Ecology and evolution of life histories

1) Life history traits

2) Trade-offs between life history traits

3) The evolution of reproductive value

4) *r* and *K* selection

5) Constraints on life history evolution

6) Mortality and the evolution of senescence

7) Example exam questions

1) Life history traits

A continuum of life history variation is present in nature between the extremes of semelparity (breeds once, dies) and iteroparity (breeds repeatedly). A major goal is to understand the causes of this variation.

Pacific salmon: breed in a single suicidal burst at an age of 2-6 years.

Agaves: Produce only leaves for up to 15 years, then sends up a flowering stalk and sets fruit, dies.

Chickadee: Breed as one-year olds, continue until they die (~50% mortality/yr)

Oak trees: High adult survival; start breeding at age 3 years, producing only a few acorns, then stepping up acorn production until they produce thousands/yr.

https://en.wikipedia.org/wiki/Sockeye_salmon

1) Life history traits

What are life history traits?

They are essentially synonymous with fitness components, eg:

survival / longevity
fecundity
offspring size
age at first reproduction

Definition: *A trait of an individual is a life history trait if, holding all other traits constant, a correlation remains between the trait and fitness.*

Life history theory treats fitness components as measurable traits of individuals that affect contribution to future generations (fitness).

1) Life history traits

These considerations imply that an ideal organism would evolve to maximize all components of fitness.

It would breed immediately upon birth, produce huge numbers of large offspring at each breeding attempt, and continue to do so for the duration of its enormously long life span (Darwinian daemon).

In light of this concept, the observed life histories of organisms are incomprehensible. Why wait 15 years to breed?

1) Life history traits

The main hypotheses to explain this conundrum involve constraints:

1) Physical constraints on rates of resource acquisition, growth

2) Trade-offs between life history traits (higher fecundity might reduce survival)

These considerations pose two of the most important questions in the study of life histories:

1. What life histories are feasible?

2. What circumstances favour the evolution of a greater reproductive effort at the expense of survival, and what favours a lesser reproductive effort?

2) Trade-offs between life history traits

Types of evidence for the presence of trade-offs:

1. Comparisons among species

2. Phenotypic correlations

3. Genetic correlations

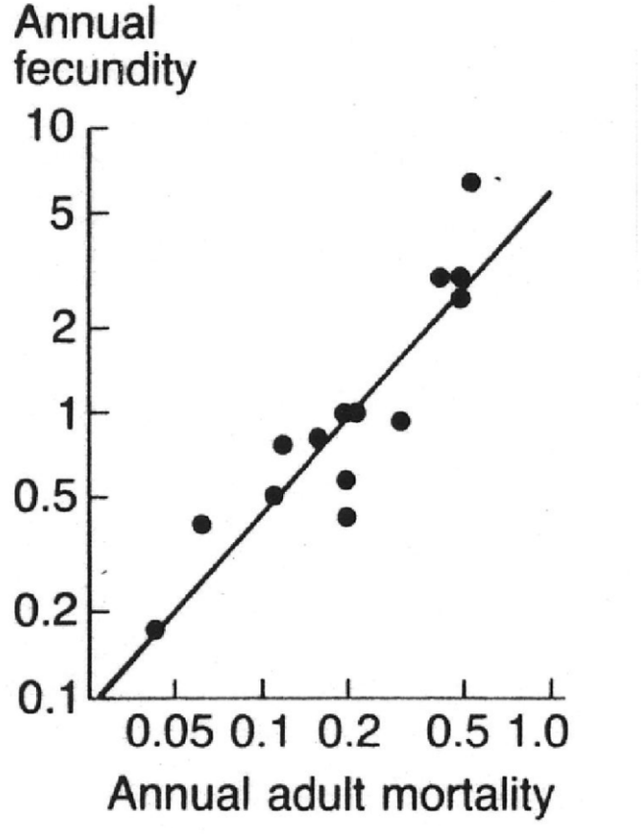
4. Manipulations of reproductive effort

5. Selection experiments (covered in a later section)

2) Trade-offs between life history traits

Evidence from comparisons among species

http://www.mountaininterval.org/photos/antarctica/highlights/pages/18-roll/15-falklands-steeple-albatross-and-chick.html



Relationship between annual fecundity and adult mortality in several populations of birds ranging from albatross (low) to sparrow (high). (From data in Ricklefs 1977).

2) Trade-offs between life history traits

Evidence from phenotypic correlations

Number of inflorescences per plant in an annual grass

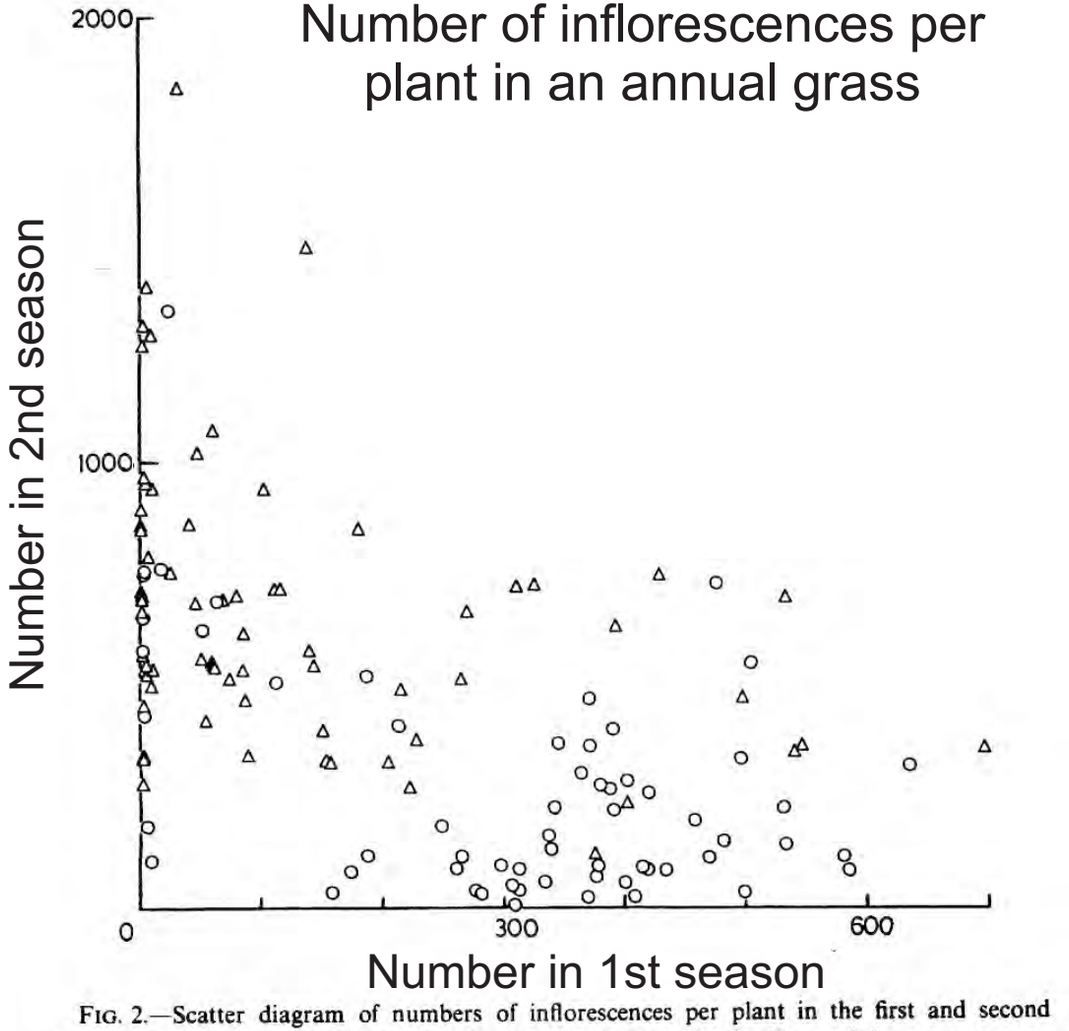


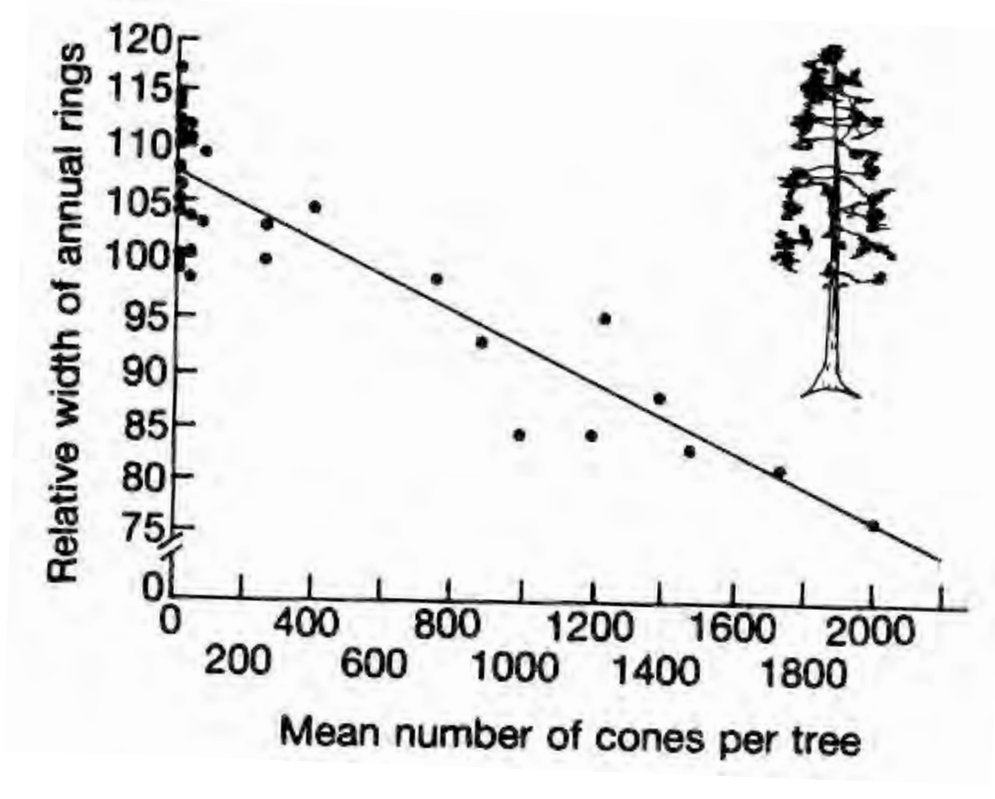
FIG. 2.—Scatter diagram of numbers of inflorescences per plant in the first and second seasons. Each point is the mean for a family; O = opportunist families and Δ = pasture ones. Only families containing at least one survivor at the end of the experiment are used ($r_s < .001$).

http://www.missouriplants.com/Grasses/Poa_annua_page.html

2) Trade-offs between life history traits

Evidence from phenotypic correlation

pine trees



2) Trade-offs between life history traits

Evidence from phenotypic correlation

Mean annual reproductive performance (± SE) of female song sparrows that did or did not survive to breed again (Smith 1981).

