity
   - ***Genes*** are the most common replicators.

Is the *interactor* or the *replicator* the **primary target** of natural selection and, therefore, of adaptation?

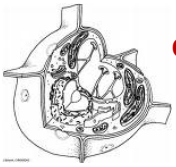
Interactors: organisms or (possibly) genomes

- Selection can't have an effect on whole organisms, because they die – they're not sufficiently permanent.
- Selection can't act on the genome either, because the offspring of the bearer of the genome inherits only genetic fragments of it (due to recombination in sexual spp).

Replicators: genes (alleles)

- The gene doesn't die.
- Nor is it fragmented across generations – it's potentially immortal, or at least it's permanent enough for its frequencies to be modified over a series of generations.
- Mark Ridley has stated that the ***need for permanence*** gives the **gene** a priority over the organism as the unit of selection.

Not everyone agrees with this view.



Conflicts between levels of selection:
what's good for the gene [species, etc.]
is **not** necessarily good for the organism

Levels (of organization) ***above*** that of the individual organism:

higher levels

- lineage – species swarms, species groups, adaptive radiations
- species (sometimes called a lineage)
- population – race, ecotype
- trait group – packs, schools, other cooperative social units
- kin group – immediate and extended families

Levels ***below*** that of the **individual organism**:

lower levels

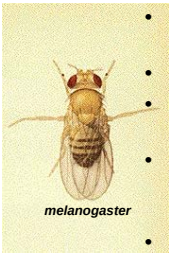
- tissue/organ – (unlikely)
- cell line – cancers
- organelle – mitochondria, chloroplasts, endosymbionts
- chromosome – X and Y sex chromosomes vs. autosomes
- gene (cistron) – segregation distorters, outlaw alleles ← (start here)

Obvious examples of conflict: sex-ratio changes, cancers, cannibalism, selfish behavior, cooperation

How is conflict between levels of organization resolved?
Some interesting examples ***at the Gene level:***
selfish genes and *genomic conflict*

Selection can act at the level of the gene (allele) if the fitness of a particular sequence is at least partially independent of the fate of other sequences in the same genome. This is ***(intra)genomic conflict***.

E.g., Segregation distorter alleles ("meiotic drive"): *Drosophila*, house mouse, *Neurospora*, maize (corn), etc.


melanogaster

- 2-locus, strongly linked system of a *distorter* locus (where the presence of ***sd*** allele distorts meiosis) and a ***recognition*** locus (which shows the distortion).
- ***sd*/+** males produce mostly ***sd*** sperm; "**+**" (wild-type) sperm fail to develop.
- One result: ***sd*** will increase nearly to fixation, after which the effect will disappear (since segregation is normal in homozygotes).
- Meanwhile: all other genes at other loci suffer a disadvantage because of ***sd***. Because they're all net losers, selection will act on all other loci (at the genome level) to shut down ***sd*** and restore the *status quo*.
- ***sd* alleles are *selfish genes* or "outlaw genes."**

Segregation distortion in any case is short lived – it either becomes **fixed** or is **suppressed** – which is probably why it's rarely seen.

Organelle level

Levels (of organization) ***above*** that of the individual organism:

higher levels
- lineage – multiple species from a common ancestor (clade)
- species (sometimes called a lineage)
- population – race, ecotype
- trait group – packs, schools, other cooperative social units
- kin group – immediate and extended families

Levels ***below*** that of the individual organism:

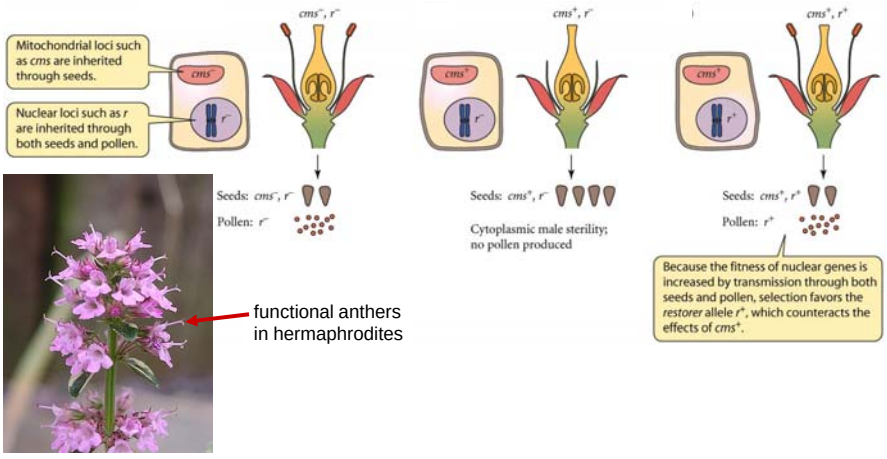
lower levels
- tissue/organ – (unlikely)
- cell line – cancers
- **organelle** – mitochondria, chloroplasts, endosymbionts
- chromosome – X and Y sex chromosomes vs. autosomes
- gene (cistron) – segregation distorters, outlaw alleles

