

**Big Idea 4: Biological systems interact, and these systems and their interactions possess complex properties.**

Enduring understanding 4.C:  
Naturally occurring diversity  
among and between  
components within biological  
systems affects interactions  
with the environment.

***Essential knowledge 4.C.3: The level of variation in a population affects population dynamics.***

a. Population ability to respond to changes in the environment is affected by genetic diversity. Species and populations with little genetic diversity are at risk for extinction. [See also 1.A.1, 1.A.2, 1.C.1]

To foster student understanding of this concept, instructors can choose an illustrative example such as:

- California condors
- Potato blight causing the potato famine
  - Prairie chickens
- Corn rust affects on agricultural crops
  - Black-footed ferrets
- Tasmanian devils and infectious cancer

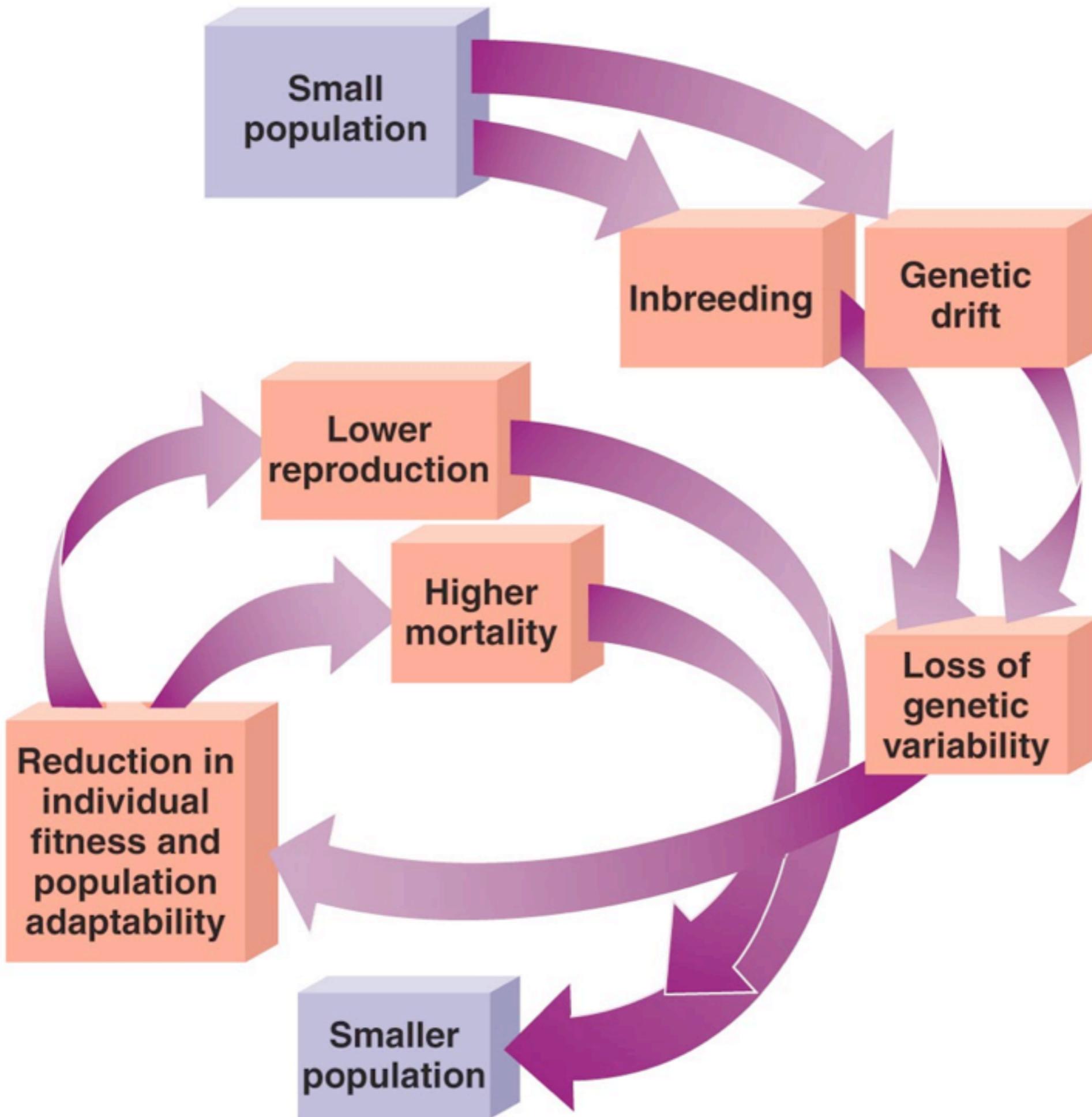
# **POPULATION CONSERVATION FOCUSES ON POPULATION SIZE, GENETIC DIVERSITY AND CRITICAL HABITAT**

## **Small Population Approach**

- Particularly vulnerable to over harvesting and habitat loss
- study processes that cause extinctions of small populations

## **The Extinction Vortex: Evolution Implications**

- Small populations suffer from Inbreeding and Genetic Drift which result in a loss of genetic variation.



# Minimum Viable Population Size

- Minimal population size at which a species is able to sustain its numbers..(MVP)
- Depends on the organism and other factors

# Effective Population Size

- Population size by itself can be misleading, Effective population size is based on breeding potential.
- Consider a population of 1000 with only 5 females and a population of 100 with 40 females. See a difference?

How might the age of individuals in a population play a role?

*\*Read Case Study on Grizzly Bears if you get a chance*

# Case Study: Greater Prairie Chicken

Booming mating calls rocked the Illinois prairie in the mid-1800s, announcing that colorful greater prairie chickens were near and abundant. As pioneers moved west, the birds were hunted for food. They fell to predators, their habitats shrank, and, scientists say, even the birds' declining genetic diversity brought their near extinction. In the Nov. 27 issue of the journal *Science*, nine researchers report that an isolated group of the birds is making a comeback.