Status	N	No. nesting attempts	Total no. eggs laid	Total no. young raised (6 days)	Total no. young raised (30 days)
Survived	71	2.52 ± 0.08	8.52 ± 0.29	5.62 ± 0.25	3.82 ± 0.22
Did not survive	55	2.47 ± 0.11	7.62 ± 0.43	4.69 ± 0.26	2.98 ± 0.24

song sparrows

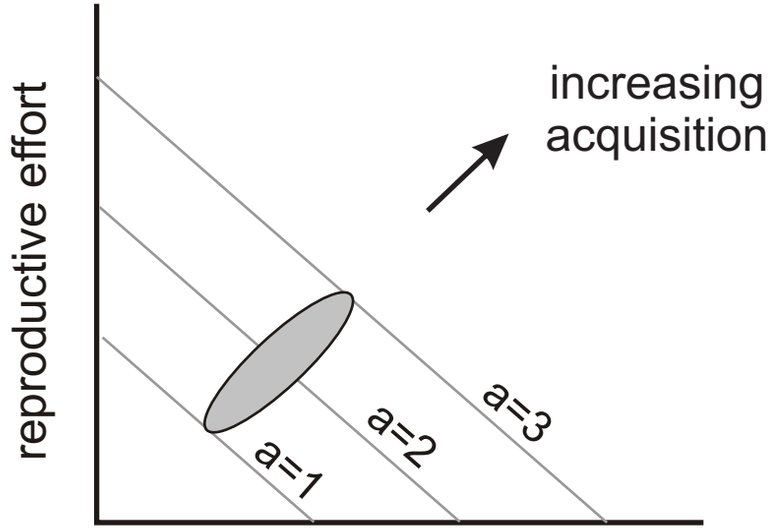
Jeff Foott. http://animal.discovery.com/guides/wild-birds/s-y/song-sparrow.html

2) Trade-offs between life history traits

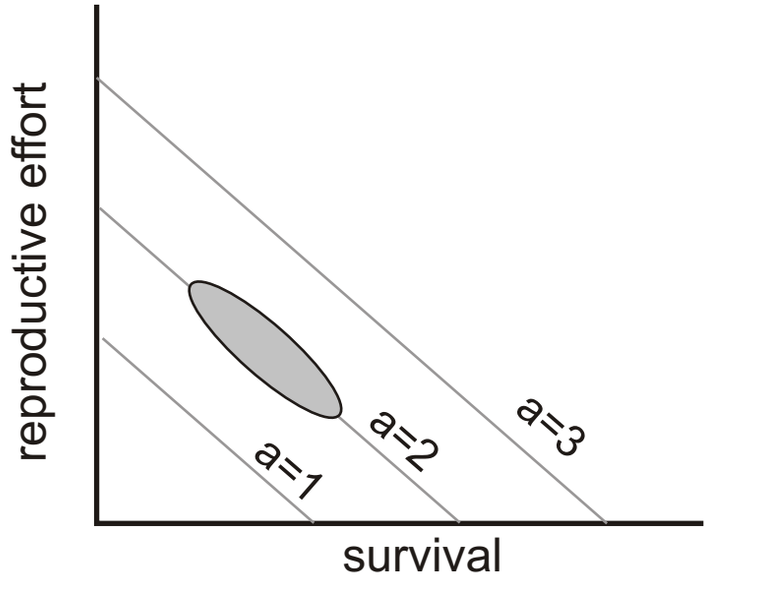
Problem: phenotypic correlations should depend on type of variation present in the population

Variation in the population indicated by shaded ellipse.
a = 1, 2, 3 refers to increasing “acquistion”.

When individuals vary mostly in “acquisition”
(no trade-off is apparent)



When individuals vary mostly in “allocation”
(trade-off is apparent)

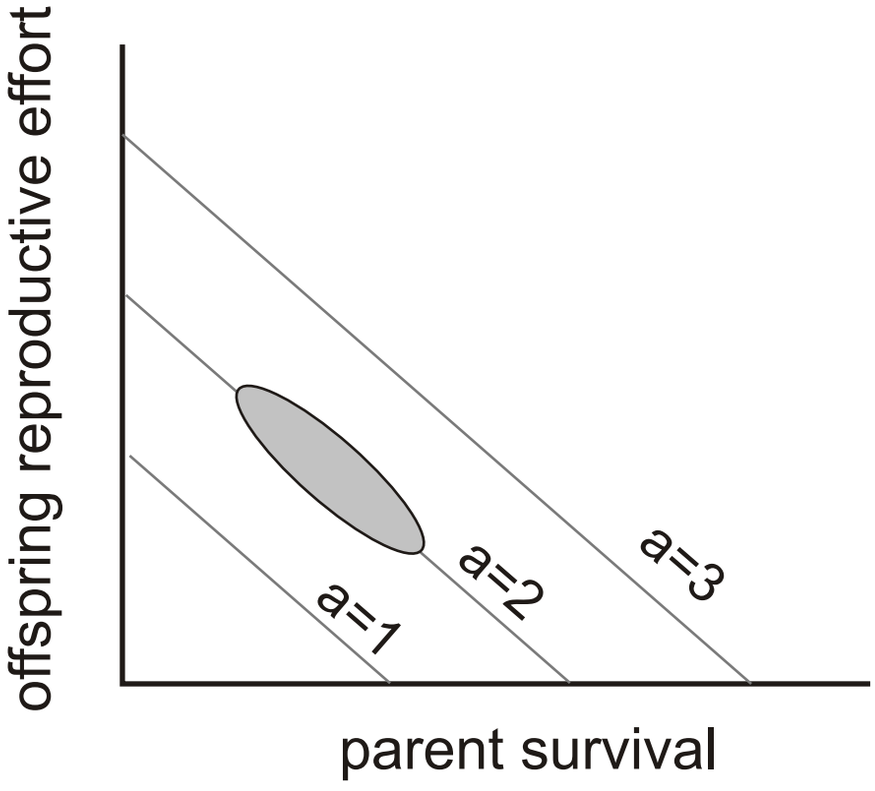


2) Trade-offs between life history traits

Evidence from genetic correlations

Logic: there should be little or no genetic variation in acquisition.

Individuals should therefore vary mostly in “allocation” in which case trade-offs should be evident as negative genetic correlations.



2) Trade-offs between life history traits

Evidence from genetic correlations

Result: Genetic correlations between life history traits are usually positive too! They are less frequently positive than are phenotypic correlations. But that so many were found to be positive was unexpected.

Conclusion: Trade-offs are difficult to measure using variation within populations.

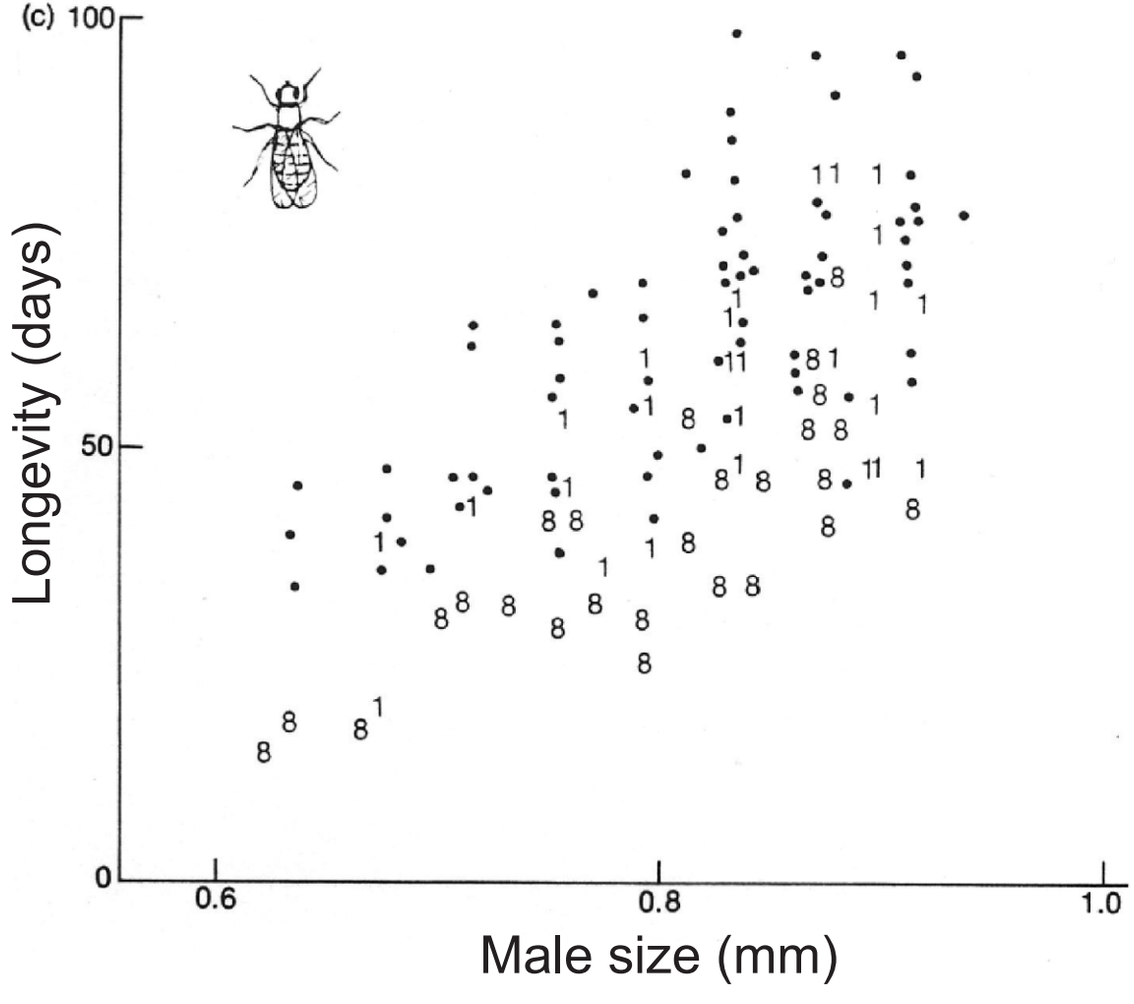
Table 3.7 A comparison of genetic and phenotypic correlations for life history traits (after Roff and Mousseau 1987)

	Genetic correlation		
	Positive	Negative	All studies
Phenotypic correlation:			
Positive	16	8	24
Negative	1	5	6
All studies	17	13	

2) Trade-offs between life history traits

Evidence from manipulation of reproductive effort

(c)The longevity of male fruit-flies (*Drosophila melanogaster*) generally increases with size (thorax length). However, longevity was reduced in males provided with one virgin and seven mated females per day ('1') compared with those provided with eight mated females (•), because of the increase in courtship activity, and reduced still further in males provided with eight virgins per day ('8'). (After Partridge & Farquhar, 1981.)