Selfish genes and **genomic conflict** involving ***organelles:***

Cytoplasmic male sterility in a flowering plant, *Plantago lanceolata*

- Hermaphroditic mating system.
- A mutation has been documented in the *mitochondrial genome*.
  - It suppresses male function: certain individuals produce no viable pollen…
  - …and thus allocate more resources to female function.
- This allele will be strongly favored, because mitochondria are passed down in the female line only.
- It's an outlaw gene from the perspective of the *nuclear genome*, which favors hermaphrodites.
  - i.e., nuclear genes in those inviable pollen grains won't survive.

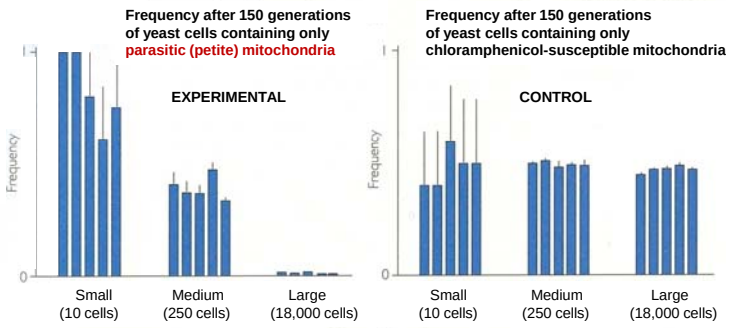
Or cytoplasmic male sterility **(CMS)** in thyme, *Thymus vulgaris* – another example of a selfish **organelle** allele



Organelles versus cells, individuals, and colonies: "Petite" mutations in yeast, *Saccharomyces cerevisiae*

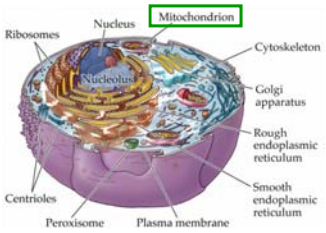
- The *petite* mutation is a large deletion in the *mitochondrial genome*.
- It allows *more rapid replication of mitochondria* and cell division, because the mitochondrial genome is smaller.
- But it impairs the growth of yeast **cells** (individuals), which form "petite" colonies that are **more anaerobic**.
- In asexual reproduction, the "petite" colonies are outcompeted by normal (aerobic) yeast colonies and overgrown.
- (note that in sexual reproduction, the situation is more complicated.)

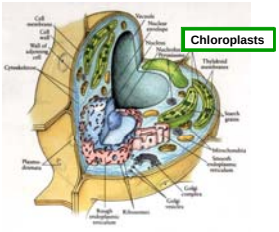
Data from *Saccharomyces cerevisiae*: (Taylor *et al*. 2002)



- Start with yeast cells with both *normal* and *parasitic mitochondria*
- Intracellular competition favors faster-replicating parasitic mitochondria
- But at the cellular (organism) level, **aerobic** yeasts replicate faster.
- Where competition is low (small colonies), parasitic MT increase; where it's high (large colonies), parasitic MT disappear.
- Control uses 2 types of mitochondria whose differences are neutral.

Is genomic conflict the ultimate cause of
***unisexual transmission* of organelles?**





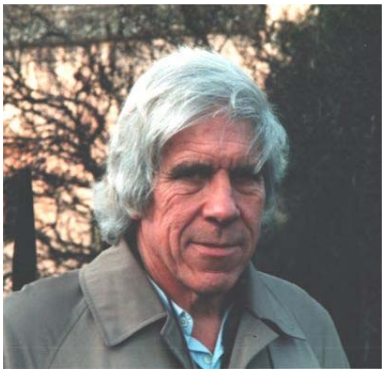
- Mitochondria and chloroplasts are usually transmitted in the cytoplasm of the egg, so they are maternally inherited.