**The greater prairie chicken is estimated to have numbered in the millions in Illinois when pioneers moved west. They were "shot by the wagonload for food," said Ronald Westemeier, a recently retired scientist with the Illinois Natural History Survey.**

**As the prairie diminished, so did the birds' habitat - from more than 60 percent of the state to less than 0.01 percent and in just two isolated populations in Jasper and Marion counties.**

**From 1962 to 1994, in Jasper County alone, their numbers fell from 2,000 to less than 50.**

**"The message is that fragmentation and habitat loss can really lead to a number of problems in conserving species.**

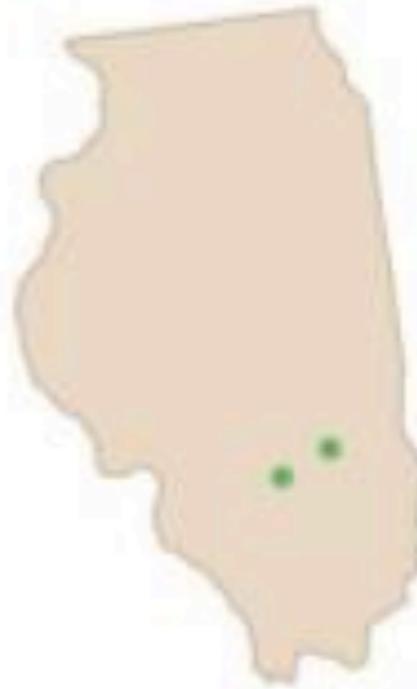


 Range of greater prairie chicken

Pre-bottleneck  
(Illinois, 1820)



Post-bottleneck  
(Illinois, 1993)