3) The evolution of reproductive value

Definition:

Reproductive value is the expected contribution to future population growth of an individual of a given age, given that it has survived to that age.

Alleles that increase reproductive value will increase in frequency over time.

Life history traits should evolve such that they tend to maximize an individual's reproductive value.

This means that we can use the concept of reproductive value to predict how life histories should evolve, assuming that there are trade-offs among them.

3) The evolution of reproductive value

If an individual has survived to age x, then its reproductive value is

$$RV_x = m_x + \sum_{t=x+1}^{\infty} m_t \, S_{x \to t} \, N_{(x)}/N_{(t)}$$

reproductive value at the current age x

reproductive effort at age x

residual reproductive value

RV at age x is the sum of current reproductive output plus reproductive effort *m* at future ages x+1, x+2, x+3, ... discounted by *S*, the probability of surviving to each future age (and by changes in population size).

3) The evolution of reproductive value

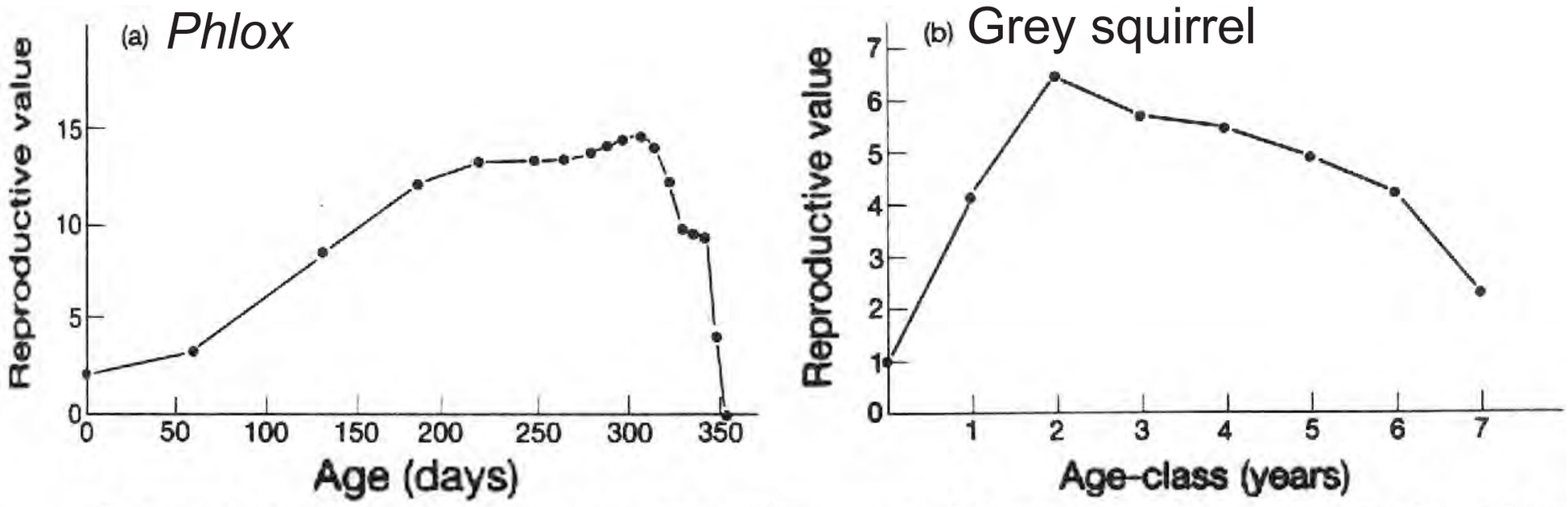
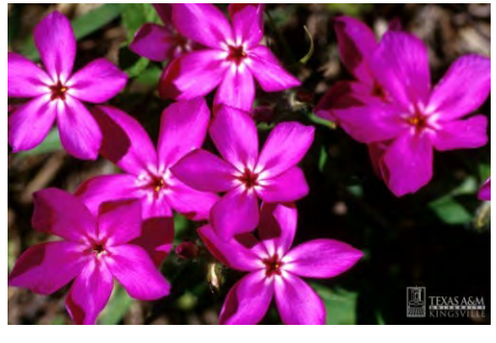


Figure 14.4. Reproductive value generally rises and then falls with age, as explained in the text. (a) The annual plant *Phlox drummondii*. (After Leverich & Levin, 1979). (b) Female grey squirrels. (After Barkalow *et al.*, 1970. From Charlesworth, 1980.)



http://www.tamuk.edu/TAIC/wildFlowers.html

3) The evolution of reproductive value

If an individual has survived to age x, then its reproductive value is

$$RV_x = m_x + \sum_{t=x+1}^{\infty} m_t \, S_{x \to t} \, N_{(x)}/N_{(t)}$$

reproductive value at the current age x

reproductive effort at age x

residual reproductive value

The formula can be used to make predictions about how life histories should evolve under different circumstances. For example:

How should current reproductive effort evolve if mortality is raised on every age class?

How should reproductive value evolve if future reproductive effort is expected to be much higher than current reproductive effort, because of future growth of body size?

4) *r* and *K* selection

Table 8.2. Some of the Correlates of *r* and *K* Selection

	r selection	*K* selection
Climate	Variable and/or unpredictable; uncertain	Fairly constant and/or predictable; more certain
Mortality	Often catastrophic, nondirected, density independent	More directed, density dependent
Survivorship	Often Type III	Usually Types I and II
Population size	Variable in time, nonequilibrium; usually well below carrying capacity of environment; unsaturated communities or portions thereof; ecologic vacuums; recolonization each year	Fairly constant in time, equilibrium, at or near carrying capacity of the environment; saturated communities; no recolonization necessary
Intra- and interspecific competition	Variable, often lax	Usually keen
Selection favors	1 Rapid development 2 High maximal rate of increase, r_{max} 3 Early reproduction 4 Small body size 5 *Single reproduction* 6 Many small offspring	1 Slower development 2 Greater competitive ability 3 Delayed reproduction 4 Larger body size 5 *Repeated reproduction* 6 Fewer larger progeny
Length of life	Short, usually less than a year	Longer, usually more than a year
Leads to	Productivity	Efficiency
Stage in succession	Early	Late, climax

Source: After Pianka (1970).

Now largely abandoned by life history theorists.

Confused descriptions of life history syndromes with selection pressures. They are labels more than hypotheses.

Has drawn attention to the effects of population density on life histories.

But age-specific models make more useful predictions about the evolution of life histories in natural environments and experiments.

5) Constraints on life history evolution

Other constraints on life history evolution have been suggested (in addition to the fundamental constraints on life history evolution resulting from trade-offs).

1) Phylogenetic constraints

Traits shared by species of specific lineages that show little variation and propensity to change despite apparent variation in the ecological environments the species inhabit:

About 600 species of geckos: virtually all lay 2 eggs/clutch

All hummingbirds lay 2 eggs, tropics to temperate zone

All barnacles have 6 naupliar stages

2) Body size

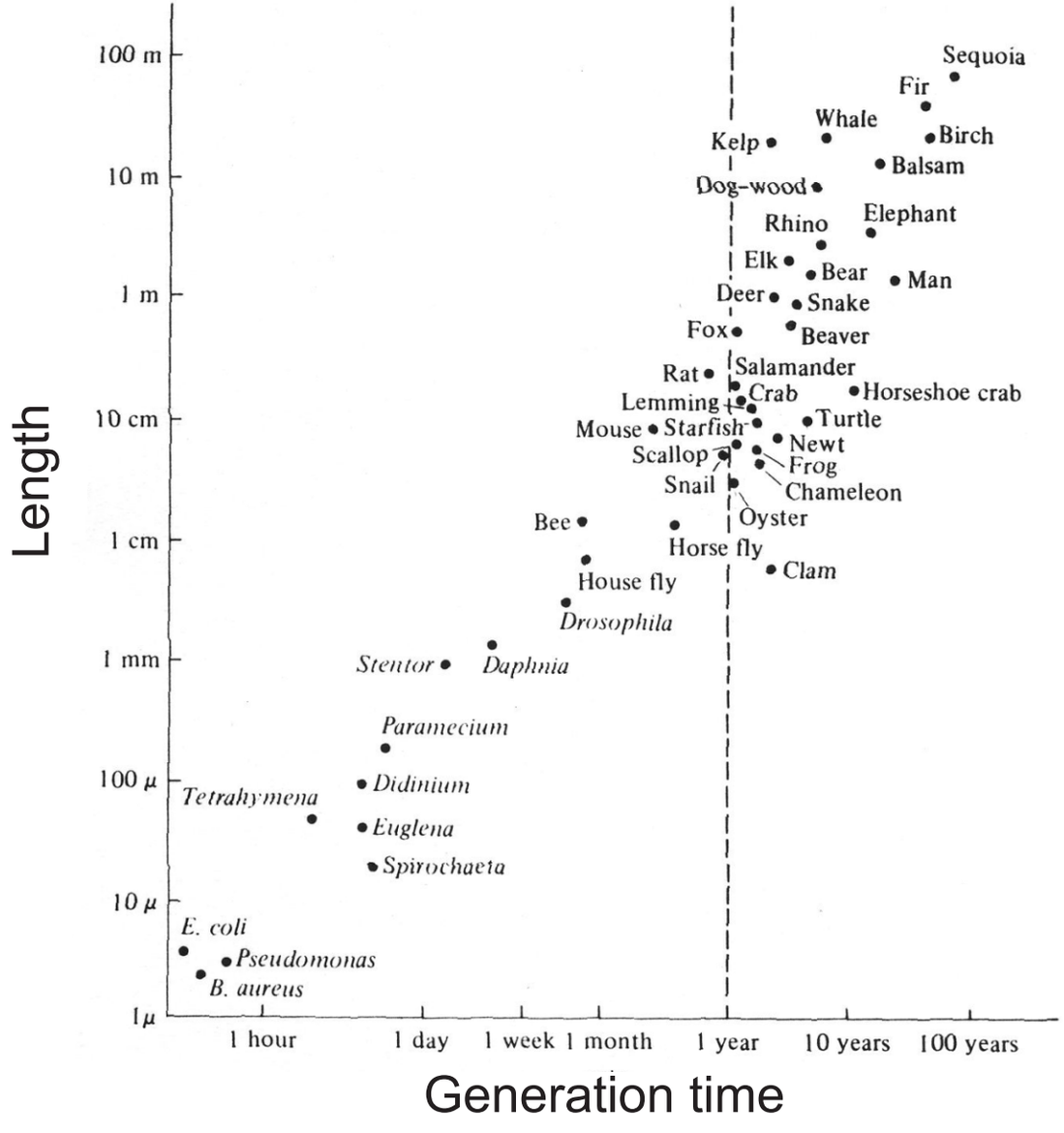
Life history traits are said to be dictated strongly by body size

5) Constraints on life history evolution

Body size strongly predicts life history variation: is it a constraint?

