Understanding life history traits I: Demography

1. At what age should I start to reproduce (earlier or later)?
2. At what size should I start to reproduce (small size or large size?
3. How many offspring should I produce at a time? (includes propagule number and propagule size)
4. **How often should I reproduce during my lifetime? (once or many)**
5. How long should I live?

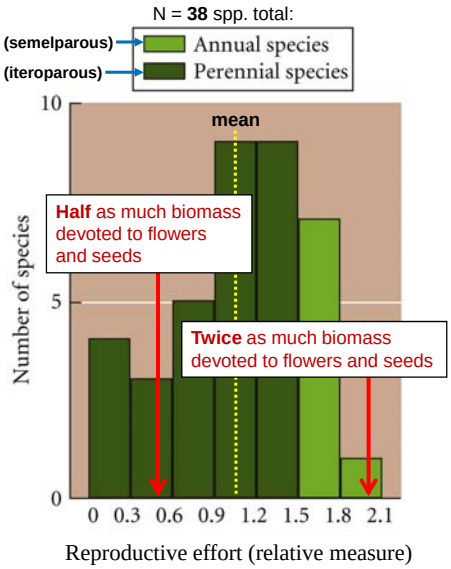
Demography: *Number of cycles of reproduction*

- An organism can reproduce once, or many times in its lifetime.
  - Once = ***semelparity*** (includes "annual" and "big-bang" life cycles).
  - Multiple bouts = ***iteroparity*** (includes "perennial" life cycles)
- **Expectation**: many bouts of reproduction (iteroparity) is better. But selection pulls organisms in many directions.
  - In general, an organism will be selected to engage in additional bouts of reproduction *only if it is likely to survive the first bout*.
  - If so, it will be selected to invest *less* in the first bout *in anticipation of future reproduction* – the iteroparous (bet-hedging) strategy.
  - So the number of cycles of reproduction is a consequence of simultaneous interactions (including trade-offs) among such factors as:
    - (i) harshness of the environment
    - (ii) mating opportunity
    - (iii) life span.

Reproductive effort in **semelparous** **vs.** **iteroparous** British grasses

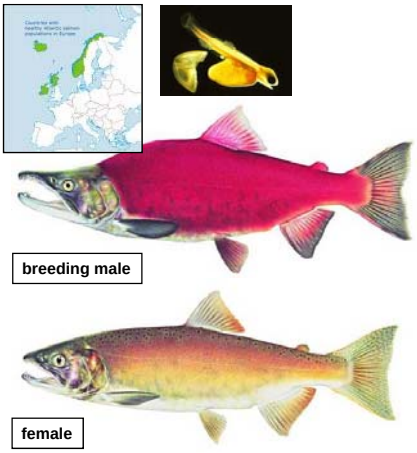
- Repeated reproduction is more likely to evolve if:
  - adults have high survival rates from one age class to the next, and…
  - the rate of population increase is low.
- Allocation to current reproduction is greater in the semelparous annual species (Wilson & Thompson 1989).

- But as individuals **age**, the intrinsic disadvantages of reproducing late in life cut in (lower reprod. value)
- So at some point, *the proportion of energy or other resources devoted to reproduction by iteroparous species should increase with age*.



"Big-bang" semelparous reproduction

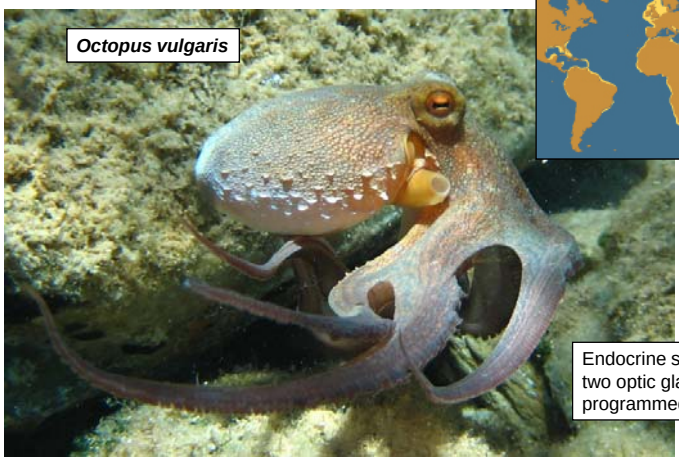
- A special form of semelparity, in which a single lifetime episode of reproduction characterizes a species with a long lifespan.
- It is thought to occur when the *conditions for reproduction are very* ***special****, and/or occur very* ***rarely****.*
  - **Atlantic salmon** (*Salmo salar*):
    - hatches in freshwater streams,
    - swims to the sea and lives there for years,
    - swims back up its natal stream to reproduce once.
  - **Explanation**? Perhaps due to the tremendous effort entailed in the swim upstream. It is then to the individual salmon's advantage to do this just once.



"Big-bang" reproduction: another animal example

(see Kirkwood & Melov 2011 on lack of real evidence for programmed death)

The females of most species of octopus deposit up to 200,000 eggs, which they care for by defending them and wafting oxygenated water over them. When the eggs hatch, she dies.