**Habitat  
Loss**

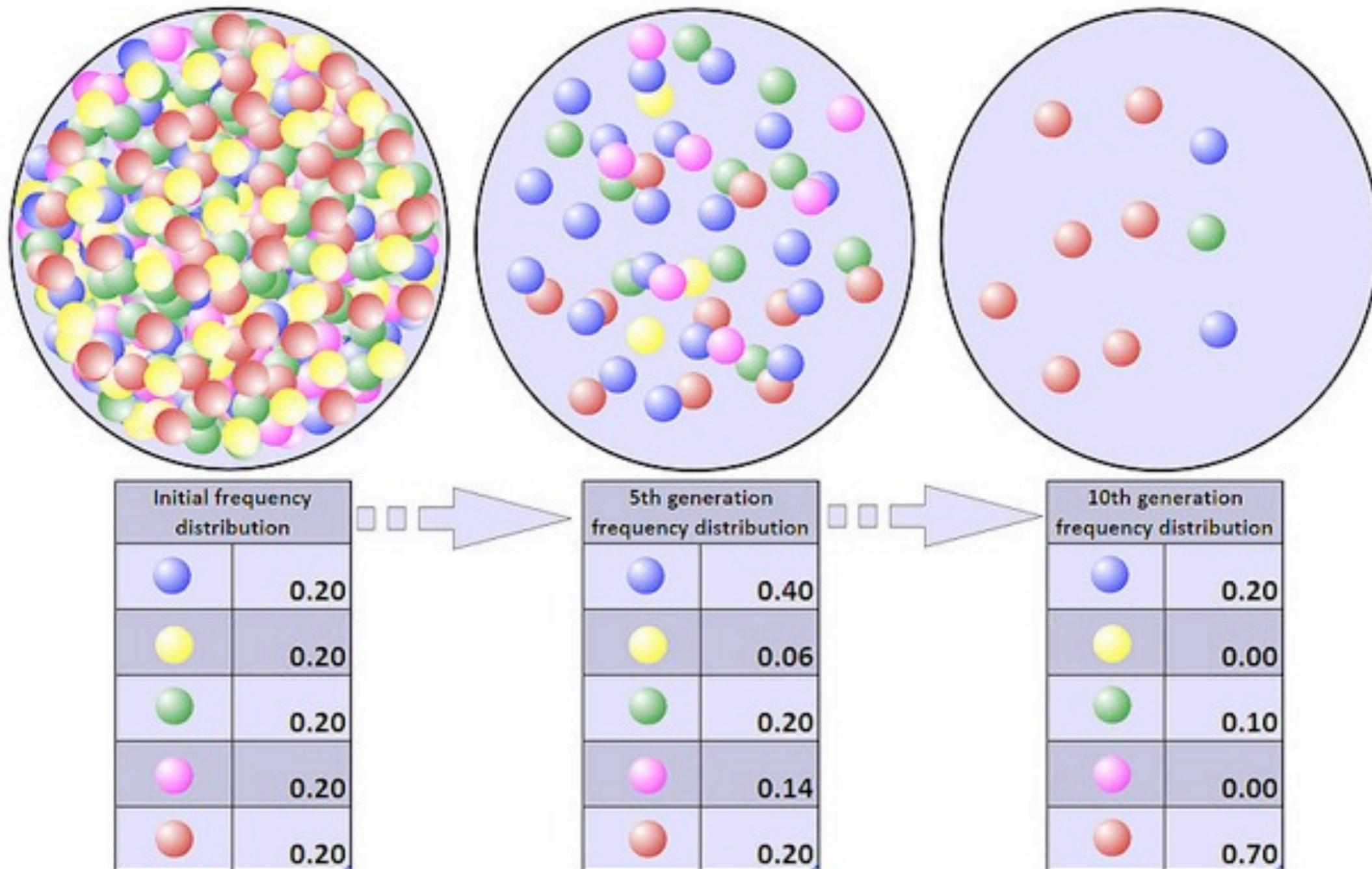
# The Problem



**In this case, these factors led to genetic problems.**

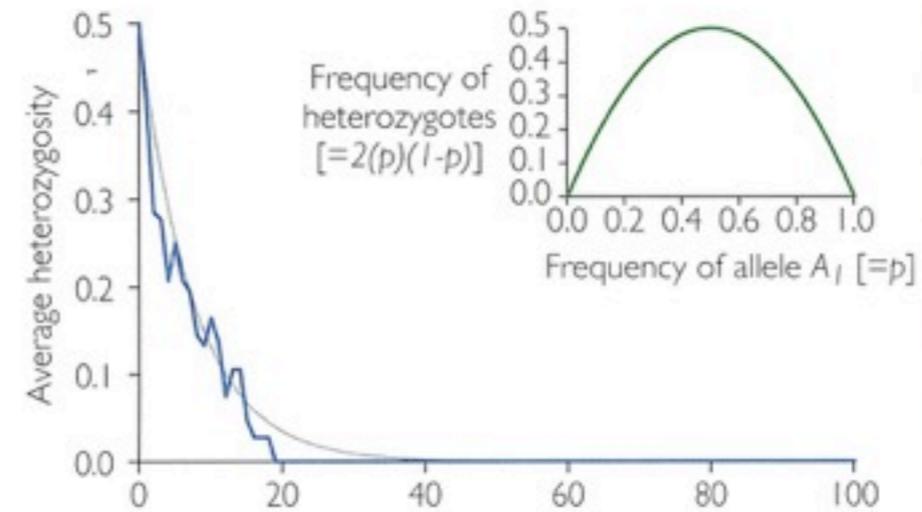
**What was interesting is that the people who manage the population did their best, going to extraordinary measures to preserve this population over the years, yet it kept going down and down, owing to the fact that it was just a small relic population that had low genetic diversity.**

...by genetic analysis, including that of museum specimens from the 1930s, that the isolated Illinois birds had been part of a much larger population, and they concluded that a lack of genetic diversity resulted from population decline.