EXCEPTIONS:
- Several <u>conifers</u>: mitochondria maternally, chloroplasts paternally
- <u>Bananas</u>: mitochondria paternally, chloroplasts maternally

- There's no mechanism equivalent to **Mendelian segregation** to divide the mtDNA "individuals" evenly and **fairly** during reproduction.
- It has been suggested (Hurst 1991) that two sexes, with uniparental transmission of organelles, originated to eliminate this source of genomic conflict. ***Nuclear genes can thereby police the DNA in organelles.***

A perspective from one of the pioneers
in the field, William D. Hamilton
Narrow Roads of Geneland, vol I (1996)

"As I write these words, even so as to be able to write them, I am pretending to a unity that, deep inside myself, I now know does not exist. I am fundamentally mixed, male with female, parent with offspring, warring segments of chromosomes… interlocked in strife …"



"I will leave a sum in my last will for my body to be carried to Brazil and to these forests. It will be laid out in a manner secure against the possums and the vultures just as we make our chickens secure; and this great *Coprophanaeus* beetle will bury me. They will enter, will bury, will live on my flesh; and in the shape of their children and mine, I will escape death. No worm for me nor sordid fly, I will buzz in the dusk like a huge bumble bee. I will be many, buzz even as a swarm of motorbikes, be borne, body by flying body out into the Brazilian wilderness beneath the stars, lofted under those beautiful and un-fused elytra which we will all hold over our backs. So finally I too will shine like a violet ground beetle under a stone."

(W. D. Hamilton died of a perforated duodenum after a trip to Congo to explore the Oral Polio Vaccine hypothesis for the origin of HIV in the 1950s.)

Cell level

Levels (of organization) ***above*** that of the individual organism:

higher levels

- lineage – multiple species from a common ancestor (clade)
- species (sometimes called a lineage)
- population – race, ecotype
- trait group – packs, schools, other cooperative social units
- kin group – immediate and extended families

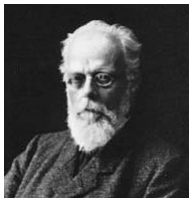
Levels ***below*** that of the individual organism:

lower levels

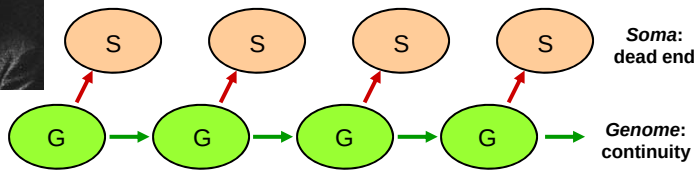
- tissue/organ – (unlikely)
- **cell line** – cancers
- organelle – mitochondria, chloroplasts, endosymbionts
- chromosome – X and Y sex chromosomes vs. autosomes
- gene (cistron) – segregation distorters, outlaw alleles

Conflict between ***cells*** and individual organisms

- Relatedness: **all cells** start out related to each other, and to the organism as a whole, by 100%, i.e., ***r* = 1**.
  - The **coefficient of relatedness (*r*)** between two individuals is defined as the percentage of genes that those two individuals *share by common descent* (more on ***r*** & ***IBD*** later).
- So why should there be any conflict?
- Possibly, triggered by subsequent mutations that make cells slightly different from one another…creating individual cell lines.
- But can these new cell types (and cell lines derived from them by reproduction) be **replicators** – having ***permanence*** as well as reproduction?
- That depends on whether an organism is ***Weismannist*** or ***non-Weismannist*** (Weismann 1891)



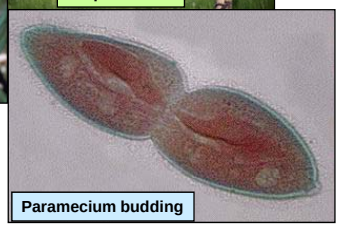
Weismannist Organisms
(like us: non-clonal plants or animals)



- Weismannist organisms: **Somatic** cell line is independent of the **germ** cell line (actually, true of *less than 20%* of organisms: Niklas & Kutschera 2014)
- Somatic cell lines, no matter how prolific, die when the organism dies.
- Therefore, ***adaptive genomic changes are limited to the germ cell line*.**
- This separation limits the possibilities for selection at the level of individual cell lines.
  - A cell may mutate to a super reproducer – a "cancer" – but this "adaptation" will not be passed on (it's ***impermanent***).