Figure 7.25. Log-log plot of organism length against generation time for a wide variety of organisms. [From John Tyler Bonner, ***Size and Cycle: An Essay on the Structure of Biology*** (Copyright © 1965 by Princeton University Press), Fig. 1, p. 17. Reprinted by permission of Princeton University Press.]

http://nintharticle.com/solar3.html



5) Constraints on life history evolution

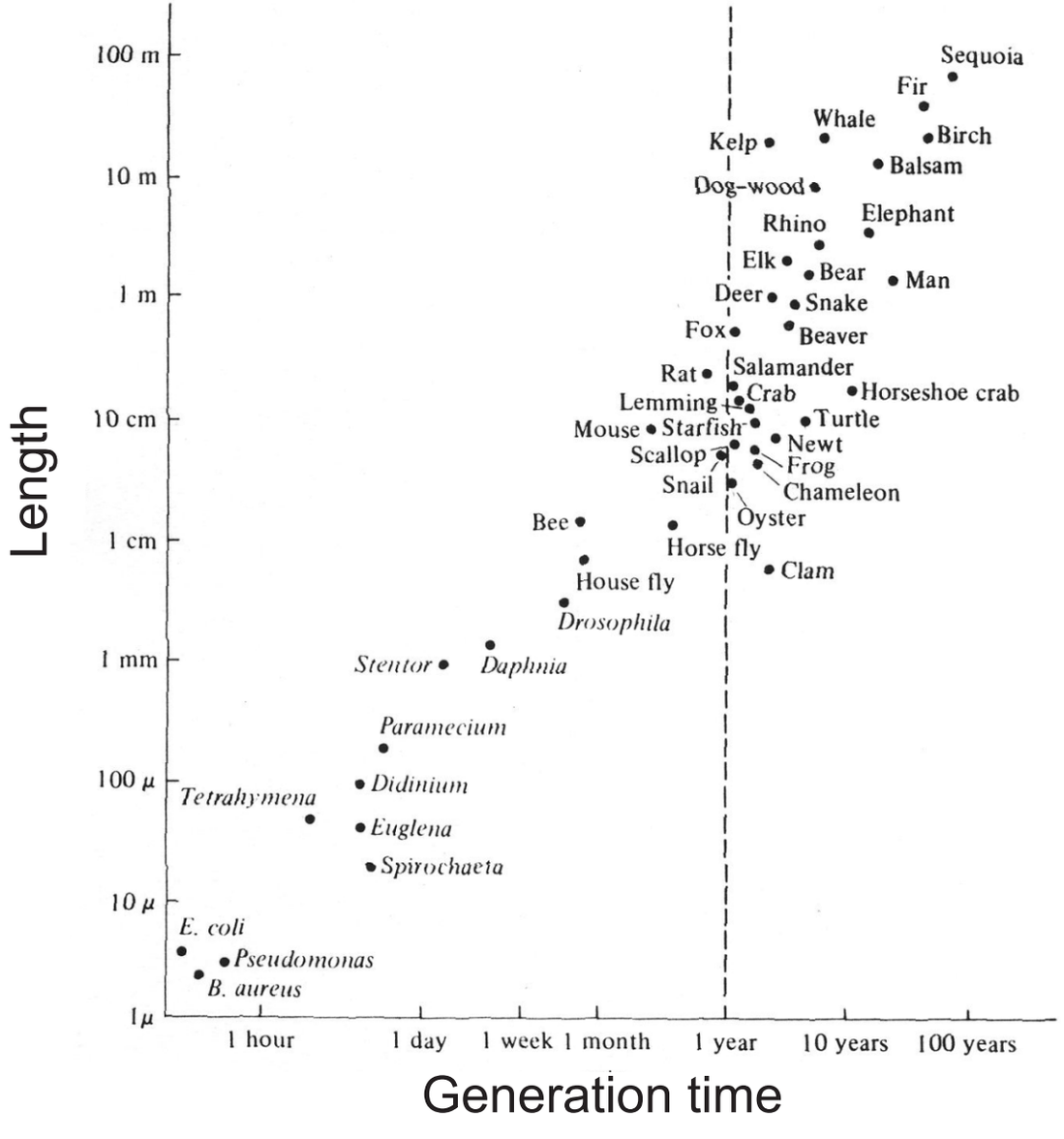
Body size strongly predicts life history variation: is it a constraint?

But:

1. There is plenty of scatter, and so plenty of life history variation is possible for a given body size.

2. All heritable differences in life history must lie in their differences in traits. Beak size is also (remember Galapagos finch example). Body size is a trait that has a large effect overall.

3. Best to think of body size not as a constraint but as an important trait that underlies life history evolution.



6) The evolution of senescence

Senescence: the physiological deterioration that accompanies aging, and leads to accelerated mortality and decline in reproductive performance.

Why does it occur?

Hypotheses:

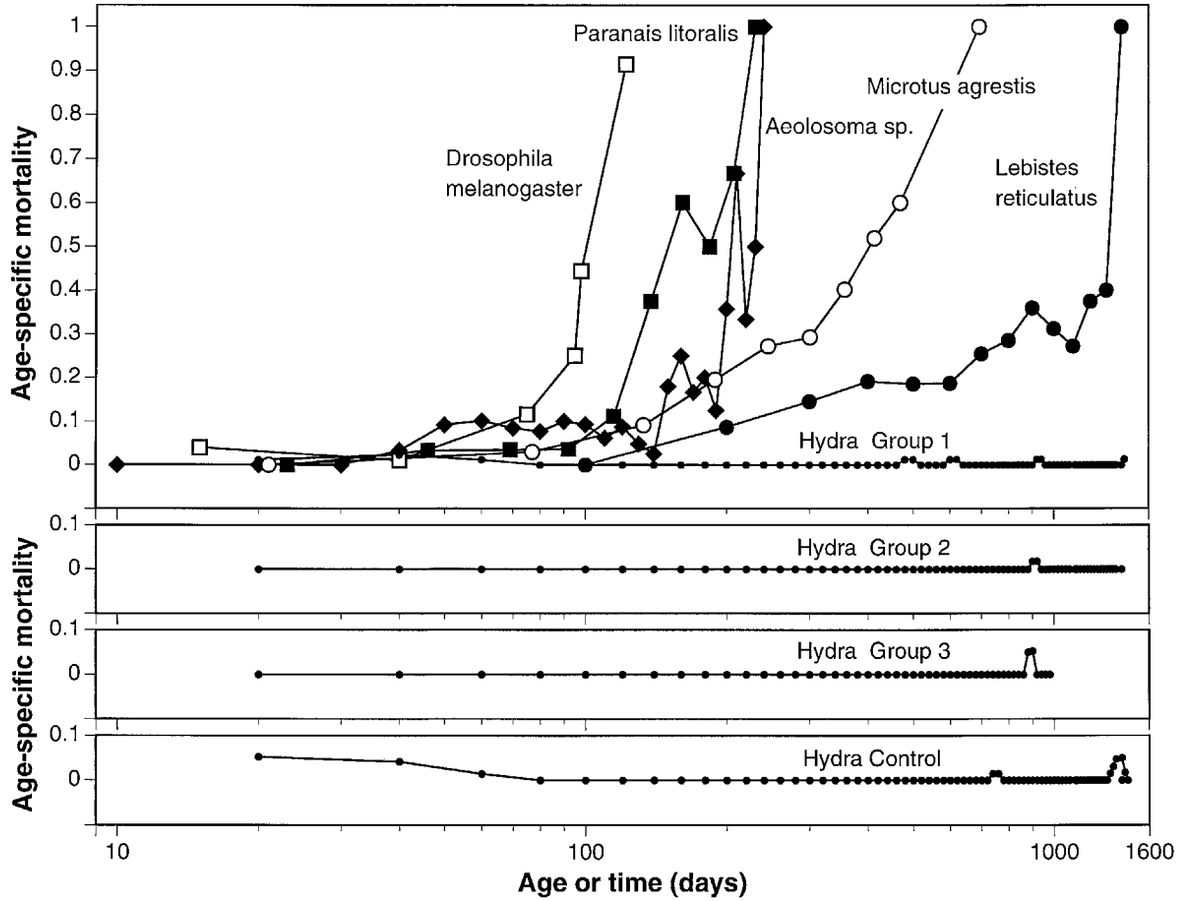
1) A fundamental constraint: senescence is an inevitable consequence of wear and tear. We are like washing machines. (Almost certainly incorrect: hypothesis does not account for variation among organisms in the rate of aging. Aging is evolvable.)

2) Senescence evolves because the strength of selection declines with advancing age. Two main ideas:

   a) Mutation-accumulation hypothesis
   b) Antagonistic pleiotropy hypothesis

6) The evolution of senescence

Hydra are apparently immortal



Hydra can reproduce sexually but usually reproduces by asexual budding. It can regenerate a new individual from almost any part of the organism---it lacks clear separation of germ line and soma (Kirkwood and Austad 2000, *Nature)*

FIG. 1. Age-specific mortality rates. Abscissa (logarithmic scale) represents age (groups 1, 2, and 3) or time (control). Animals that were accidentally killed during handling were excluded (control: three; group 1: five; Group 2: two). Data on *Drosophila melanogaster*: Miquel, *et al*, 1976; *Microtus agrestis*: *Leslie and Ranson, 1940; Lebistes reticulatus* Comfort, 1961; *Paranais litoralis* and *Aeolosoma* sp.: my own experiments.

Martínez et al (1998) *Experimental Gerontology*

6) The evolution of senescence

On immortality...

“The presence or absence of ageing is sometimes attributed to the presence or absence of sexual reproduction, but this is erroneous. It is the ***distinction between soma and germ line*** *(a common but not universal correlate of sex) that holds the key.”*

Kirkwood and Austad (2000) *Nature*

6) The evolution of senescence

On immortality...

“The transition from multicellularity in colonial protozoans to cellular differentiation some 500-1000 million years ago marked a crucial point for the evolution of the metazoan soma. Some cells lost their capacity to produce a new individual; they became committed to functions other than reproduction. Free from the need for immortality, the metazoan soma became liable to undergo senescence. .. Once the task of passing totipotential cells, either sexually or asexually, to the next generation has been accomplished, the soma may suffer internal physiological deterioration without fitness consequences...”