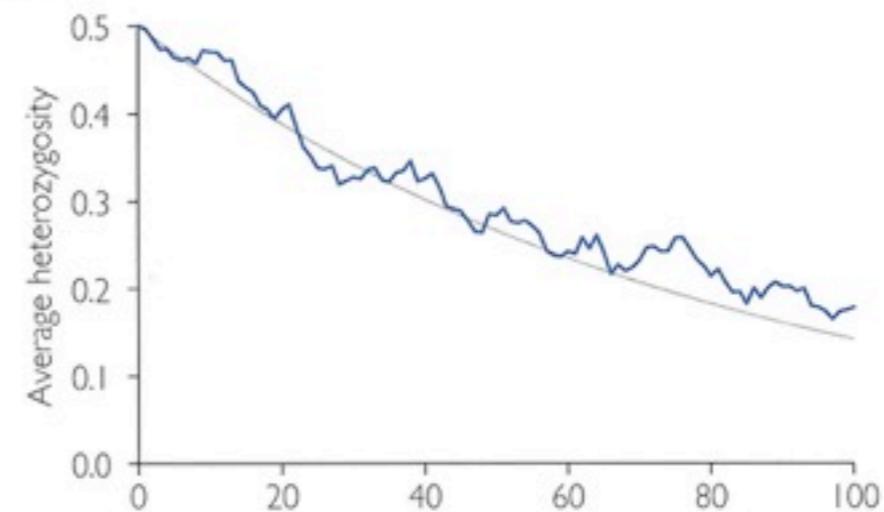


# Another Way of Viewing the Importance of Genetic Variation

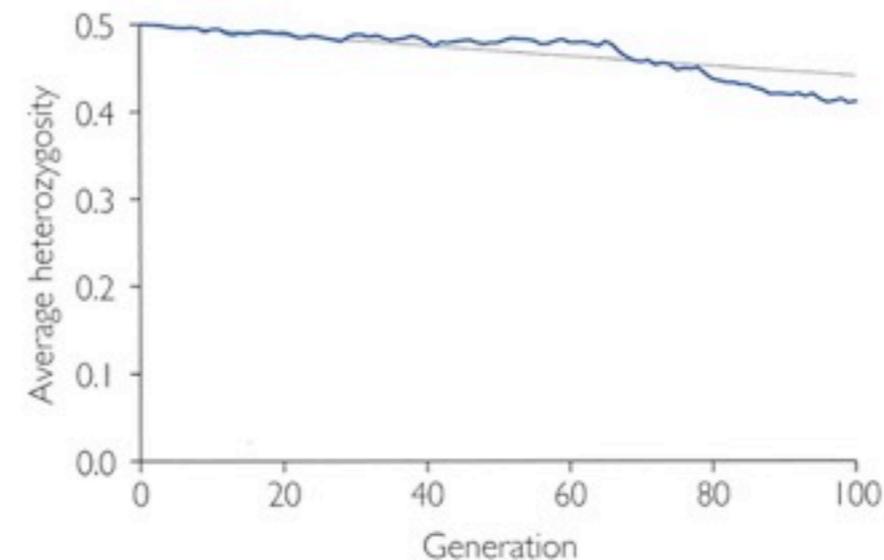
(d) Population size = 4

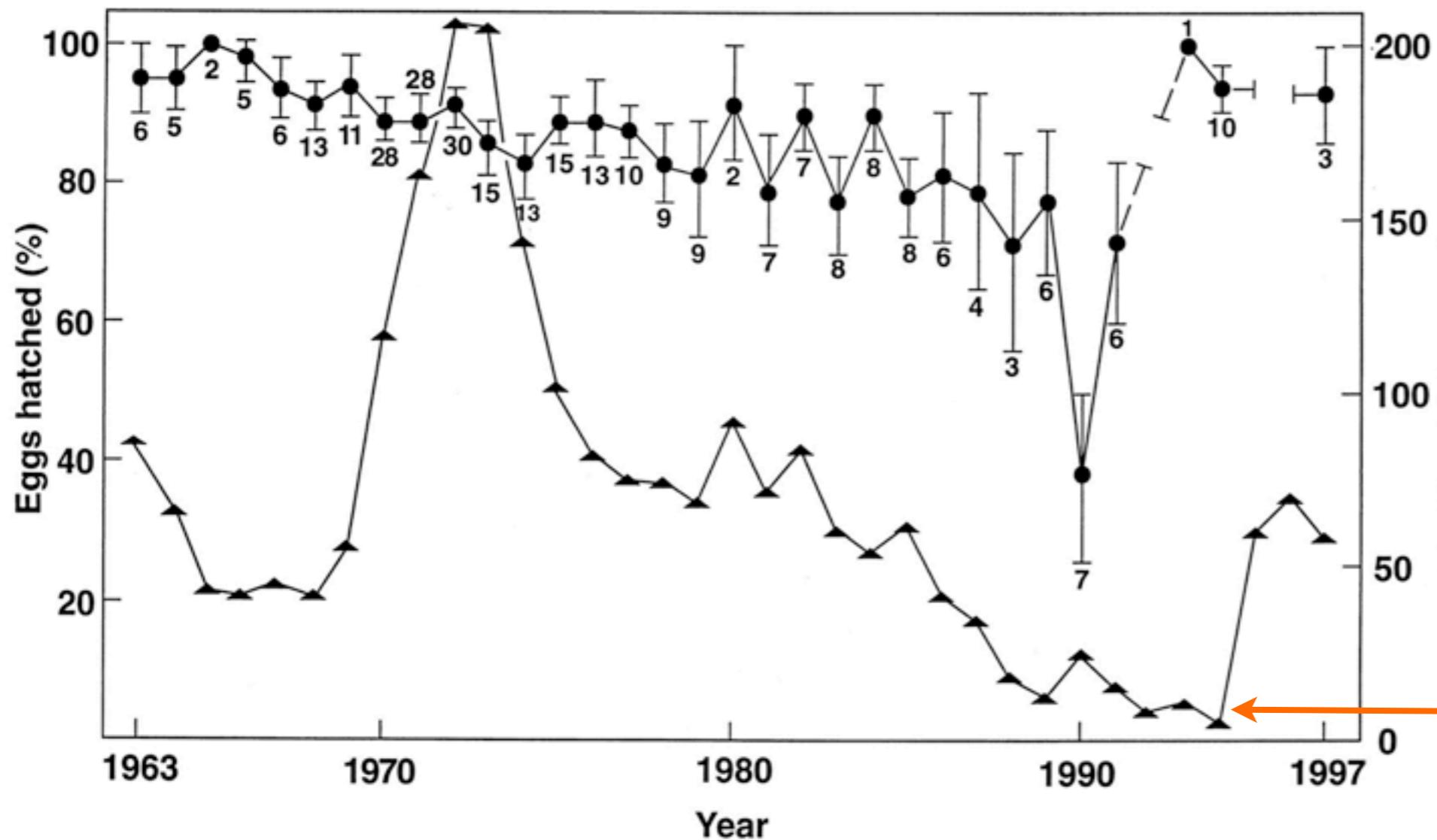


(e) Population size = 40



(f) Population size = 400





Imported birds  
from a larger  
population  
elsewhere

**Transfusion  
of genetic  
variability**

The success - in which more than 500 birds were brought in beginning in 1992 from larger populations in Minnesota, Kansas and Nebraska - could serve as a model to save dwindling populations of wild species from extinction, the researchers say. The 35 years of data represent one of the most detailed sets of data ever collected from an isolated and declining wildlife population.

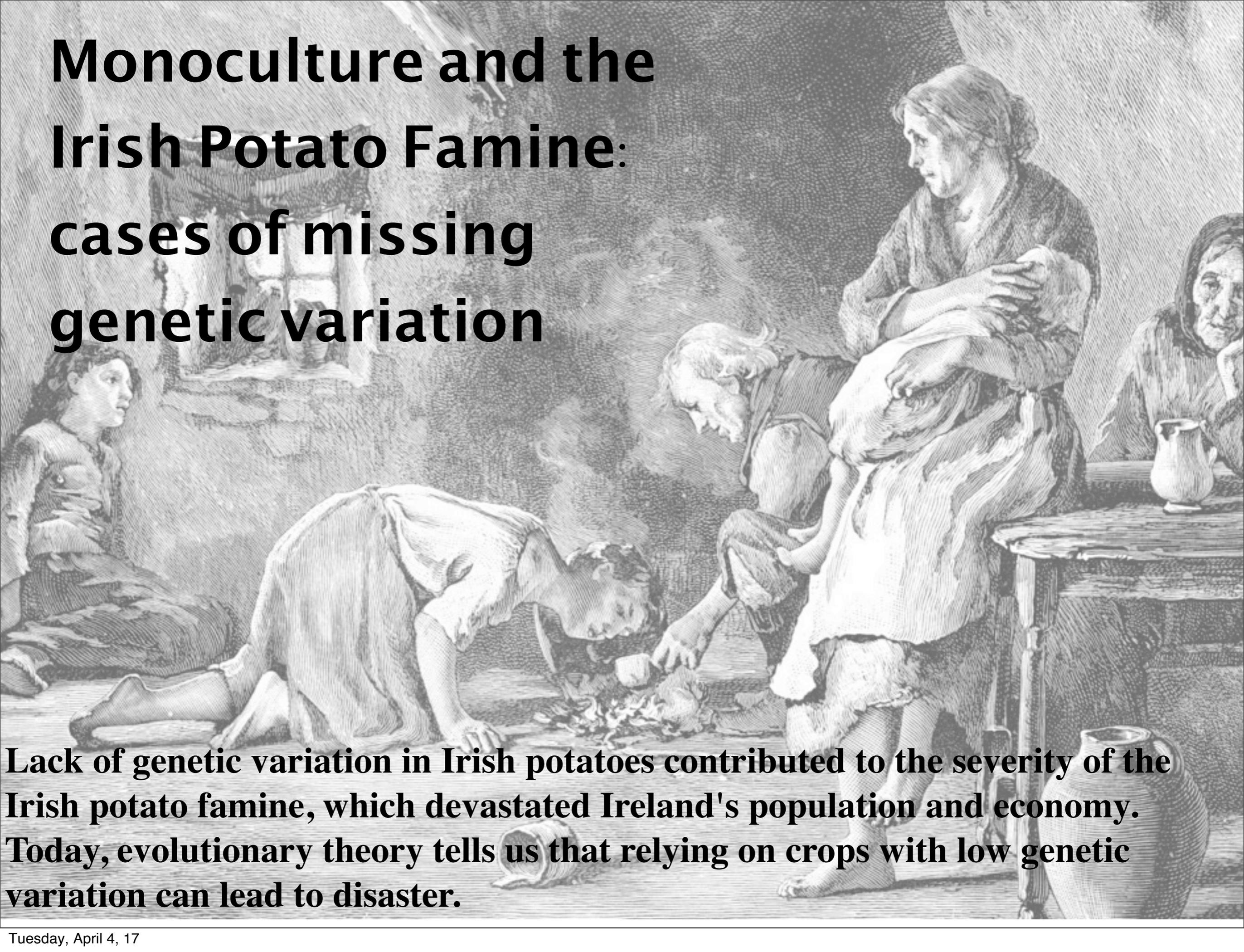
"These genetic findings are important, because no other studies have been able to show precisely what was lost, leaving alternative possible interpretations such as they were always depauperate [lacking] in genetic variation. These alleles are merely markers of overall genetic diversity and the alleles lost over time. But it can be implied that a loss in genetic variation was a factor because the introduction of new genetic material by these outside birds bolstered fitness back to original levels,"

