Life History Evolution: The evolution of senescence
Senescence

Why?

1. Constraint

2. Mutation accumulation (Medawar, 1946)

3. Antagonistic pleiotropy (Medawar, 1946; Williams, 1957)
Senescence

Constraint: ‘wear and tear’

The evolution of senescence

Theory:

Constraint
Mutation accumulation
Antagonistic pleiotropy

Empirical evidence:
Comparative tests
Selection experiments
Aging genes

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Senescence

Constraint


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Mutation accumulation

10% breakage/month
So after 1 month 10 break
100 tubes

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Mutation accumulation

10% breakage/month

So after 1 month 10 break

100 tubes

+ 1/9 fecundity per unbroken individual

100 tubes

... so population number is stable
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Mutation accumulation

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.
Senescence

Mutation accumulation

The evolution of senescence

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- ‘brittle’ mutation has 20% breakage rate

Differential breakage rate

So after 1 month:
9/90 ‘regular’ break
2/10 ‘brittle’ break

100 tubes
Senescence

Mutation accumulation

Differential breakage rate

So after 1 month:
9/90 ‘regular’ break
2/10 ‘brittle’ break

‘brittle’ mutation has 20% breakage rate

fecundity replaces broken individuals

100 tubes

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Mutation accumulation

Early expression of ‘brittle’

Late expression of ‘brittle’

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Early expression of ‘brittle’

Late expression of ‘brittle’

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.
A gene influences two traits simultaneously: selection for one trait is counteracted by selection against another:

genotypes that were associated with enhanced juvenile survival tended to reduce adult reproductive success.
Senescence

Antagonistic pleiotropy

Gene ‘A’

Positively effects fitness early in life

Negatively effects fitness late in life

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Mutation accumulation

Selection is ineffective at removing mutations with effects in old age.

Antagonistic pleiotropy

Early reproduction is favoured by natural selection.
Mutation with positive effect on fitness when young can increase even if it has negative effects late in life:
“necessary cost of processes beneficial to youth”
Senescence

Mutation accumulation & antagonistic pleiotropy:

- Senescence greatest when extrinsic mortality is high because: few individuals survive that long, therefore, there will be little selection against deleterious mutations in old age.
- Why invest in repair if you’re going to die for other reasons first?

Senescence

Empirical evidence: comparative tests

Birds in zoo


The evolution of senescence

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Senescence

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Empirical evidence: selection experiments


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Senescence

Empirical evidence: selection experiments


**Table 1.** Genetic models of senescence. Plus and minus signs indicate the effect of each allele relative to the other on either early-life or late-life fitness. Circles indicate neutrality.

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1. Select for high late-life fitness
2. Select for high early-life fecundity
No change

Senescence

Empirical evidence: aging genes


Nematode Caenorhabditis elegans

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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).
Overview

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Theory:
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References