Non-Weismannist Organisms

- Weismannist organisms are actually in the minority -- < 20%
- Non-Weismannist organisms include those capable of reproduction by cloning:
  - Protista
  - Bacteria
  - Fungi
  - Plants
  - Inverts.
- **Advantages**:
  - Mutant clones can exploit locally favorable conditions better.
  - Local somatic mutations within an organism can help in the co-evolutionary war with herbivorous insects. (Gill *et al.* 1995; Folse & Roughgarden 2012)
- Sacrifices some degree of organismic control over the cellular level, which is responsive to selection.



Interesting asides:

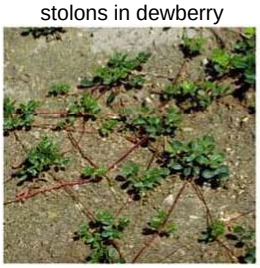
-- The "Humongous Fungus" *A. solidipes* in E. Oregon – 2200 acres, 2400 yrs old.

-- The "Trembling Giant" or "Pando" of Utah – 47,000 tree trunks, 106 acres, 80,000-yr-old roots (Mitton & Grant, var. pubs).

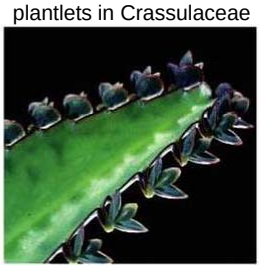
Why did Weismannist organisms evolve?

other non-Weismannist organisms:

stolons in dewberry

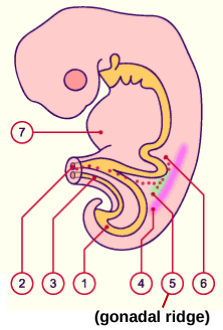


plantlets in Crassulaceae



ramet (derived individual) vs genet (population of individuals)

Weismannist organism:



- Mechanisms have evolved, such as Weismannist ***germ-line segregation***, to **reduce conflict at the cellular level.**

Human embryo, 5th week:

Primordial germ cells migrate out of yolk sac, ending up segregated in the gonadal ridge by 6th week.

(same process occurs in invertebrates, too)

Why did Weismannist organisms evolve?

Leo Buss, 1987: *The Evolution of Individuality*

> "The evolution of life from its inception must have involved the encapsulization of the germ line in an ever-expanding series of interactors [individuals] and ever-expanding levels within which competition [conflict] is suppressed."

In addition to germ-line segregation suppressing cellular-level conflict:

- Nuclear genes **police** the behavior of cells within the organism through **top-down control**, destroying outlaw cell lines that cause conflict at the cellular level.

Potential downside: a separate germ line *favors* **selfish genetic elements**, e.g., transposable elements, intracellular parasites like *Wolbachia*, and endosymbionts (Johnson 2008).

Group level

Levels (of organization) ***above*** that of the individual organism:

higher levels

- lineage – multiple species from a common ancestor (clade)
- species (sometimes called a lineage)
- population – race, ecotype
- **trait group** – packs, schools, other cooperative social units
- **kin group** – immediate and extended families

Levels ***below*** that of the individual organism:

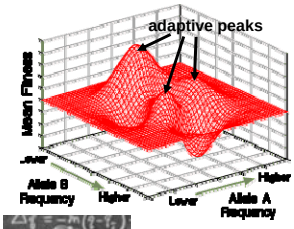
lower levels

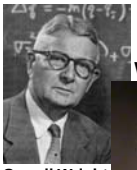
- tissue/organ – (unlikely)
- cell line – cancers
- organelle – mitochondria, chloroplasts, endosymbionts
- chromosome – X and Y sex chromosomes vs. autosomes
- gene (cistron) – segregation distorters, outlaw alleles