# Far Reaching Effects

Sometimes the lack of genetic diversity effects more than one species...for example

## Potato Blight

# **Monoculture and the Irish Potato Famine: cases of missing genetic variation**



**Lack of genetic variation in Irish potatoes contributed to the severity of the Irish potato famine, which devastated Ireland's population and economy. Today, evolutionary theory tells us that relying on crops with low genetic variation can lead to disaster.**

# Lumpers

In the 1800s, the Irish solved their problem of feeding a growing population by planting potatoes. Specifically, they planted the "lumper" potato variety. And since potatoes can be propagated vegetatively, all of these lumpers were clones, genetically identical to one another.

The lumper fed Ireland for a time, but it also set the stage for human and economic ruin. Evolutionary theory suggests that populations with low genetic variation are more vulnerable to changing environmental conditions than are diverse populations. The Irish potato clones were certainly low on genetic variation, so when the environment changed and a potato disease swept through the country in the 1840s, the potatoes (and the people who depended upon them) were devastated.

# The importance of diversity



The genetically identical lumpers were all susceptible to a rot caused by *Phytophthora infestans*, which turns non-resistant potatoes to inedible slime.

**Because Ireland was so dependent on the potato, one in eight Irish people died of starvation in three years during the Irish potato famine of the 1840s.**

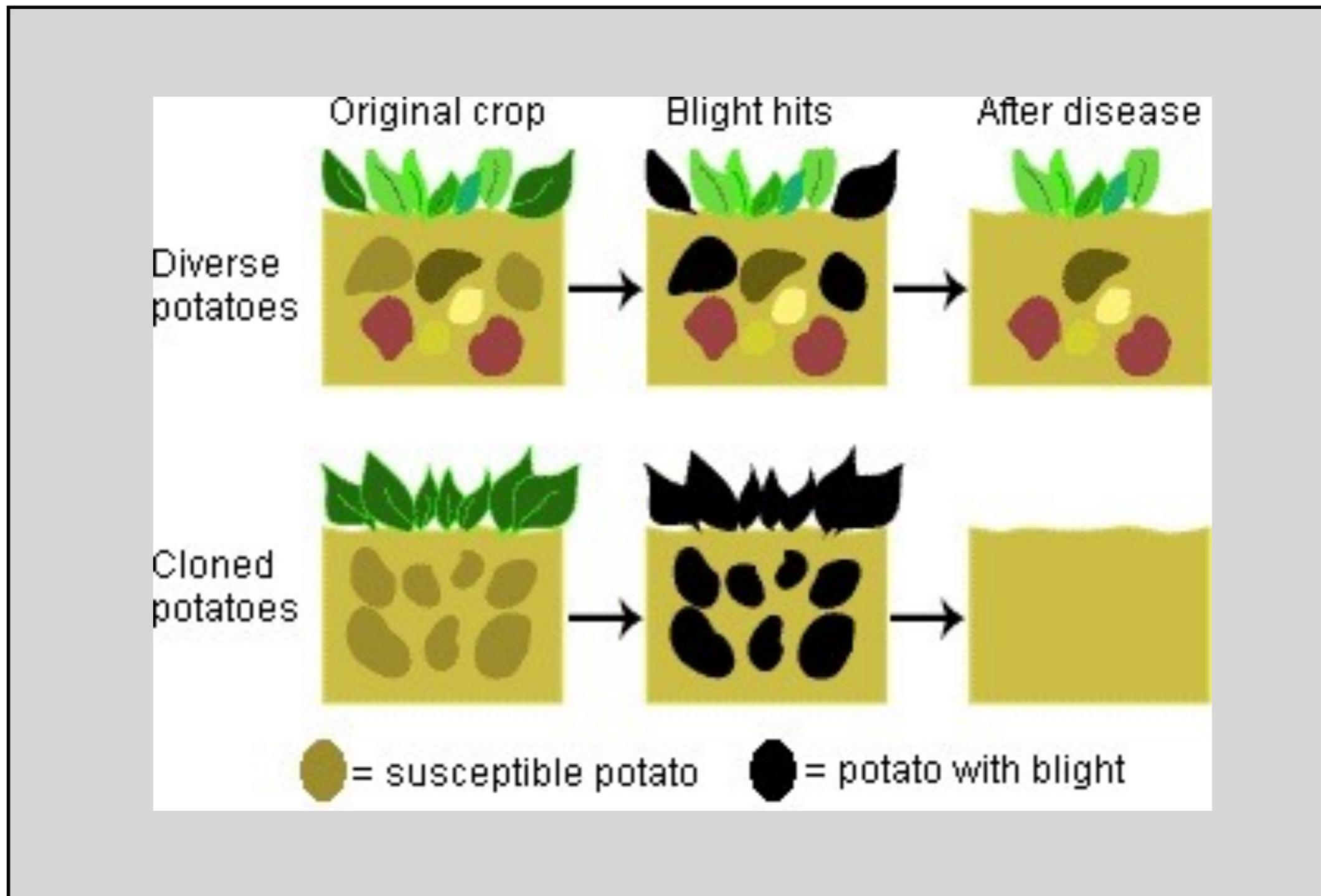
**Over 1 million people died**

Although the famine ultimately had many causes, the disaster would likely not have been so terrible had more genetically variable potatoes been planted. Some potatoes would have carried the right genes to make it through the epidemic, and more of the resistant varieties could have been planted in the years following the first epidemic.