Martínez and Levinton (1992) *PNAS*

6) The evolution of senescence

Medawar's test tube thought experiment

Imagine a population of 1000 test tubes and a monthly breakage rate of 10%.

http://www.soulsofdistortion.nl/dna9.html

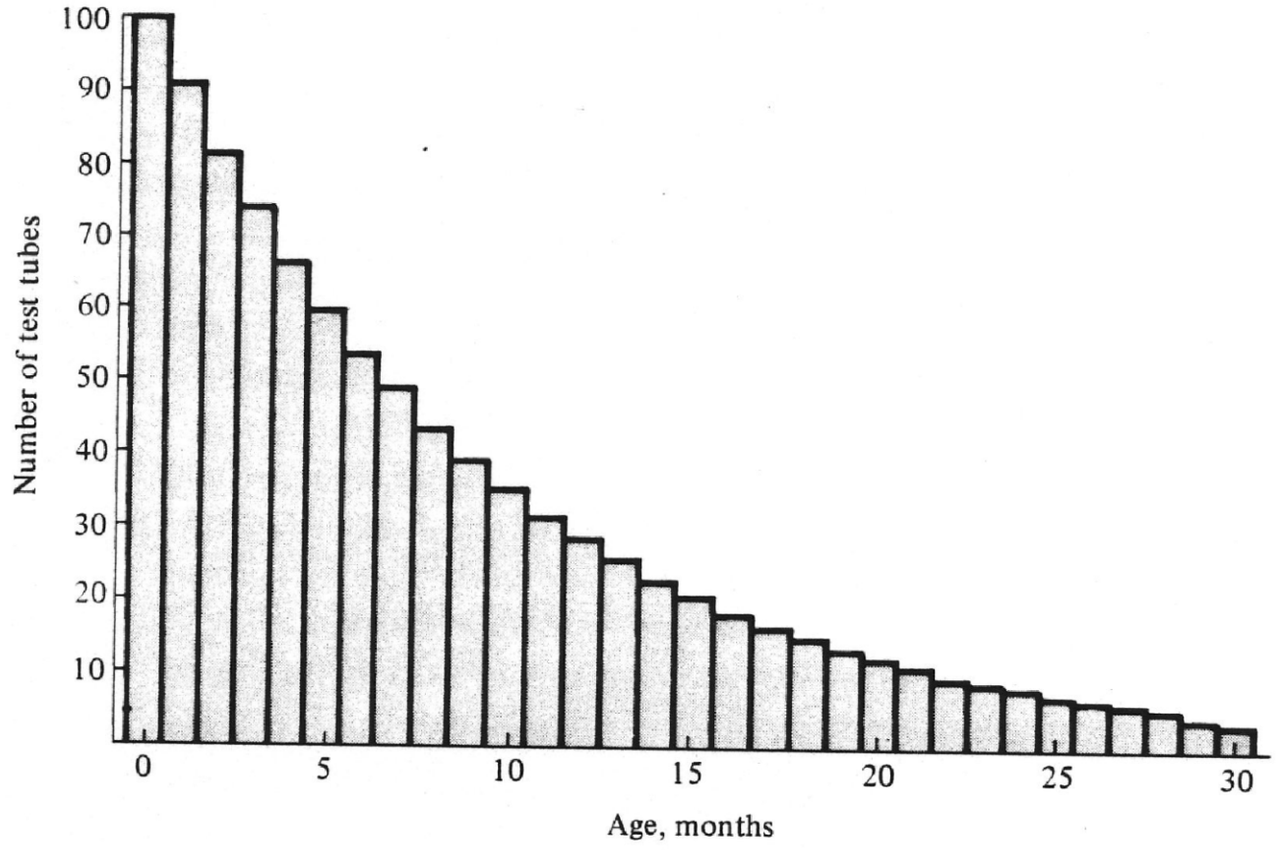


Figure 7.22. Stable age distribution of test tubes with a 10 percent breakage rate per month. Because very few test tubes survive longer than 30 months, the distribution is arbitrarily truncated at this age.

6) The evolution of senescence

Medawar's test tube thought experiment

Individual test tubes are potentially immortal, yet few survive beyond 2 or 3 years ("externally imposed mortality").

Assume that each tube of the 900 remaining each monthis assigned a fecundity of 1/9, bringing the population back to 1000.

Neither fecundity nor survival is age dependent. Reproductive value is constant. No senescence.

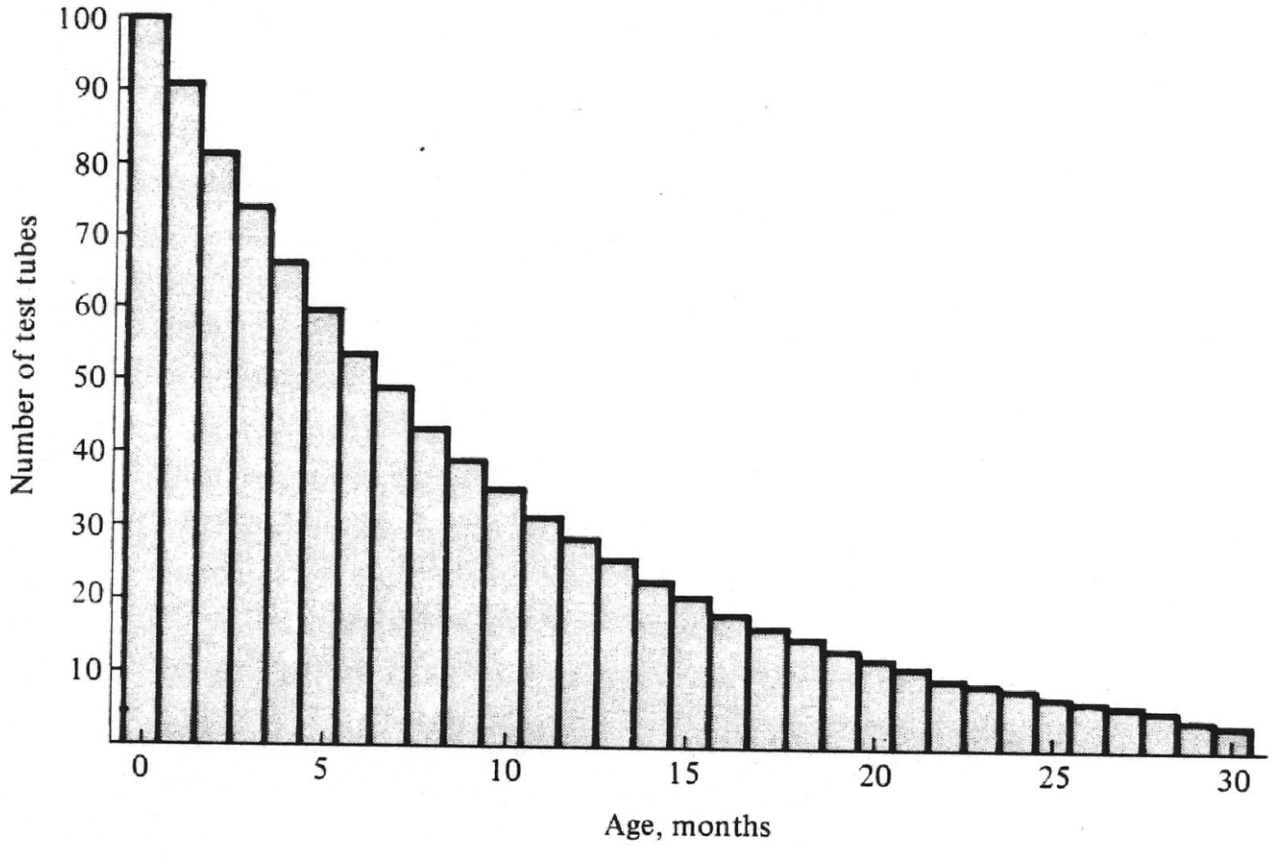


Figure 7.22. Stable age distribution of test tubes with a 10 percent breakage rate per month. Because very few test tubes survive longer than 30 months, the distribution is arbitrarily truncated at this age.

6) The evolution of senescence

Mutation accumulation hypothesis

Assume test tubes have a gene. Consider a mutation "brittle" at the gene that makes its bearer easier to break (die). Natural selection will steadily remove the mutation from the population, because of the survival disadvantage.

6) The evolution of senescence

Mutation accumulation hypothesis

Assume test tubes have a gene. Consider a mutation "brittle" at the gene that makes its bearer easier to break (die). Natural selection will steadily remove the mutation from the population, because of the survival disadvantage.

Now, consider a second gene that controls the timing of expression of the first gene. Mutations at this gene that cause "brittle" to be expressed starting at an early age will be selected against too.

Mutations that cause "brittle" to be expressed at a late age will also be selected against, but at a slower rate, because few individuals live that long.

The result of this process is the accumulation (over evolutionary time) of deleterious mutations in the population expressed at late ages.

6) The evolution of senescence

Antagonistic pleiotropy hypothesis

Consider a gene that has positive effects on reproduction early in life but deleterious effects on survival later in life (antagonistic pleiotropy).

The declining force of selection with advancing age means that such a gene would have a net positive effect on fitness. The soma becomes disposable.

6) The evolution of senescence

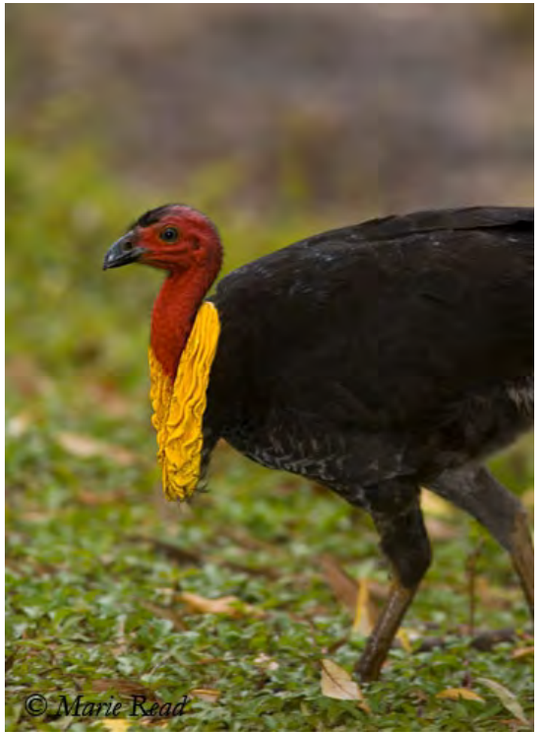
Both hypotheses predict that senescence will evolve most readily in environments with high rates of externally imposed mortality.