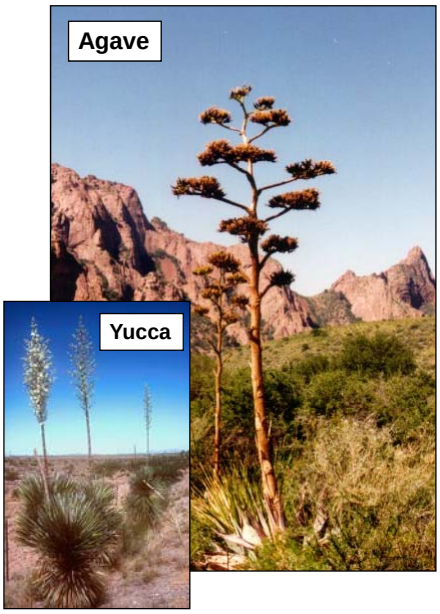
Endocrine secretions from two optic glands: "genetically programmed death?" **No**.

"Big-bang" reproduction in plants

- **Annuals** – These are the most common examples of semelparity. Here, it seems to be the interaction of inherently short life span with harsh winter conditions.
- **Bamboos** – One species grows for 130 years vegetatively, flowers *en masse* and synchronously, produces seed, and dies.
  1. **Explanation**? Successful germination in bamboo is a big problem – conditions favorable for seed germination are rare.
  2. **Or**: the seed-predator satiation hypothesis.
  3. **Or**: the fire cycle hypothesis, providing seedlings with a (fire) gap to grow in.

"Big-bang" reproduction in plants (Young 1990)

- **Agaves** (Century plants) – These grow to "100" years, then produce a huge reproductive spire and die.
  - **Explanation**? They live in very arid areas with erratic rainfall.
  - As in bamboos, the opportunities for germination are very rare.
  - With their *shallow, fibrous root systems*, century plants forego reproduction for many years, until conditions are just right.
- **Yuccas**, which are highly convergent on agaves in many ways, have *deep* ***taproots*** and therefore a different life history strategy (iteroparous).



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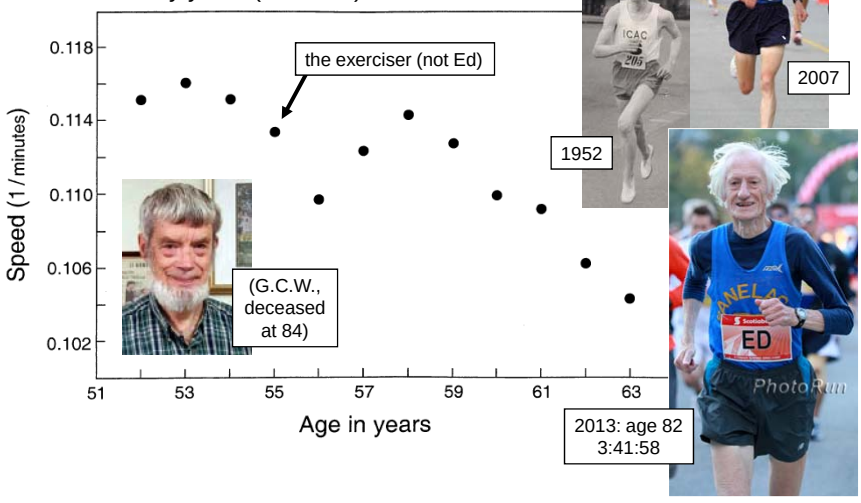
Demography: *Aging and Senescence*

- Most (sexually reproducing) organisms do not exhibit the "programmed death" of bamboos, agaves, or octopuses, but they *DO* experience a gradual increase in mortality and decline in fecundity with age: a kind of "physiological decline."
  - In humans from age 30 to 85, most functions decrease linearly, to…
    - 80-85% of nerve conduction speed,
    - 40-45% of blood volume through the kidneys,
    - and 37% of breathing capacity.
    - *Birth defects* for both sexes also increase with age – slowly at first, then accelerating rapidly.
- Senescence cannot be eliminated by natural selection, because the strength of selection declines on genes expressed at progressively greater age.
  - Fewer individuals bearing those genes survive to express them…
  - and those individuals that do are past reproductive age.
- ***Senescence begins at sexual maturity and evolves under selection.***

Demography: *Aging and Senescence*

Running performance of a dedicated exerciser, timed surreptitiously by his evil neighbor (G. C. Williams) over a period of many years (1987-'98)



Ed Whitlock, age 76: 3:04:54 = 7.05 min/mile

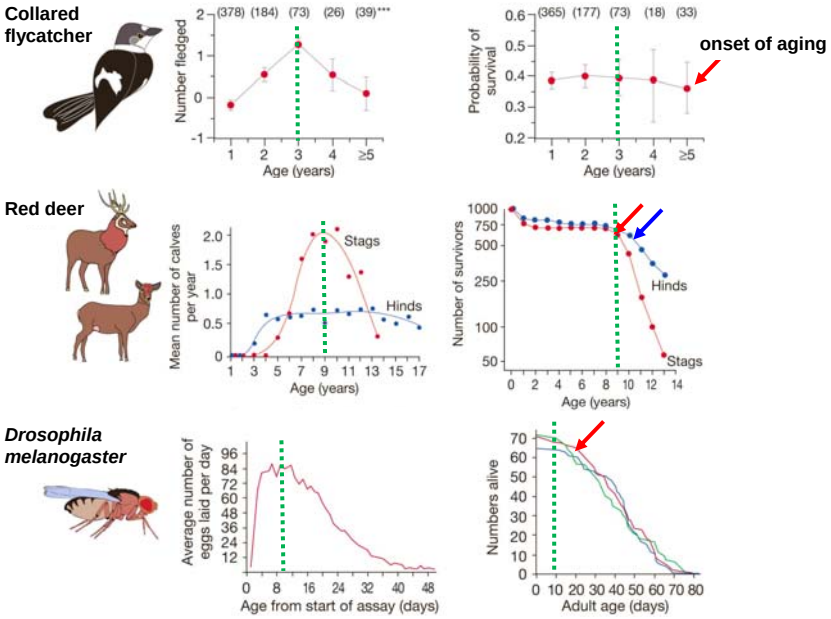
(Best: 2:31:23 in 1979)

2007

1952

2013: age 82 3:41:58

Aging in a bird, mammal & insect: *fecundity* & *survival*



Aging and Senescence

Four (somewhat overlapping) explanatory hypotheses have been proposed to account for senescence.