Later, scientists identified resistance genes in a potato from South America, where farmers have preserved the genetic variation of potatoes by growing many cultivated varieties alongside the potato's wild cousins.

# The image below compares the effect of a blight on diverse and cloned crops.



# Ignoring history

Despite the warnings of evolution and history, much agriculture continues to depend on genetically uniform crops. The widespread planting of a single corn variety contributed to the loss of over a billion dollars worth of corn in 1970, when the U.S. crop was overwhelmed by a fungus. And in the 1980s, dependence upon a single type of grapevine root forced California grape growers to replant approximately two million acres of vines when a new race of the pest insect, grape phylloxera (*Daktulosphaira vitifoliae*, shown at right) attacked in the 1980s.

Although planting a single, genetically uniform crop might increase short term yields, evolutionary theory and the lessons of history highlight an undesirable side effect. Planting genetically uniform crops increases the risk of "losing it all" when environmental variables change: for example, if a new pest is introduced or rainfall levels drop.

# Other Cases of Organisms Endangered by low Genetic Diversity

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## California Condor



The spectacular but endangered California Condor is the largest bird in North America. The population fell to just 22 birds in the 1980s, but there are now some 230 free-flying birds in California, Arizona, and Baja California with another 160 in captivity. Lead poisoning remains a severe threat to their long-term prospects.

# Other Cases of Organisms Endangered by low Genetic Diversity

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## **Black Footed Ferrets**



Once thought to be globally extinct, black-footed ferrets are making a comeback. Today, recovery efforts have helped restore the black-footed ferret population to nearly 300 animals across North America. Although great strides have been made to recover the black-footed ferret, habitat loss and disease remain key threats to this highly endangered species.

*Essential knowledge 4.C.3: The level of variation in a population affects population dynamics.*

b. Genetic diversity allows individuals in a population to respond differently to the same changes in environmental conditions.

To foster student understanding of this concept, instructors can choose an illustrative example such as:

-Not all animals in a population stampede.

-Not all individuals in a population in a disease outbreak are equally affected; some may not show symptoms, some may have mild symptoms, or some may be naturally immune and resistant to the disease.

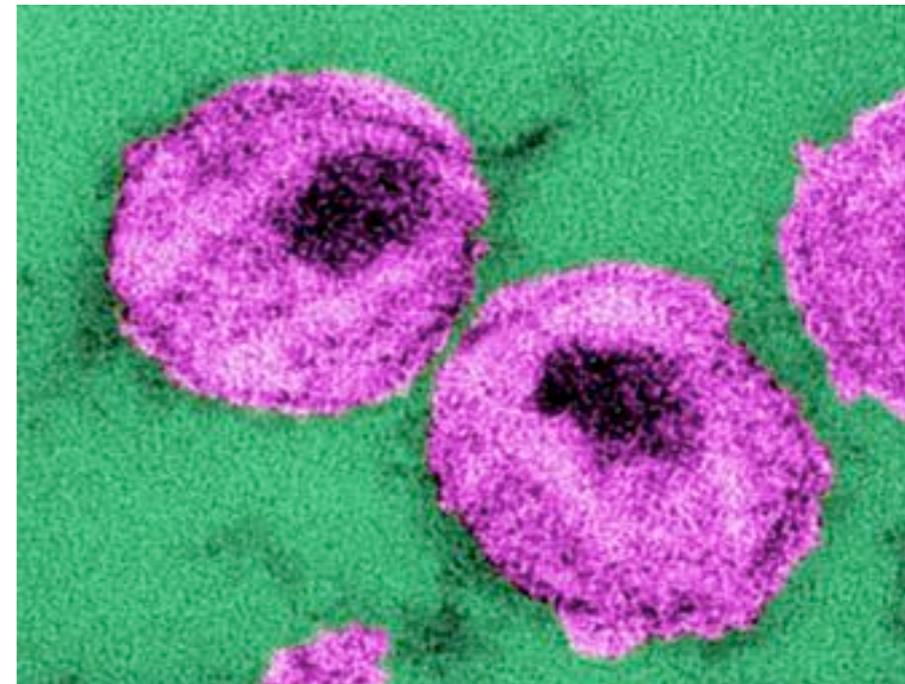
# HIV Resistant Mutation

HIV virus is one of the most pressing health concerns facing the modern world.