This could help to explain the variation seen among species in rates of senescence in the wild.

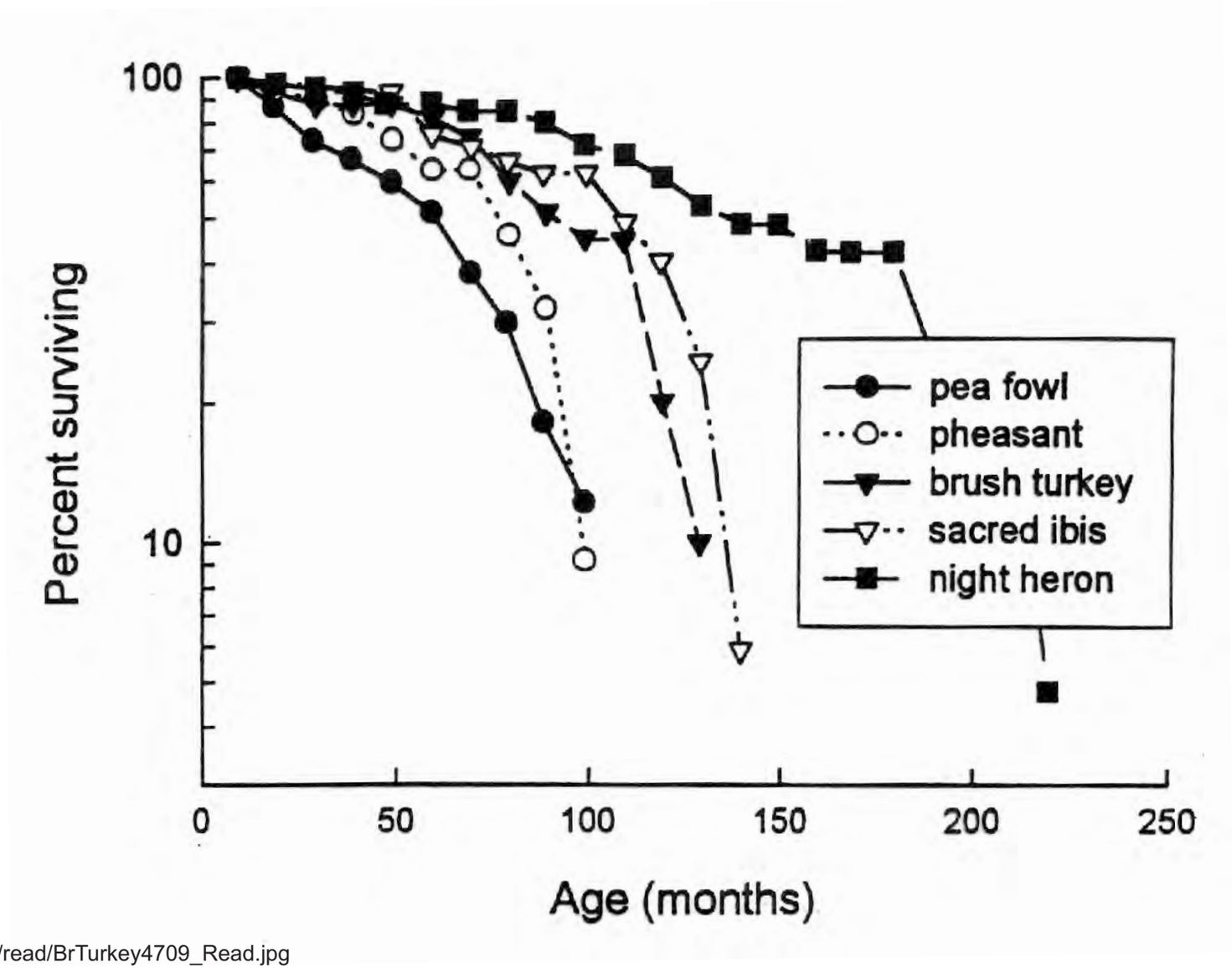
Mathematical theory reveals that subtle assumptions are required to ensure the validity of this expectation. For example, population growth must be density dependent rather than exponential (Day and Abrams 2020).

6) The evolution of senescence

Evidence from comparative study



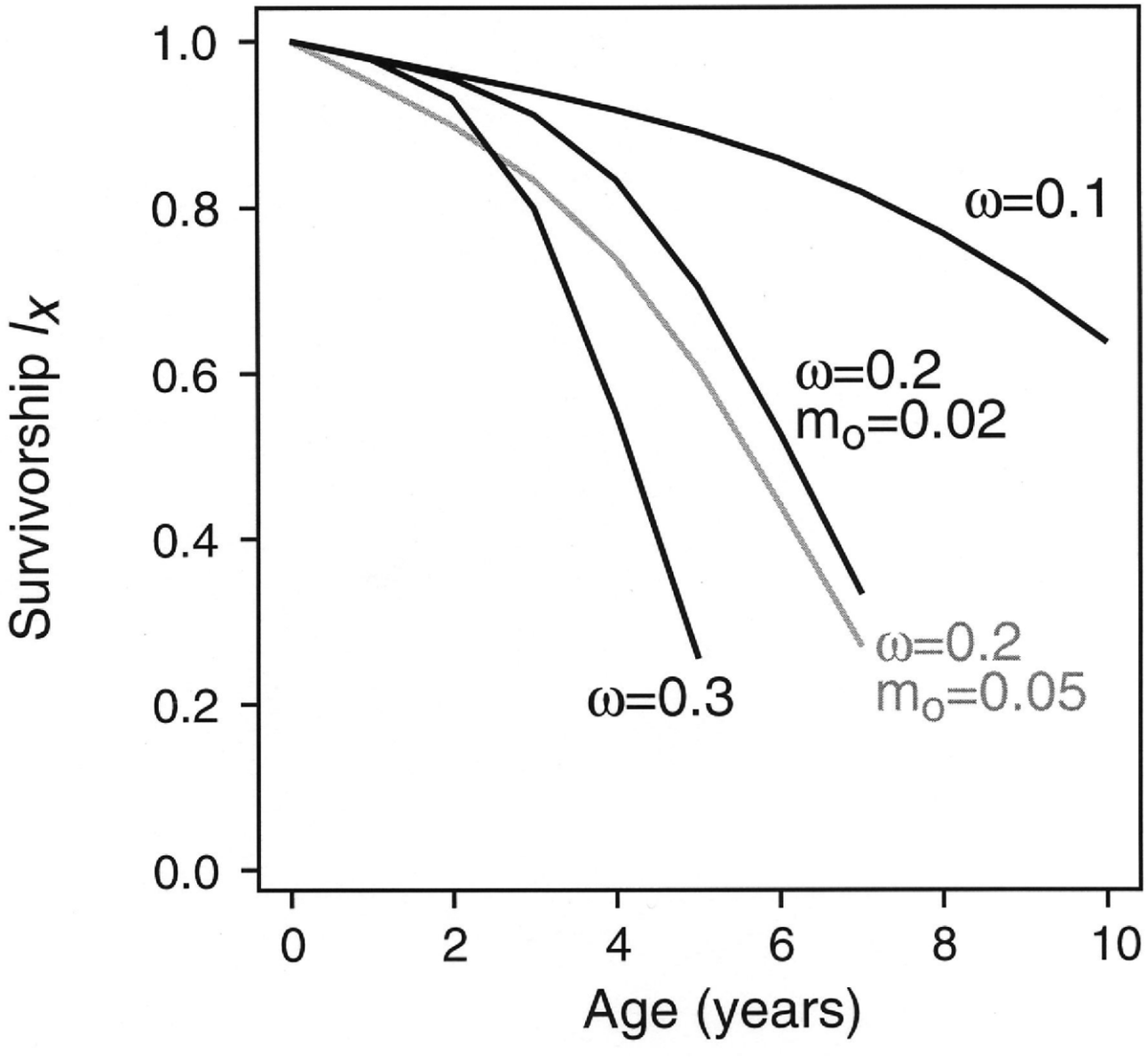
http://www.birds.cornell.edu/AllAboutBirds/bp/mread/read/BrTurkey4709_Read.jpg



Ricklefs (1998) Am Nat

6) The evolution of senescence

Evidence from comparative study



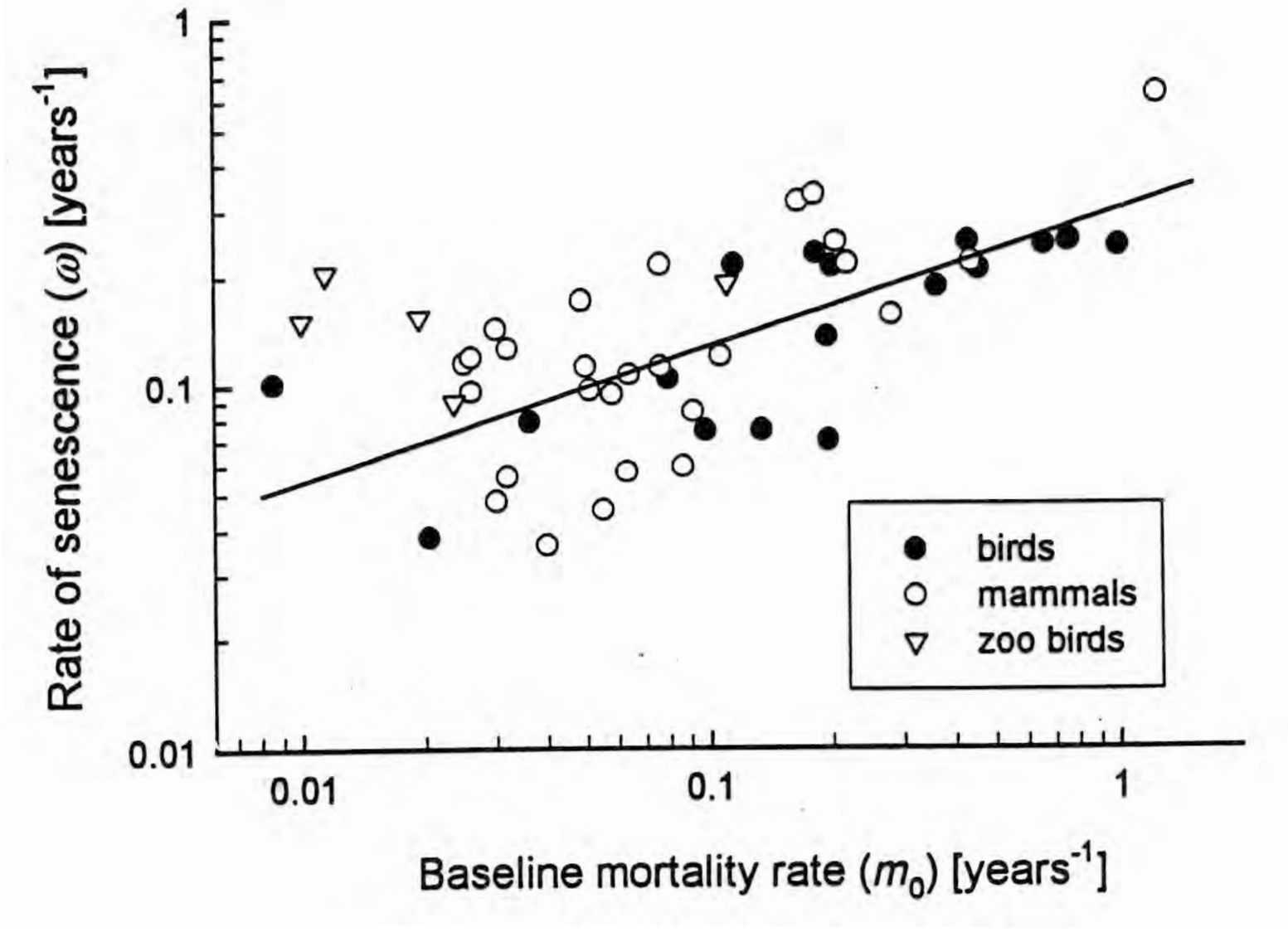
$$l_x = \exp(-m_o x - \frac{(\omega\ x)^{\beta+1}}{\beta+1})$$

$$\beta = 3$$

6) The evolution of senescence

Evidence from comparative study

http://www.photoseek.com/animals.html



Ricklefs (1998a)