I. The ***rate-of-living*** hypothesis (*energy expenditure*).

II. The ***disposable soma*** hypothesis (*time required for repair*).

III. The ***mutation accumulation*** hypothesis (*genetic neglect*).

IV. The ***antagonistic pleiotropy*** hypothesis (*genetic trade-off*).

Each is based on the various and complex trade-offs and genetic correlations between and among fundamental life-history traits.

I. & II. Rate-of-Living /Disposable Soma Hypotheses

These hypotheses often invoke an evolutionary **constraint**: Populations lack the genetic variation to respond any further to selection against aging.

The cause: ***irreparable damage to cells & tissues of individuals***

1. Accumulation of errors during replication, transcription and translation within cells, tissues, & organs.
2. Accumulation of metabolic poisons (e.g., free-radicals).
3. In an evolutionary sense: cut your losses & let the body die!

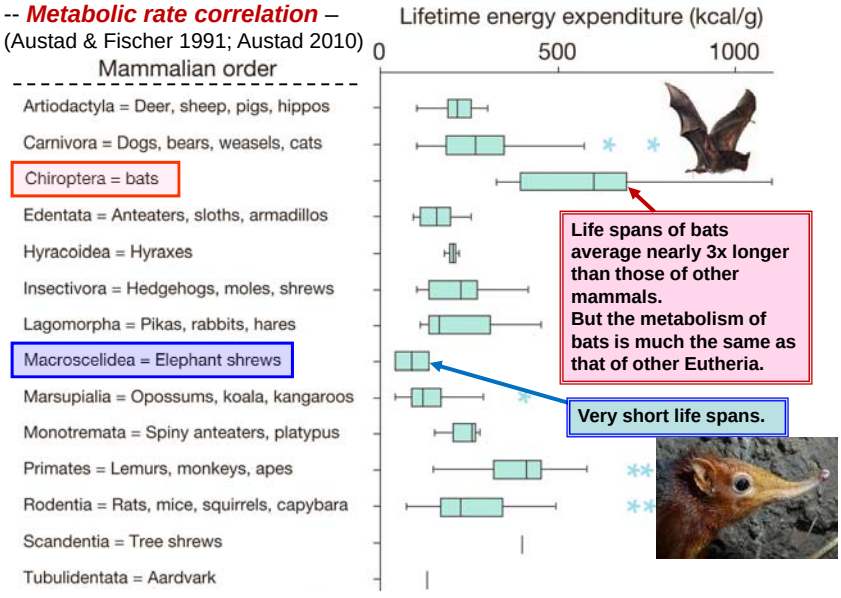
Assumption: All organisms have been selected to resist and repair damage *to the maximum extent* physiologically possible.

Predictions:

1. aging should be correlated with metabolic & mortality rates.
2. Species should *not* be able to evolve longer life spans.
3. Cellular/biochemical correlates of aging should be present.

Rate-of-living: test of *prediction 1*

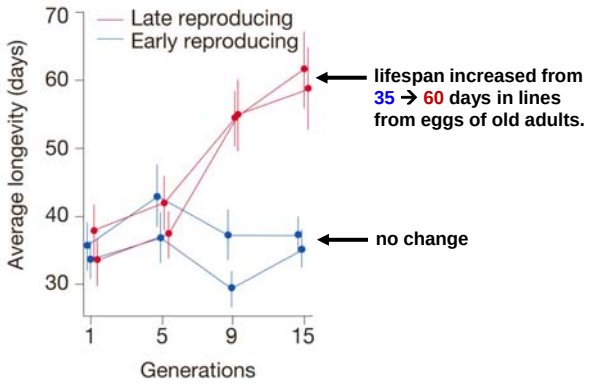
-- ***Metabolic rate correlation*** –
(Austad & Fischer 1991; Austad 2010)



***Prediction 1* is falsified. Instead? long life evolves with nonhazardous lifestyles.**

Rate-of-living: test of *prediction 2* (Luckinbill et al. 1984)

Can species evolve longer lifespans? **– YES**
Artificial selection on lifespan in *Drosophila melanogaster*



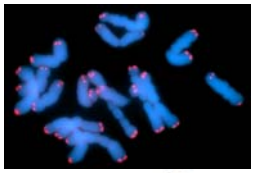
So prediction 2 is falsified.

Telomere shortening and other cell life-span phenomena

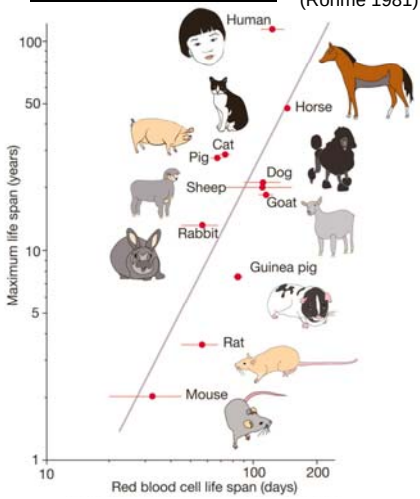
Prediction 3 (A. Olovnikov 1973)

- Somatic cells of endotherms are capable of a limited number of divisions – and duplications of their chromosomes.
- Each end (telomere) of a eukaryotic chromosome consists of many copies of a repetitive DNA sequence.
  - in most animals, it's TTAGGG
  - in arthropods, TTAGG
  - plants, TTTAGGG
- With successive cell divisions, the telomeres can shorten until cell quits dividing and dies (note telomerase).