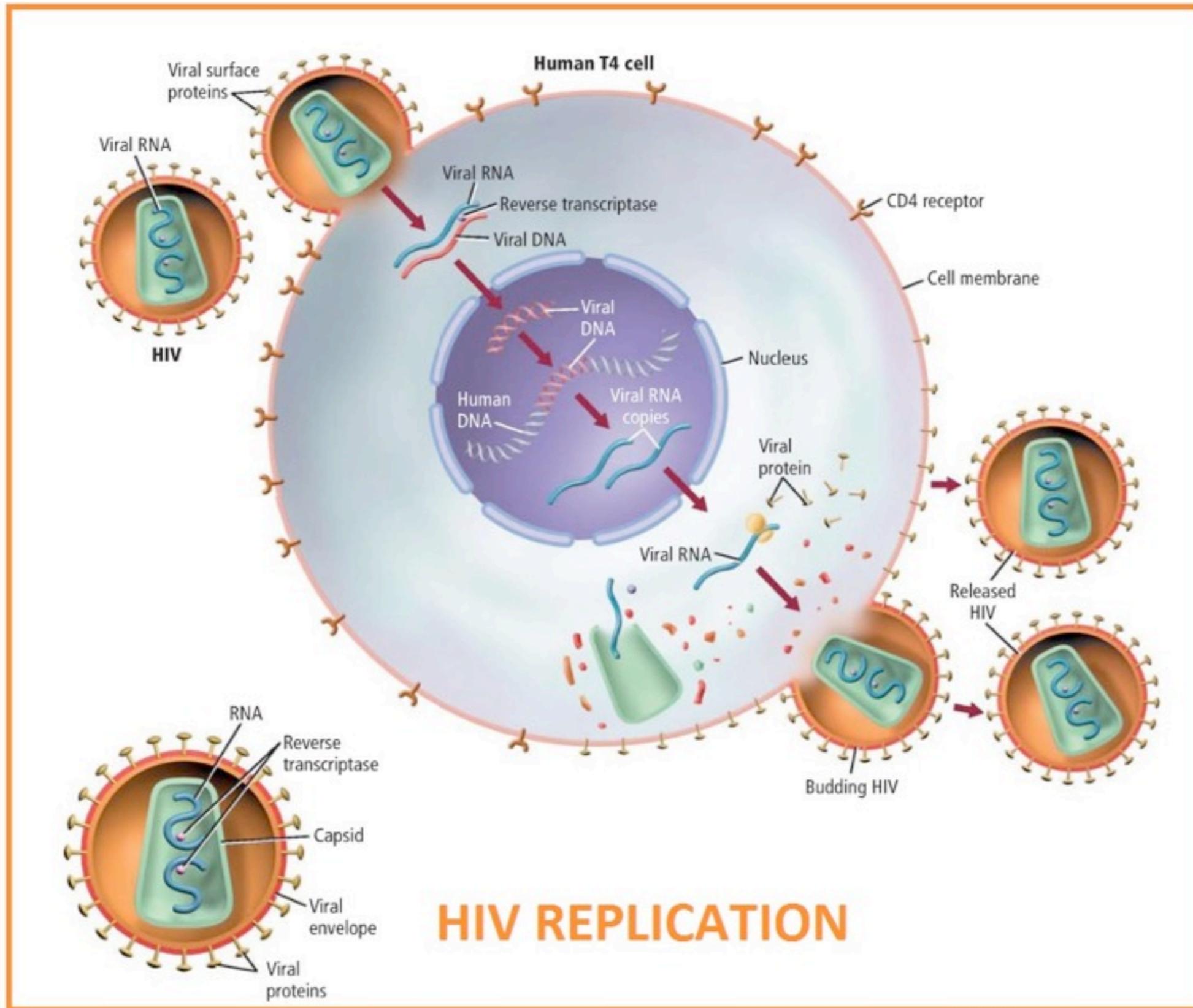
Since the first reported case of HIV/AIDS in 1981, over 25 million people have died.

Out of the millions of people infected each year with the HIV virus, a few have shown HIV/AIDS resistance.

**A genetic mutation found mostly in people of European descent delays the progression of AIDS and in some cases even brings about immunity.**