***Above* the organism: *Group* (intergroup) selection**

- Group selectionist thinking permeates our thinking: "adaptations are for the benefit of the social group, population, or species."
- So the idea of intergroup selection is very old.
- First **modern** approach, Sewall Wright (1931): The only practicable mechanism for bringing about a rapid and non-self-terminating evolutionary advance:
  1. Assume an adaptive (genetic) landscape;
  2. Divide a population across that landscape into isolated and differentiating subgroups;
  3. Genetic drift sends these subgroups to different locations on the landscape;
  4. Each subgroup has a different probability of surviving, relative to other subgroups.
- Developed and promoted along very different lines by V. C. Wynne-Edwards (1962).

an Adaptive Landscape:





Sewall Wright

Wynne-Edwards

V. C. Wynne-Edwards and group selection

A "group adaptation" is a property of a group of organisms that benefits the survival and reproduction of the group as a whole.

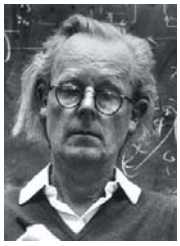
These adaptations *benefit the group but **NOT** the individual organism*.

- It's important to specify this, because many individual adaptations are adaptations at higher levels of organization as well, thus resolving any conflict between levels, *e.g.* hunting skills in lions.
- *Example of a group adaptation*: individual animals might restrain their reproduction so as not to over-eat their food supply.
  - Individual selection favors maximum reproduction rates.
  - Control of population growth generated quite a controversy in ecology about "density dependent" vs. "density independent" population regulation (Allee et al. 1948; Lack 1954)**.

Wynne-Edwards' 1962 "*Animal Dispersion in Relation to Social Behavior*." – a monumental compilation of data and relentless argumentation favoring his view.

**Back to the future: see Mitteldorf and Goodnight, 2012: "Post-reproductive life span and demographic stability."

The counter-argument of John Maynard Smith: An "infection model" (1964; 1976)



Consider a population of 2 genotypes, one for an Altruistic group-adaptive trait (allele ***A***) and another for a Selfish trait (***S***):

- *I* = groups of mainly altruistic individuals = altruistic groups.
- *II* = groups of mainly selfish individuals = selfish groups.
- *I*-groups will go extinct at a lower rate, because group selection favors altruism.

But – individual selection *HAS* to favor selfish individuals within all groups.

- If *I* gets **infected** by individuals from *II*, selfishness will take over in that group and become fixed – almost inevitably!

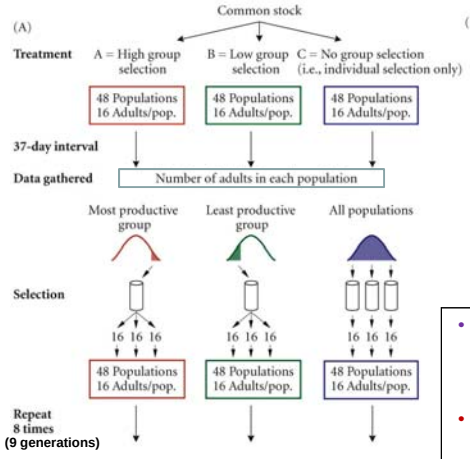
Ultimate outcome depends on relative strengths of the processes:

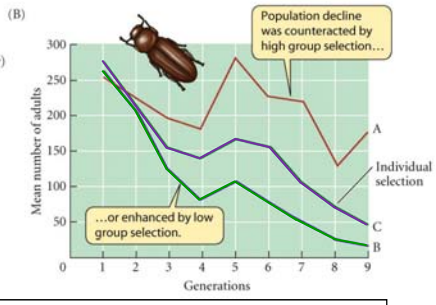
- very high rates of selfish-group extinction → altruists increasing.
- but "migration rate" of selfish individuals into altruistic groups or empty patches must be *less than one successful immigrant during the lifetime of the group*.

G. C. Williams, 1966

His simple argument: Greater abundance and more rapid turnover of individuals means that selection will be ***stronger*** at the individual level than it will be at the group level.

Experimental group selection over 9 generations
in *Tribolium castaneum* flour beetles (Wade 1977, 1979)



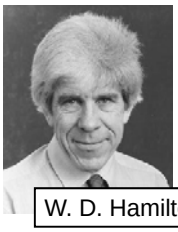


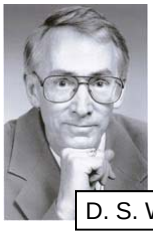
- **Control (C):** individual selection causes population size to decrease. Reason is cannibalism – advantageous at individual level.
- **Population A:** group selection counteracts individual selection.
- **Population B:** group selection reinforces individual selection.