  (note: shortening = anti-cancer: cancers deploy telomerase)
- Is life span assoc. w/ telomere rate of change and cell life span?

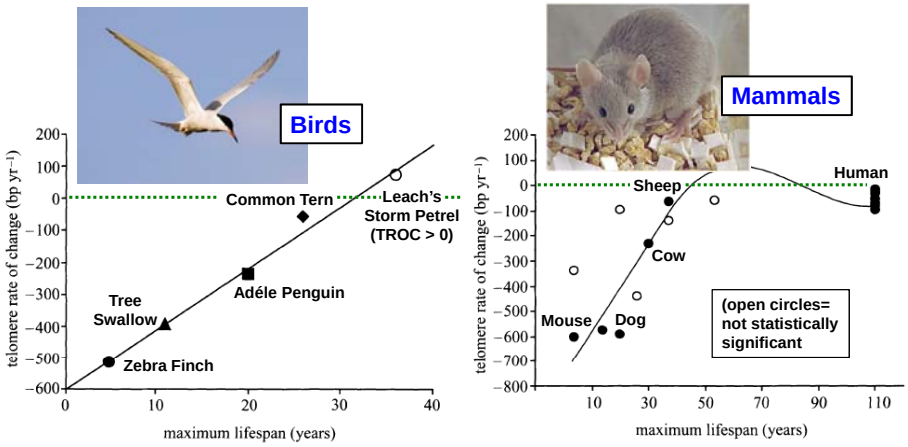


(Röhme 1981)



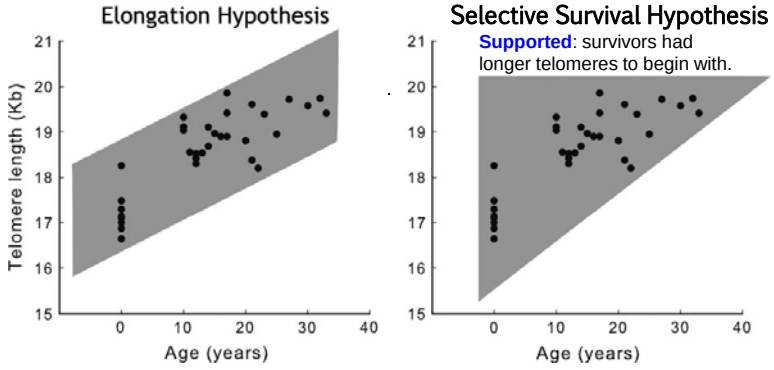
Telomere rate of change (TROC) *is less in longer-lived taxa*

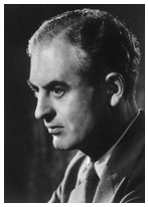
(Haussmann, Winkler et al. 2003)



Telomere *lengthening* in a long-lived bird, Leach's Storm-Petrel, *Oceanodroma leucorhoa*

(Haussmann & Mauck 2007)





III. The Mutation Accumulation Hypothesis

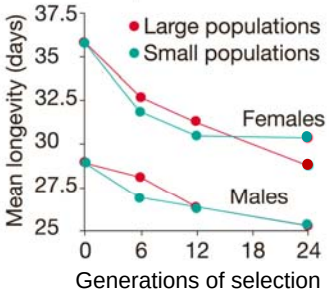
Hypothesizes that senescence is due to failure to completely repair the inevitable damage that is inflicted on tissues & organs by living (as for *Rate of Living*), but…

The cause: ***deleterious mutation*** (P. Medawar 1952)

- Deleterious mutations that are expressed *after maturity* **persist** in a population because *they reduce fitness less than early-acting deleterious mutations*.
- Their impact on fitness is an inverse function of how late they are expressed. If expression is so late in life that the individual bearing the mutation is likely to have finished breeding, the deleterious mutation is *invisible to natural selection*.
- Such late-acting deleterious mutations will **accumulate** in the population, manifesting their bad effects later in life.
- An example of such a mutation: one that reduces an organism's ability to maintain itself in good repair, e.g:
  - Germ-line mutations in genes coding for enzymes that repair DNA mismatches can cause cancer late in life.

Support for the mutation accumulation hypothesis: ***Shorter lifespans***

Musca domestica (house fly)



- Started with wild house flies.
- Each generation, adult flies were allowed to reproduce for only 4-5 days.
- ***Hypothesized*** *that any late-acting deleterious mutations would be rendered neutral in such young flies.*
- Some should therefore drift to high frequency (accumulate).
- Flies were periodically allowed to live out their natural lifespans.
- ***Result***: **Longevity declined significantly** in selected lines.