6) The evolution of senescence

Evidence from a selection experiment

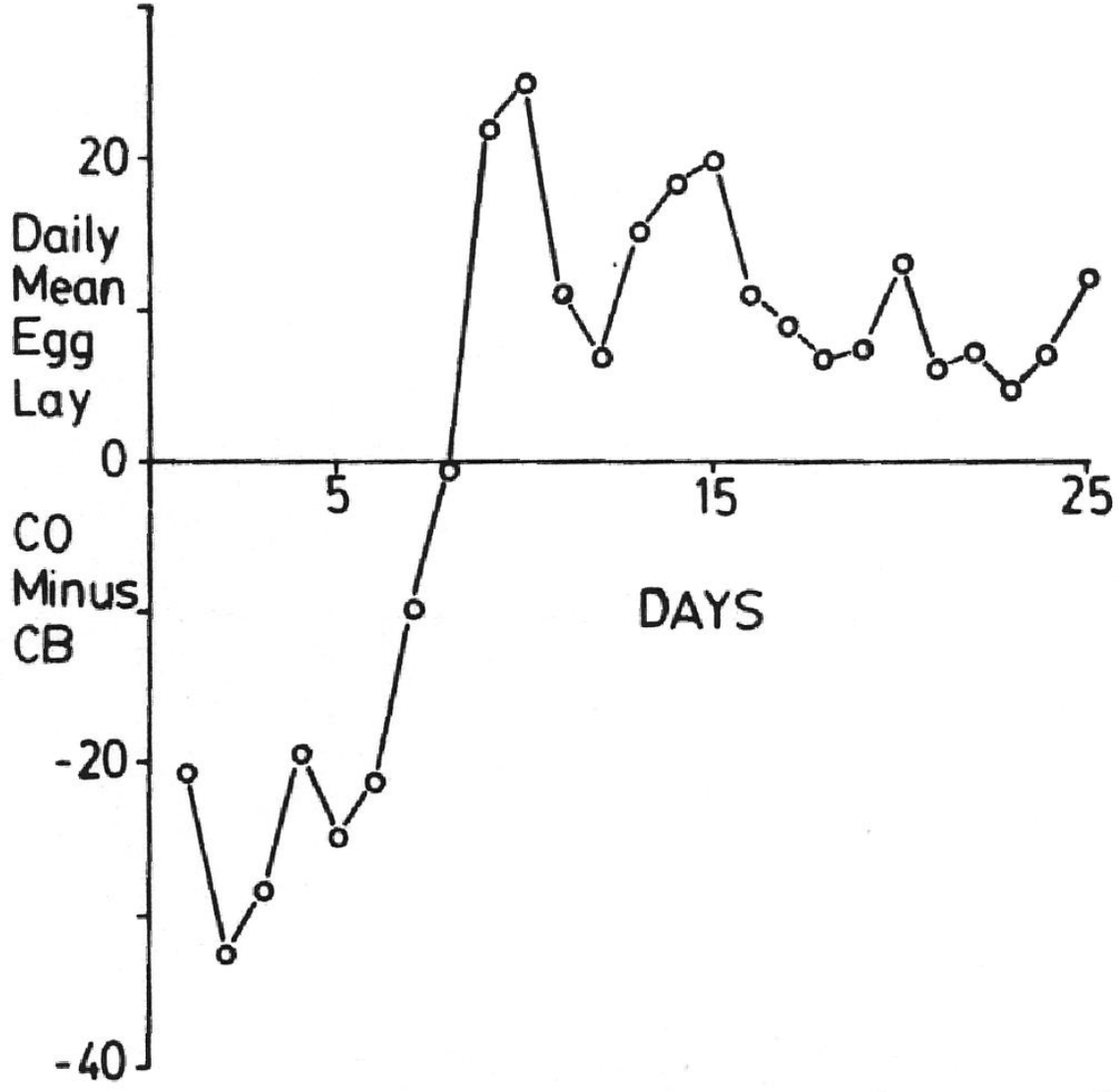


FIGURE 1.—Differences in mean daily conditional fecundities between samples of the CO and CB populations during assay days 1–25.

Rose & Charlesworth (1981)

6) The evolution of senescence

Evidence from selection experiments

Table 8.7 Mean lifespans in days survived from eclosion for populations of *Drosophila melanogaster* that had been forced to reproduce either early or late in life for 25 generations. From Hutchinson and Rose (1990)

Late-reproduced populations	Early-reproduced populations	Difference
71.3	60.7	10.6
55.1	46.7	8.4
64.4	49.5	14.9
59.6	47.2	12.4
62.2	46.3	15.9

6) The evolution of senescence

Prediction of mutation-accumulation hypothesis

Proc. Natl. Acad. Sci. USA
Vol. 93, pp. 6140–6145, June 1996

Age-specific inbreeding depression and components of genetic variance in relation to the evolution of senescence

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ABSTRACT **Two major theories of the evolution of senescence (mutation accumulation and antagonistic pleiotropy) make different predictions about the relationships between age, inbreeding effects, and the magnitude of genetic variance components of life-history components. We show that, under mutation accumulation, inbreeding decline and three major components of genetic variance are expected to increase with age in randomly mating populations. Under the simplest version of the antagonistic pleiotropy model, no changes in the severity of inbreeding decline, dominance variance, or the genetic variance of chromosomal homozygotes are expected, but additive genetic variance may increase with age. Age-specific survival rates and mating success were measured on virgin males, using lines extracted from a population of *Drosophila melanogaster*. For both traits, inbreeding decline and several components of genetic variance increase with age. The results are consistent with the mutation accumulation model, but can only be explained by antagonistic pleiotropy if there is a general tendency for an increase with age in the size of allelic effects on these life-history traits.**

a population even if they have adverse affects on late performance (3, 5, 7, 8, 10, 11).

Although there is widespread agreement that the general evolutionary theory of senescence provides a powerful tool for interpreting the genetics and comparative biology of aging, the relative contributions of mutation accumulation and antagonistic pleiotropy to senescence have not been elucidated (2, 3, 7, 12). The two theories are clearly not mutually exclusive, but it is legitimate to ask whether there is evidence for or against either of them. Recent advances in the genetics of Alzheimer disease suggest that both mechanisms may contribute to this aspect of senescence in humans (13). In *Drosophila*, there is evidence that both supports and casts doubt on the tradeoffs postulated by the antagonistic pleiotropy model (3, 14, 15). There is also conflicting evidence on the importance of mutation accumulation (16–20). In this paper, we show that inbreeding depression is expected to increase with age under the mutation accumulation model but not under antagonistic pleiotropy (unless the effects of segregating alleles themselves increase with age). We present data on inbreeding effects on

6) The evolution of senescence

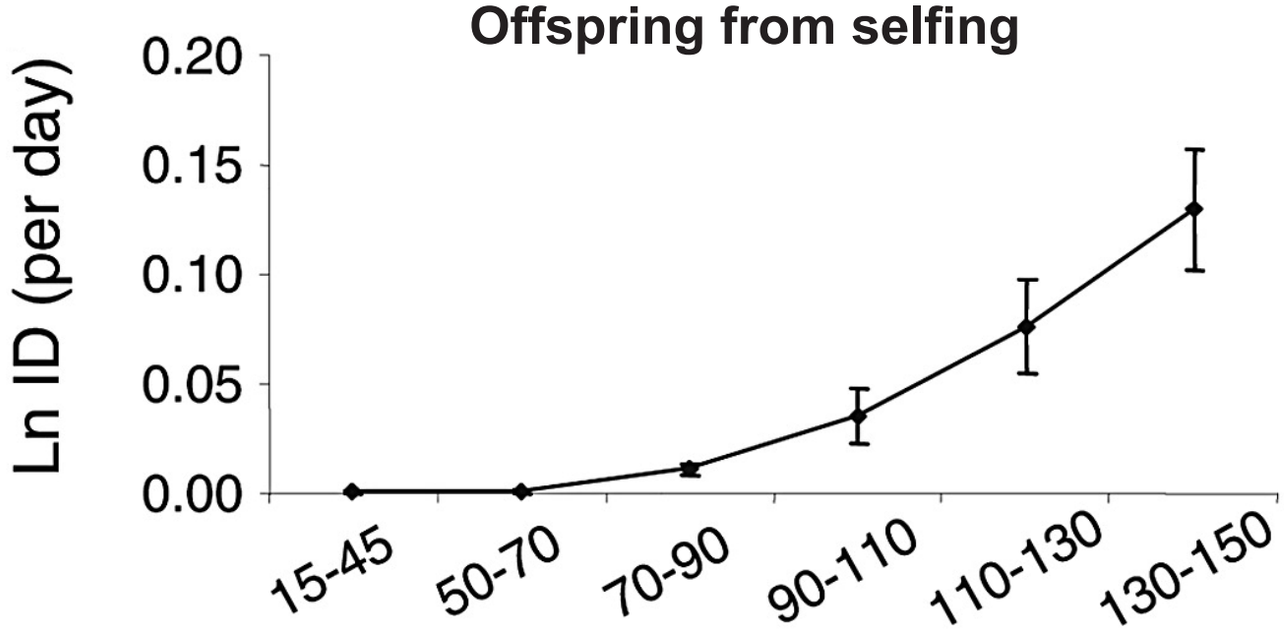
Test of mutation-accumulation

Inbreeding exposes recessive, deleterious mutations. Study tested if the effects of inbreeding on survival depended on age in the hermaphroditic snail, *Physa acuta*

Argument: The deleterious effects of inbreeding (inbreeding depression, ID) should increase with the frequency of expressed deleterious mutations.