The "group" characteristic under selection:
POPULATION SIZE **(large, or small)**

So group selection *IS* possible. But in what *natural* situations?

1. ***Kin* selection** (William D. Hamilton, 1963, 1964): Altruistic behavior in individuals toward very ***close relatives***, so that helping your relatives to reproduce helps your own genes increase in the next generation.
   - **Inclusive fitness** (kind of an extended phenotype)
   - Translates group-level adaptations into individual-level adaptations, thereby *resolving any possible conflict* between the two levels.


W. D. Hamilton

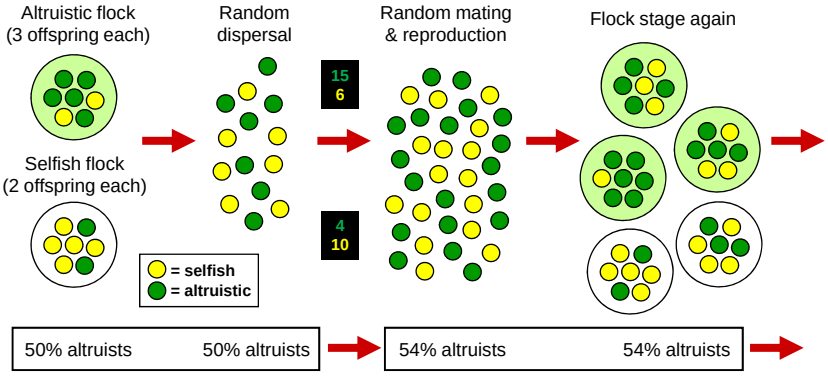

D. S. Wilson


E. O. Wilson

2. ***Trait-group* selection** (David Sloan Wilson, 1975): ***Unrelated*** groups with certain individual traits, e.g., altruism, ***out-reproduce*** those without those traits because of differential group-level reproductive success.

***Trait-group* selection is the closest to "real" group selection**
(there's more of G. C. Williams than D. S. Wilson in this model)

Each individual derived from an *altruistic* flock can fledge **3** offspring, while each individual derived from a *selfish* flock can fledge only **2** offspring (parents all die).



Note that trait-groups do not reproduce and **have no persistence** – ***they are not replicators***. Nor does trait-group selection do anything to reduce *conflict*.

Another type of group selection that can reduce conflict between levels:

Species & ***lineage*** **selection**

Group selection can have evolutionary consequences, even if it *never* over-rides individual selection.

1. Individual selection could lead to the establishment of different adaptations in different groups:
   - *Example*: large body size in a clade of organisms might be…
     - favored by individual selection (defense, fecundity, etc.), but
     - disfavored by group selection (consumption of limited resources).
   - Long-term consequence: body size decreases over evolutionary time.
2. Those different adaptations result in different rates of ***group extinction*** or group expansion (***reproduction***).
   - The kinds of groups that become extinct less often will increase in frequency…
   - …as also will the kinds of groups that multiply faster.
   - Evolutionary innovations and empty niches obviously influence this process.
3. Requires (**?**) that the species or lineage have the property of **individuality**.

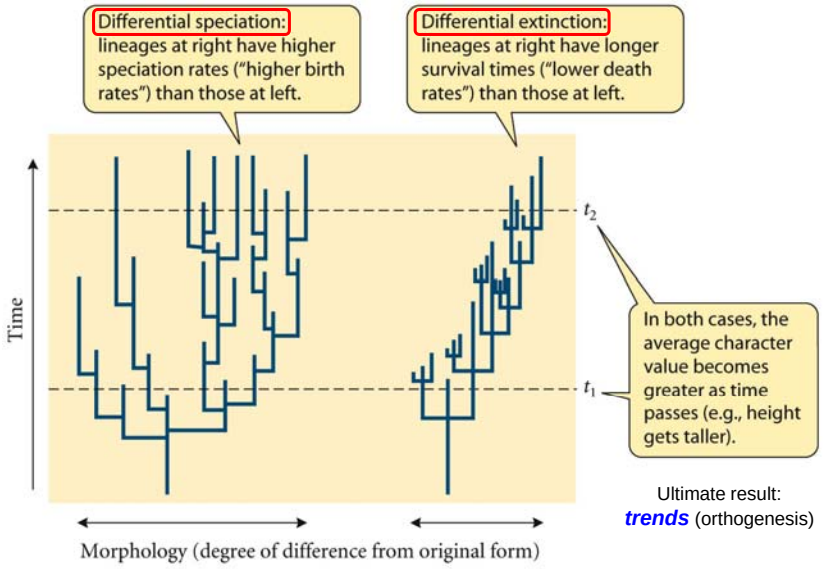
Contributors: Stanley 1975; Van Valen 1975, 1976; Gould (w/Lloyd) 1998-2000.
Recent reviews: Jablonski 2008; Raboski & McCune 2010).