(Reed & Bryant 2000)

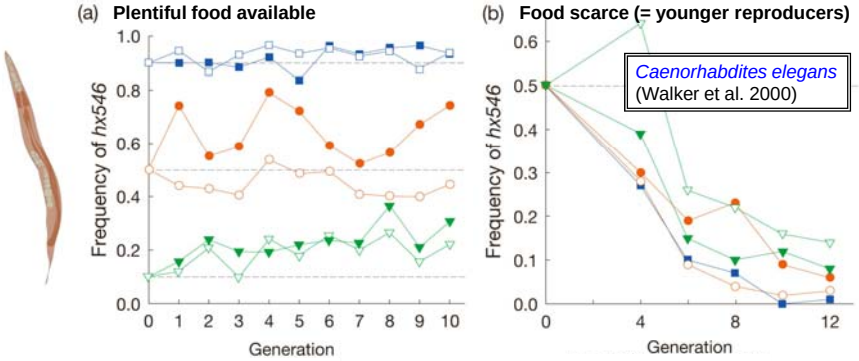
IV. The Antagonistic Pleiotropy Hypothesis (Williams 1957)

Also hypothesizes that senescence is due to failure to completely repair the inevitable damage that is inflicted on tissues & organs by living.

The cause: ***trade-offs*** *between repair and reproduction*

- When an allele affects more than one life-history trait, it is *pleiotropic*.
- And when the mutation involves a trade-off, e.g. between reproduction early in life and survival late in life, its pleiotropic effects are *antagonistic*.
- Selection will favor the allele for early maturation and early senescence, because a young age for first reproduction has high fitness and early senescence comes when reproductive value would be low anyway.
- Such an allele might cause *less* energy to be allocated for repair and *more* energy to be devoted to reproduction early in life .
- Allocating less energy to repair shortens lifespan.

Support for the antagonistic pleiotropy hypothesis: the ***age-1*** **allele**



- An allele at the *age-1* locus (*hx546*) **increases longevity**.
- Under plentiful food, this allele persists – the benefit of the longer lifespan is balanced by an equivalent cost = selective neutrality.
- But when food is scarce, the true cost of the allele is revealed.
- Rate of decline indicates fitness is <80% that of *normal* allele.
- *"Normal"* increases early fitness, with a cost later in life.

Ecological (extrinsic) mortality
and its relevance to senescence

A. Ecological mortality and ***late-acting deleterious mutations***:

1. ***Lower*** mortality means that a higher fraction of zygotes will live long enough to experience the late-life costs.
2. Therefore, such mutations are more strongly selected against.
3. Late-acting deleterious mutations will be held at lower frequency.

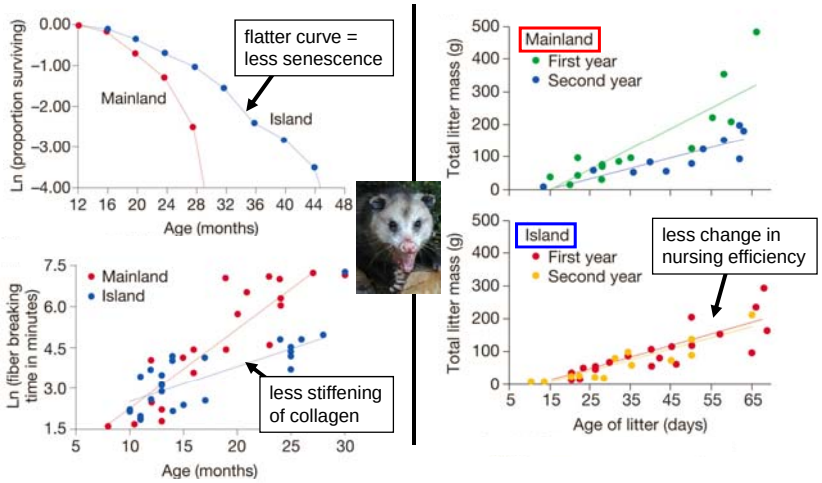
B. Ecological mortality and ***antagonistic pleiotropy***:

1. ***Lower*** mortality means that a higher fraction of zygotes will live to experience *both* the early-life benefits and the late-life costs.
2. But the *increase* in the fraction is larger for the late-life costs.
3. Therefore, such late-acting deleterious mutations are less strongly favored earlier in life.

End result: **Lower ecological mortality leads to later senescence**.

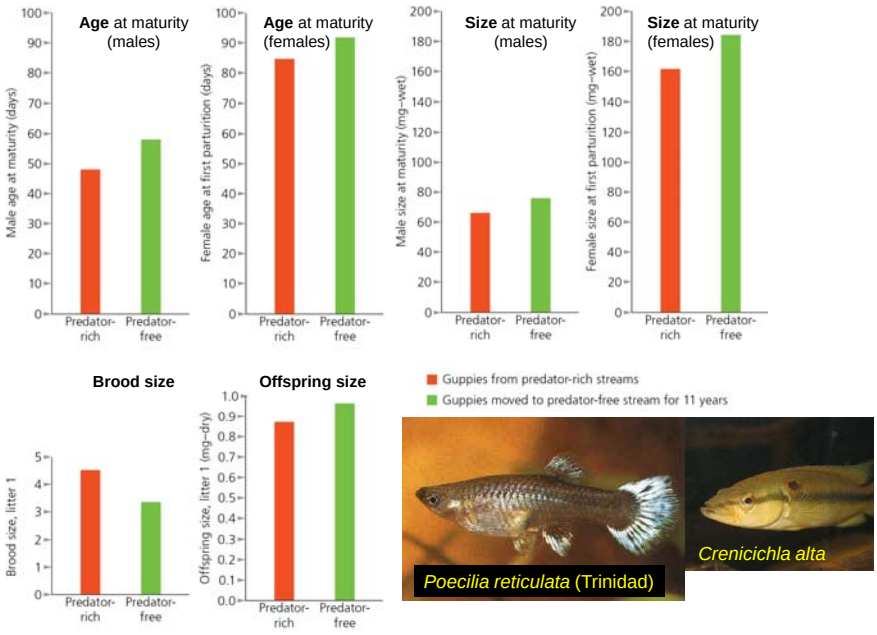
ANY EXAMPLES ?

