



NATURAL SELECTION: Fundamentals

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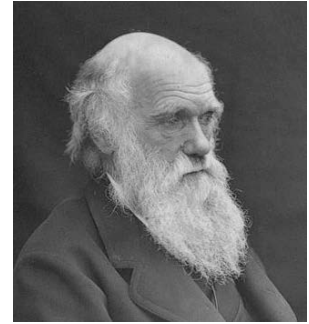




Natural selection



- “This preservation of favourable variations and the rejection of injurious variations, I call Natural Selection.”
--**Darwin** (1859)
- Presented simultaneously with **A. R. Wallace**
- Emphases differed, though both correct—
 - **Darwin**: emphasized *competition within species*
 - Wallace**: emphasized *environmental pressures*



Is evolution by natural selection “just a theory”?

- Let's imagine that you're working with a population of squirrels.

Type A: run randomly Type B: fear asphalt

Average: 1 offspring 2 offspring

Start: A: 100 B: 100 50% B

Gen 1:

Gen 2:

Gen 3:

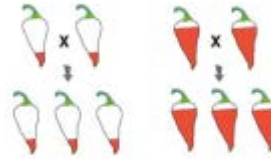


Requirements for evolution by natural selection

- Variation in traits



- Heritability of traits



- Trait variants affect survival/ reproduction



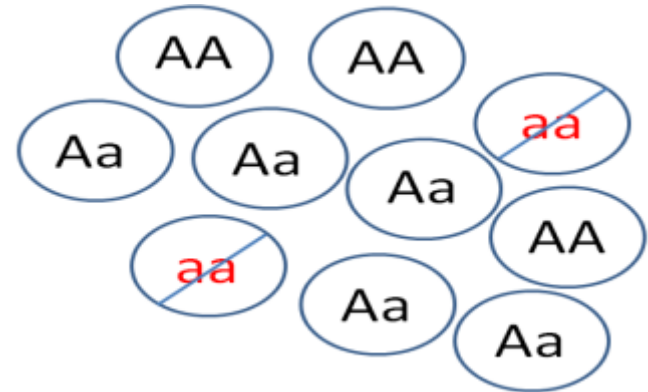
Quantitative Traits vs. Single Locus

- Already discussed selection in the context of heritability:
Heritability = Response/Selection
 - Genetic component of variation dictates selection's response
 - Response often from change in allele frequencies at **multiple loci**
- Can also be studied at *single locus/ gene*
 - We'll come back to phenotypes shortly



What does selection do to alleles at individual loci?

- Affects abundance of particular genotypes
e.g., **AA**: good, **Aa**: good, **aa**: “less good” (dead?)
- Affects frequency of alleles in population
—In example above, fewer “a” alleles remain in population
- DOMINANCE OF ALLELES MATTERS FOR SELECTION



Strong selection in humans:

Single loci

- Spontaneous bad mutations are *common*
- Half of pregnancies never detected because spontaneously abort very early
- Half of spontaneous abortions result from genetic problems
 - ~25% of all human fertilizations immediately eliminated by natural selection!



Weak(er) selection in humans:

Single loci

- Historically, all humans adult lactose intolerant
- Estimate ~5% fewer kids if lactose intolerant
- New mutation arose— now most people “lactase persistent” (lactose tolerant) as adults
 - What is effect of 5% more kids???



Can simulate with **AlleleA1**

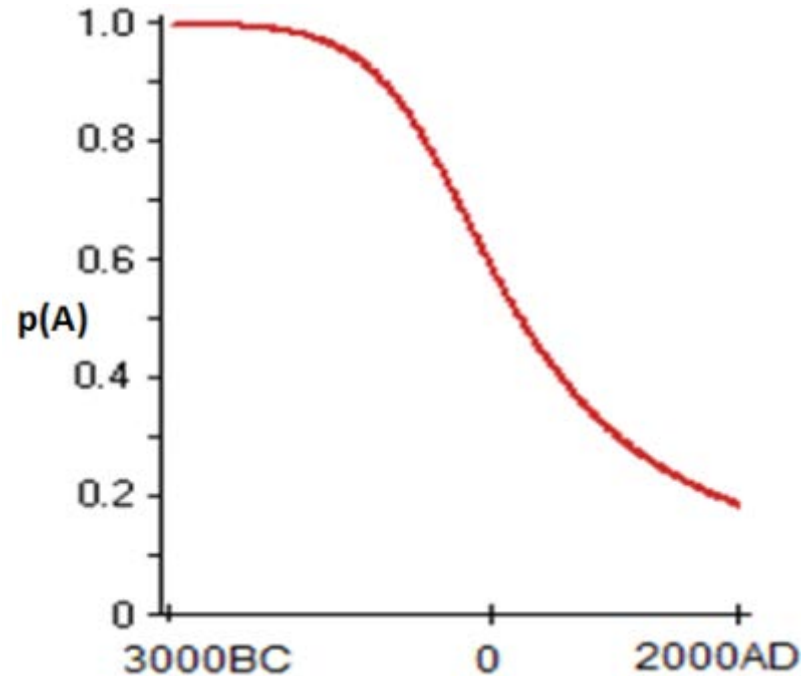
- Fitness of “AA” (intolerant) is 0.95
- Fitness of “Aa” and “aa” (tolerant) is 1.00
- Time: 5000 years
- All were “AA” and then new mutation (a) arose in Africa ~5000 years ago