Species selection & **lineage selection**

Species/lineage selection probably requires that the species or lineage have the property of ***individuality***. Is this a problem?

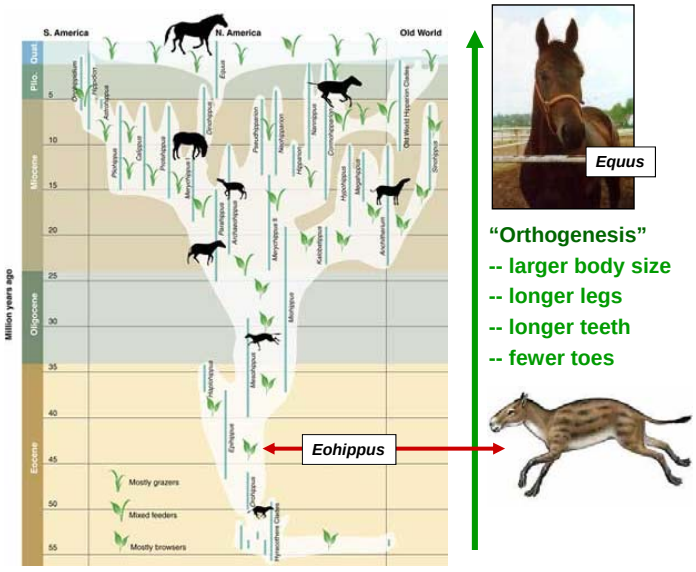
1. To be considered an individual, the species/lineage must possess or show:
   - variation [among species] in traits that are ***heritable;***
   - variation in traits that influence the probability the species will…
     - ***survive*** [avoid extinction], and…
     - ***reproduce*** [speciate];
   - reasonable ***continuity*** [stasis] over time.
2. From this perspective, species DO have some properties of ***interactors*** and ***replicators***, because
   - species reliably pass on some structure or information to their descendants.
   - these inherited structures influence the “phenotype” of species.
   - species persist in a condition of stasis for reasonably long time intervals.
3. But their status as ***either*** is uncertain. All quite confusing and philosophical…

Group selection at the level of species and lineages (Stanley)



Ultimate result:
trends (orthogenesis)

The effect of lineage selection on horse evolution (Benton & Harper 1997)



Kin-group selection and *inclusive fitness*

- "Kin group" – a special type of group that contains closely related individuals.
- Like species-level selection, kin-group selection eliminates conflict between group-level and individual-level selection.
- If group members are kin, then you are favoring (fractionally) your own genes, and therefore yourself, when you help them.
- Your actions can enhance your individual fitness, even though they are directed toward your relatives in the group at some direct cost to you.
- So: *Your fitness is the sum of your own direct fitness plus some proportion of the fitness you confer on genes that you share with your relatives by direct descent, minus the cost of your unselfish actions*.
- This is *inclusive fitness*, formalized in a series of brilliant papers by William D. Hamilton in 1963 and 1964.

The basic problem addressed by this concept is **altruism** – itself a part of ***cooperative behavior***.

Cooperation and Altruism

- An altruistic trait is a trait (or an act) that confers a ***benefit*** on someone else at a ***cost*** to the actor.
- Costs & benefits are in units of reproductive success (fitness).
- Cooperation, which includes altruism, is a basic ingredient of social interactions, social behavior, and societies.
- Different evolutionary paths to cooperation:
  1. Group selection (earlier).
  2. By-product mutualism
  3. Kin-selected cooperation
  4. Reciprocity
  5. Manipulation