Female **'possums on Sapelo Island** age more slowly than females on the mainland (Austad 1993)



- Sapelo Island, off Georgia, has been isolated 4000-5000 years.
- No mammalian predators & lower ecological mortality on the island (otherwise identical ecology).
- Island possums show delayed reproductive and later physiological senescence.

Life-history responses of guppies to predation (Reznick & Travis 2002)



Understanding life history traits II: Genetics

We've already mentioned some genetic aspects of life history traits:

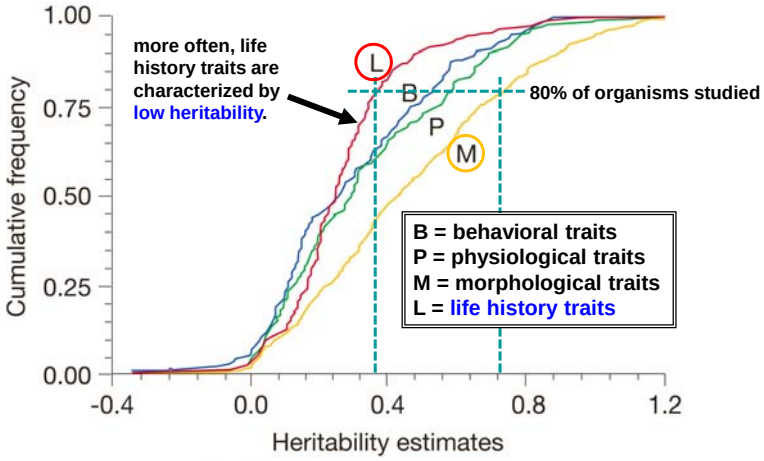
- *Positive* genetic correlations often exist between traits responsible for resource *acquisition*.
- *Negative* genetic correlations always exist between traits responsible for resource *allocation* .
- There is negative and positive *frequency dependence* of alleles controlling specific life-history traits.

Recall also that life history traits have been shown to be polygenic, meaning that quantitative genetics is the mode of analysis.

- Do life-history traits show sufficient heritability to respond to selection?
- And even if heritability is low, can phenotypic plasticity help organisms solve some types of life history conflicts?

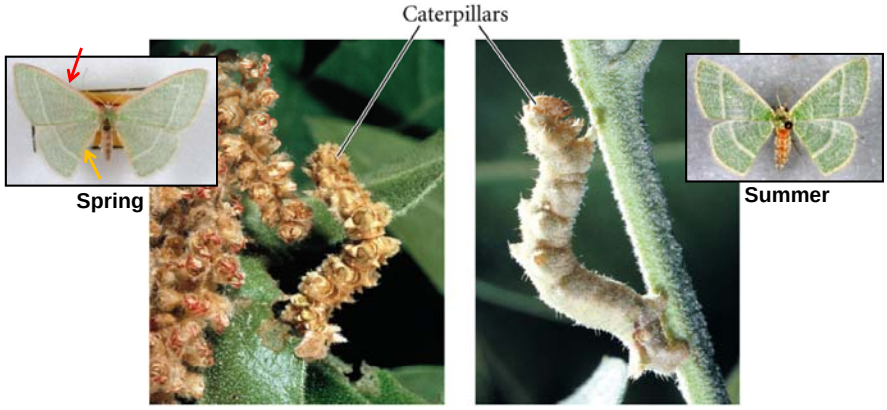
Life history traits have *lower heritabilities* than other kinds of traits

(Mousseau & Roff 1987 based on 1120 estimates)



An example of **phenotypic plasticity** in a life history trait:

The larva of the geometrid moth *Nemoria arizonaria* (Greene 1989)



Embedded in a life history, such plasticity enables an individual to alter major strategies within its life span, via genetic switches.

Example = **complete metamorphosis** (holometaboly)

Understanding life history traits III: Phylogenetics

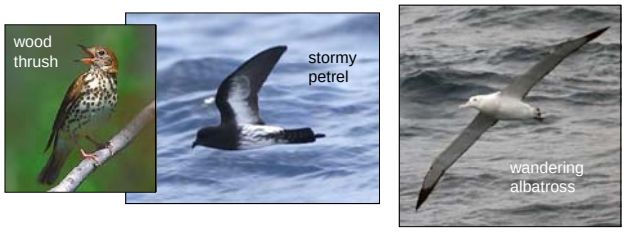
- The phylogenetic, or historical, context of life history evolution is very important but often overlooked.
- Evolutionary history can exclude certain life history adaptations in a particular lineage of organisms, even though similar adaptations are well developed in other lineages.
- In other words, evolutionary history (phylogeny) can be a major ***constraint*** on life history evolution, preventing the organism from trying out certain solutions to life history conflicts.
- The effect of phylogeny is usually expressed through ***taxonomically conservative traits***.
- Strikingly, some of these taxonomic associations of life history traits seem *independent of environmental differences*.