<http://faculty.washington.edu/herronjc/SoftwareFolder/AlleleA1.html>



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Selection uses

“*relative fitness*” of genotypes

- In lactase example:
 - AA: 0.95 Aa: 1.00 aa: 1.00
 - AA has “5% fewer kids” successfully on average than Aa or aa
- Because something is “selected against”, doesn’t mean it’s “bad” by itself, just not as good as the alternative...
 - Humans survived for a long time as AA (lactose intolerant)...





Analogy...

“I love rock & roll”

Forgotten original is
by the Arrows, 1975

- A new mutation is like a released cover of a previously released song
 - Original song was popular/ successful
 - Cover may be more successful
 - Spreads (sells), and everyone forgets the original
 - Cover may be less successful
 - Around briefly but then dies off

1982



Joan Jett

vs

Britney Spears

2002



What is relative fitness? *Example.*

- BB genotypes produces on average 3.2 surviving offspring
- Bb genotypes produce on average 3.0 offspring
- bb genotypes produce on average 2.4 offspring



What is relative fitness? *Example.*

- BB genotypes produces on average 3.2 surviving offspring
- Bb genotypes produce on average 3.0 offspring
- bb genotypes produce on average 2.4 offspring
- Most fit genotype: BB
 - Call it “100% of maximum”, fitness = $w(\text{BB}) = 1.00$
 - Others are percentage of *maximum*
 - $w(\text{Bb}) = 3.0 / 3.2 = 0.94$ (~6% less fit than BB)
 - $w(\text{bb}) = 2.4 / 3.2 = 0.75$ (~25% less fit than BB)





Effects on Hardy-Weinberg

- Assume all “aa” individuals die **at age 10**
- At age 8:
 - AA: 490 Aa: 420 aa: 90
 - Is this 8-year-old population at HW?





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Effects on Hardy-Weinberg

- Assume all “aa” individuals die **at age 10**
- At age 8:
 - AA: 490 Aa: 420 aa: 90 $p(A) = 0.70$
- At age 25:
 - AA: 490 Aa: 420 aa: --dead--





Effects on Hardy-Weinberg



- Assume all “aa” individuals die **at age 10**
- At age 8:
 - AA: 490 Aa: 420 aa: 90 $p(A) = 0.70$
- At age 25:
 - AA: 490 Aa: 420 aa: 0 $p(A) = 490/910 + \frac{1}{2} (420/910) = 0.769$





Effects on Hardy-Weinberg



- Assume all “aa” individuals die **at age 10**
- At age 8:
 - AA: 490 Aa: 420 aa: 90 $p(A) = 0.70$
- At age 25:
 - AA: 490 Aa: 420 aa: 0 $p(A) = 0.77$
- Deviation from Hardy-Weinberg
 - Selection altered genotype frequencies
- Selection also altered allele frequencies



Effects on Hardy-Weinberg



- Assume all “aa” individuals die **at age 10**
- At age 8:
 - AA: 490 Aa: 420 aa: 90 $p(A) = 0.70$
- At age 25:
 - AA: 490 Aa: 420 aa: 0 $p(A) = 0.77$
- Deviation from Hardy-Weinberg
 - Selection altered genotype frequencies
- Selection also altered allele frequencies
- **Are aa's gone for good? What happens next?**

What happens next?

AA: 490

Aa: 420

aa: 0

Total number of survivors: 910

$$\text{freq}(\text{AA}) = 490 / 910 = 0.538$$

$$\text{freq}(\text{Aa}) = 420 / 910 = 0.462$$

$$p(\text{A}) = 0.538 + \frac{1}{2} 0.462 = 0.769 \quad (\text{was } 0.70)$$

$$q(\text{a}) = 0.000 + \frac{1}{2} 0.462 = 0.231 \quad (\text{was } 0.30)$$

Babies in next generation (assuming random mating):

AA: 0.591

Aa: 0.355

aa: 0.054

They're back!



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