Therefore, under the mutation-accumulation hypothesis, the effects of inbreeding depression (ID) should be greater at greater ages.

http://www.cofc.edu/~fwgna/species/physidae/P_acuta_Liebman.jpg



Escobar et al (2008)

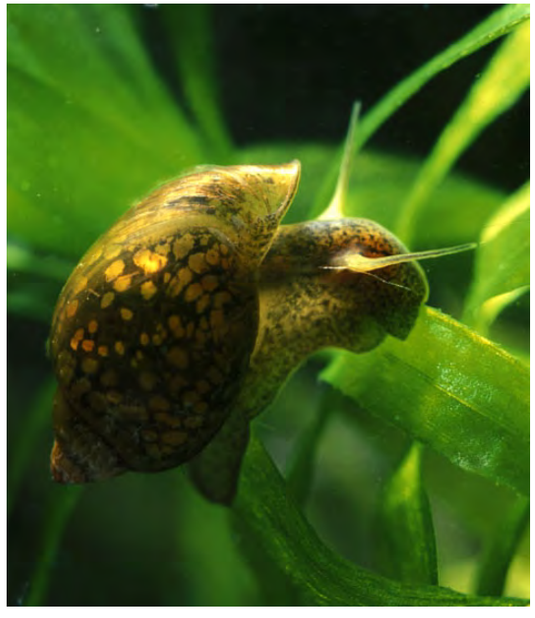
6) The evolution of senescence

Test of mutation-accumulation

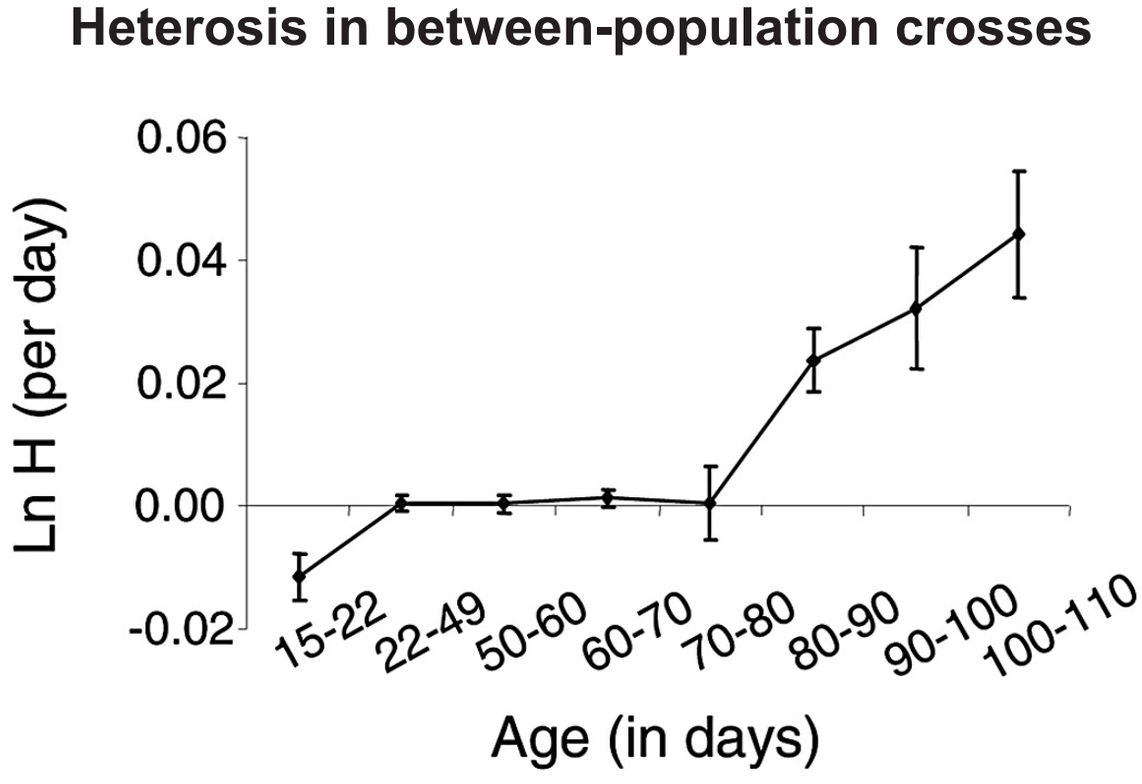
The study also measured age-dependence of effects of heterosis for survival.

Heterozygote advantage (heterosis) results when effects of deleterious mutations are masked in hybrids. Greater numbers of deleterious mutations should lead to greater heterosis when hybrids are made between populations having different deleterious mutations.

Under the mutation-accumulation hypothesis, effects of heterosis should increase with age.



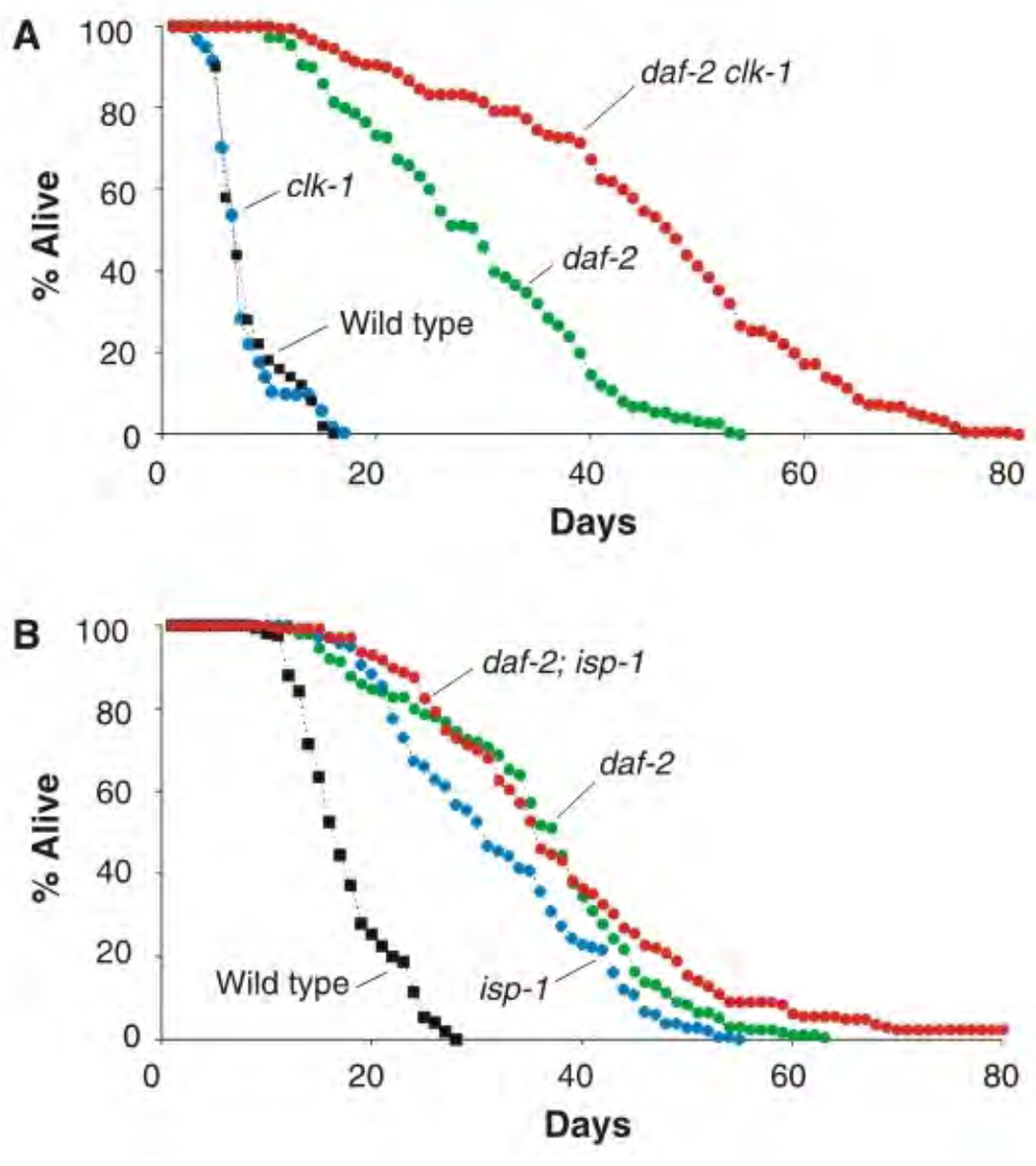
http://www.cofc.edu/~fwgna/species/physidae/P_acuta_Liebman.jpg



6) The evolution of senescence

Genetics of aging in *C. elegans*

Fig. 1. Genetic interactions among longevity genes. (**A**) *daf-2(e1370) clk-1(e2519)* double mutants live a very long time, indicating that almost all age-dependent degenerative processes in the worm can be substantially prevented by altering only very few genes at a time. Animals were raised at 20°C and then permanently placed at 25°C. Adult life-span is shown. The data are from (*2*). (**B**) *daf-2(e1370)* and *isp-1(qm150)* increase life-span to a similar degree but do not show a positive interaction, because the double mutants *daf-2; isp-1* do not live appreciably longer than the single mutants. Adult life-span at 20°C is shown. The data are from (*45*).



7) Example exam questions

Define and give an example of: reproductive value; life history trade-off; phylogenetic constraint

To measure life history trade-offs, one might study a population in nature and examine the phenotypic correlation between the reproductive output of individuals and their subsequent survival. Comment on the validity of this approach to measuring life history trade-offs.

Why don't all species of animals and plants mature at an early age?

Comment on the advantages and disadvantages of a “fecundity manipulation” in tests of life history theories.

What evolutionary changes in life history might we expect to see in a seal population following the commencement of a program by humans to reduce seal numbers by hunting adults? Base your expectations on life history theory, and use examples where possible.

Discuss the consequences of mortality caused by predators on the evolution of life history of prey species.

Why might rates of externally-imposed mortality influence the evolution of senescence?

Provide two hypotheses to explain an observed positive correlation between rates of externally imposed mortality on animal populations and the rate of senescence in these populations. Design a test to distinguish the hypotheses.

Suggest a way in which the human species might evolve a longer average lifespan. (Note emphasis on the word “evolve”---the change must take place over future generations, not within an individual lifetime). Justify your proposal on theoretical and experimental grounds.