Some examples of phylogenetic life history constraints in birds

1. All species of ducks lay 8-10 eggs per clutch.
   - But all ducks are able to feed themselves when young (precocial), so large clutches are OK…
2. All shorebirds seem to lay about 4 eggs per clutch.
3. All hummingbirds lay 2 eggs.
4. Petrels and their relatives lay 1 egg.

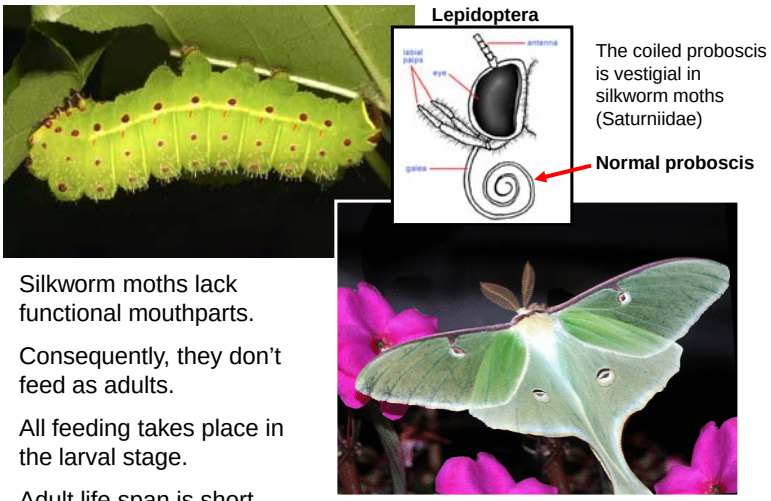
We do not have explanations for the other clutch sizes.

The single egg of petrels (Procellariiformes) is mysterious



- The typical petrel is thrush-sized; matures at 4-5 yrs old; and lives 30-40 years.
- Compare to a thrush: it's the same size as a petrel, but matures in 1 year and lives only 4-5 years. Clutch size = several eggs.
- The trait of raising one offspring per year is undoubtedly a phylogenetic constraint.
- The same limitation characterizes ***all*** petrels from the 30 g (1 oz) stormy petrel to the 10 kg (22 lb) wandering albatross.
- Long-term evolution of petrel clade of under sparse, unpredictable food supplies? **But see** Lecomte et al. 2010 on albatrosses.

Semelparity (one egg mass) is constrained by vestigial mouthparts



- Silkworm moths lack functional mouthparts.
- Consequently, they don't feed as adults.
- All feeding takes place in the larval stage.
- Adult life span is short, constraining reproduction to one episode (& one clutch).

Lepidoptera: Saturniidae – 1500 species

Final question: How many of my offspring should be male or female?

…and should that decision….

a) depend on ecology or social circumstances?
b) be fixed at birth?
c) if sequential, start as male and turn female, or vice versa?

- **Sex ratio** is an excellent example of the importance of ***frequency dependence*** in life history evolution.
- It is also an **allocation** problem: in this case, *the allocation of time and energy to different sexes*.

Mating in the cicada-killer wasp, *Sphecius speciosus*

Three possible patterns occur in nature

1. **Separate sexes** – dioecious or gonochoristic: Each individual is one sex or the other.

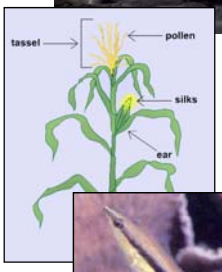
   Advantage? This system avoids the cost of maintaining both male and female organs in one individual.

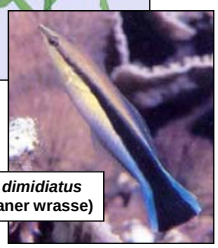
2. **Sexes together** in one individual – monoecious or hermaphroditic.

   Advantage? If you are self-compatible, you can fertilize yourself, in the event that males are hard to find.

3. **Change sex** sequentially and opportunistically.

   Advantage? If mates are hard to find, you can switch sex to the rarer (or better) of the two sexes and enjoy higher fitness.





Labroides dimidiatus **(striped cleaner wrasse)**

What should the sex ratio be?

- It could be any ratio, but the **primary sex ratio** is constrained *if we assume that parents invest equally in each sex*.
- This means that frequency-dependent selection will always favor a 50:50 ratio of males to females, no matter what the sex-determining method. Any genotype with a 50:50 individual sex ratio is an ***ESS***.
  1. Fisher (1930) showed that the sex ratio in one generation affects the reproductive success of these individuals in the following generation by ***negative frequency-dependent selection***.
  2. If fewer males are produced in one generation (say, 4 females for every male), then each male will on average fertilize 4 females, which means there will be an advantage to being a male at low densities.
  3. The result will be a selective advantage to a female that produces extra sons.
  4. That will persist until the sex ratio returns to 50:50.

Recurrent pressure for a 50:50 sex ratio likely led to the evolution of sex chromosomes (XX, XY), which ***genetically constrain*** the ratio to 50:50.

Sex-determining mechanisms

Genetic sex determination:

- ***Sex chromosomes***: homogametic (XX, ZZ) vs. heterogametic (XY, XZ, XO, ZW) sexes. *Examples* = many animals, dioecious plants.
- ***Haplodiploidy***: fertilized (diploid) vs. unfertilized (haploid) eggs. *Examples* = Hymenoptera, mites, thrips, & scale insects.
- ***Autosomes***: sex-determining factor(s) at single or multiple loci.

"Environmental" sex determination:

- Incubation ***temperature*** (turtles and crocodilians)
- Body ***size*** (some mollusks)
- ***Social*** interactions (reef fishes)
- Cytoplasmic ***parasites*** that have evolved the ability to feminize their hosts to enhance transmission (*Wolbachia* in arthropods)

(sex ratio not chromosomally constrained)

One "solution" to this has been to incorporate the sex-determining part of the *bacterial* genome into the nuclear genome (as in Isopoda – Juchault & Mocquard 1993).

Evolutionary causes of unequal sex ratios

- **Eusociality in haplodiploid insects**: Sterile workers alter the queen's 50:50 sex ratio toward more females (= sisters), due to asymmetrical relatedness of sisters to sisters (***r*** = 0.75) vs. sisters to brothers (0.25).
- **Local mate competition** (Hamilton 1967): Rather than competing with the sons of many females, parasitic wasp males compete only with one another in a local group founded by their mother.
  - Thus, the founding female's genes can be best propagated by mostly daughters, with only enough males to fertilize all of them (the other males are genetically redundant).
  - Again, *female-biased sex ratios* evolve in such systems.

Nasonia vitripennis (Pteromalidae)

Calliphora vomitoria (the host)

Unequal sex ratios can also evolve because of social interactions

Dominance relationships (Trivers & Willard 1973): Where males do the disproportionate amount of mating, as in polygynous, strongly sexually-selected organisms, the sex ratio can be affected.

- Females can establish dominance hierarchies.
- Dominant mothers produce offspring that are stronger than average, due to genetics and environment.
- Dominant mothers should produce more sons, and lower-ranked mothers should produce more daughters.

Reason? Relatively few males get to mate, and those sons of dominant mothers are the most likely to.

- It's not so important for daughters, for most females will get to breed anyway.

Condition-dependent sex allocation

The red deer of Rhum
(Clutton-Brock & Iason 1986)

Note: females must be able to assess their dominance status, *and* manipulate sex ratio.

But wait…. (Fawcett et al. 2011)

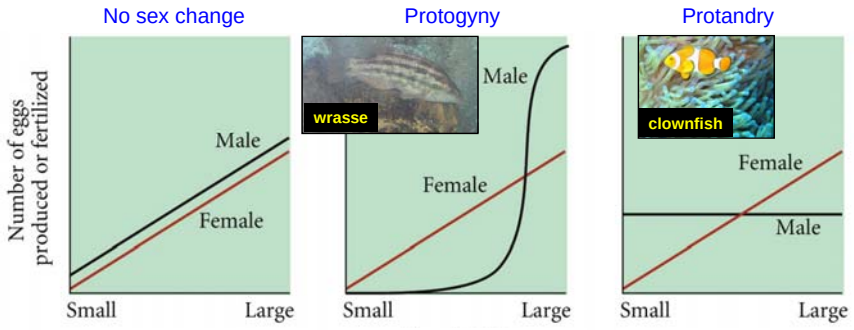
The mere ability to determine offspring sex reduces the fitness difference between females with attractive and unattractive partners.

- If mothers adaptively control offspring sex in relation to their partner's attractiveness….
- …**sexual selection is weakened and male ornamentation declines**.
- The evolution of conspicuous male ornaments favors sex-ratio adjustment, but this conditional strategy then undermines the very same process that generated it, eroding sexual selection.
- Consequently, the most elaborate sexual displays should be seen in species with little or no control over offspring sex.

Tuatara (New Zealand): temperature-based sex determination

The evolution of sex change in sequential hermaphrodites

(Ghiselin 1969; Warner 1984)



Reproductive success increases equally with body size in both sexes.

Male reproductive success increases more steeply with size than female success.

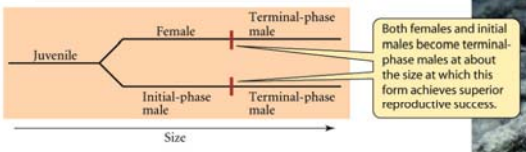
Female → Male

Female reproductive success increases more steeply with size than male.

Male → Female

*In sequential hermaphrodism, organisms should be born into the sex that **loses less** by being small or young.*

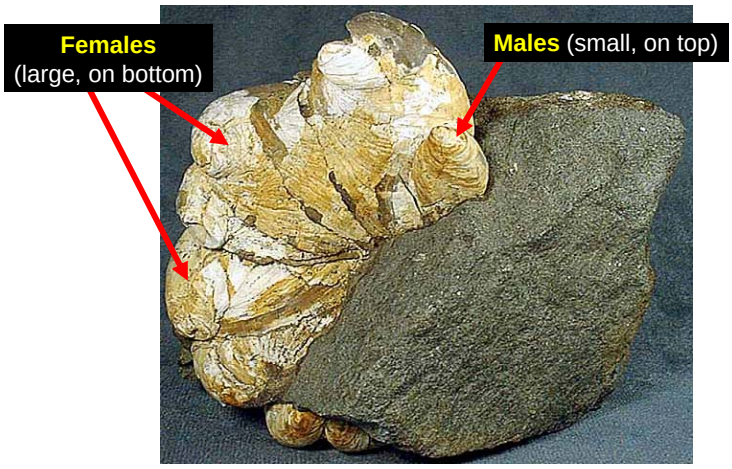
Protogyny (“female first”) in bluehead wrasse



Thalassoma bifasciatum

- Initial-phase males resemble females and spawn in groups.
- Initial females later become brightly colored “terminal phase males,” defending territories.
- Sex-change occurs at the same *critical size* at which initial-phase males become terminal-phase males.

Protandry (“male first”) in the slipper limpet, *Crepidula fornicata*



(enough life history …)