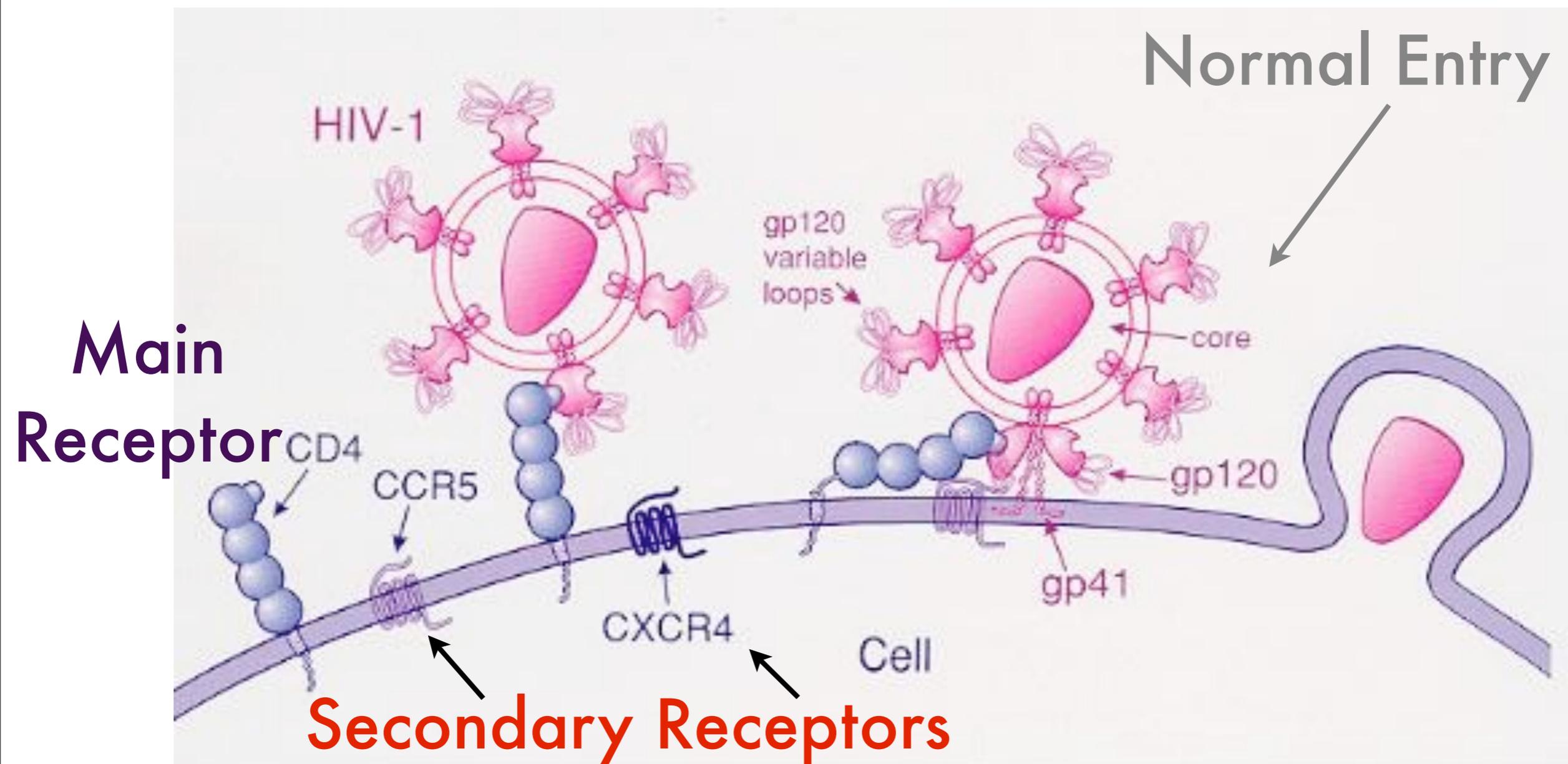


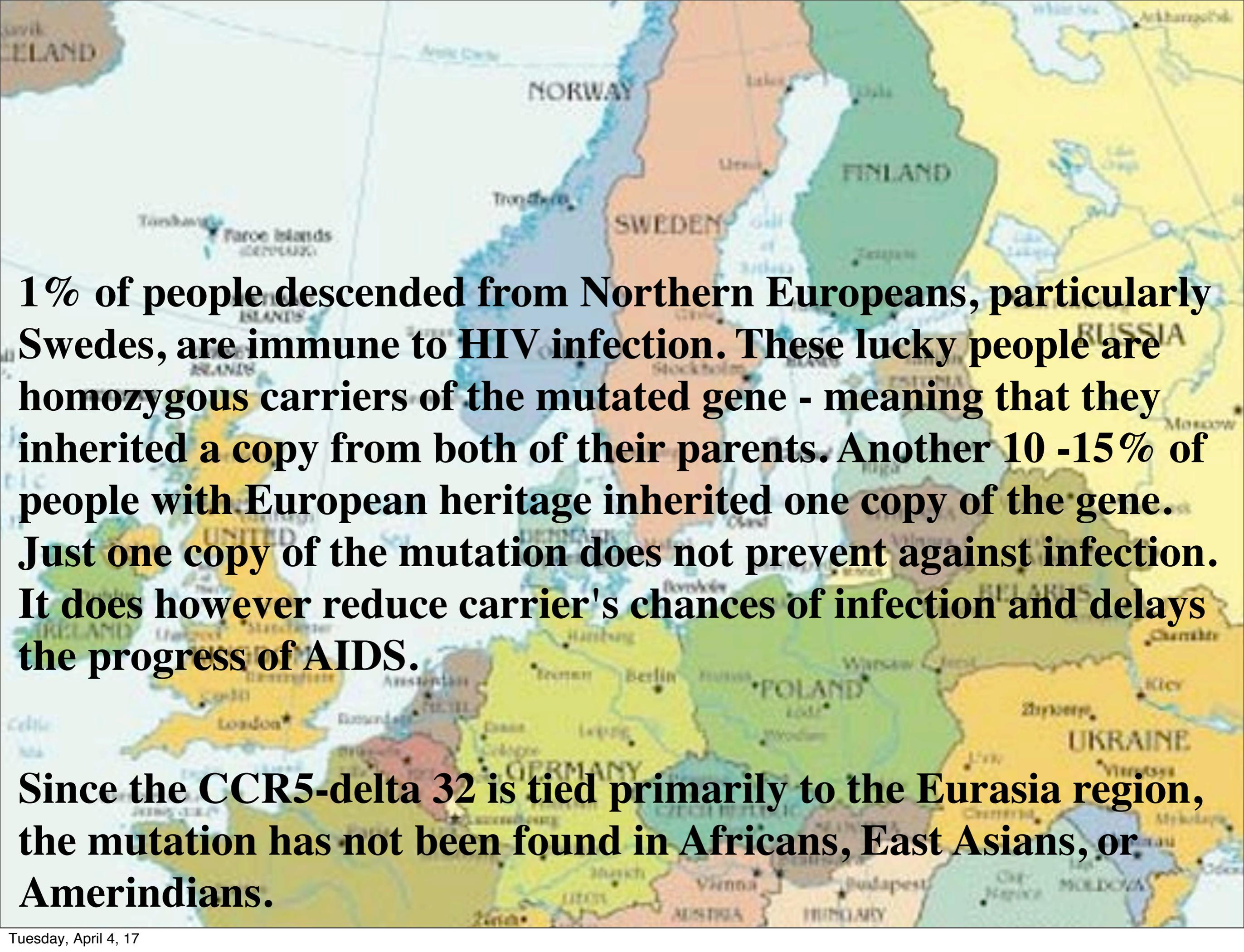
**HIV is the virus that causes the syndrome AIDS, which stands for acquired immune deficiency syndrome.**



**HIV's "main target" are CD4 immune cells. In the acute stages of infection the virus destroys a lot of CD4 cells and produces many virus particles.**

A genetic mutation known as CCR5-delta 32 is responsible for the two types of HIV resistance that exist. CCR5-delta 32 hampers HIV's ability to infiltrate immune cells. The mutation causes the CCR5 co-receptor on the outside of cells to develop smaller than usual and no longer sit outside of the cell. CCR5 co-receptor is like door that allows HIV entrance into the cell. The CCR5-delta 32 mutation in a sense locks "the door" which prevents HIV from entering into the cell.



A map of Europe and surrounding regions, including parts of North Africa, the Middle East, and Iceland. The map is color-coded by country: Norway (orange), Sweden (light blue), Finland (green), Poland (yellow), Germany (light green), and the UK (yellow). Major cities like London, Berlin, Warsaw, and Moscow are labeled. The text is overlaid on the map.

**1% of people descended from Northern Europeans, particularly Swedes, are immune to HIV infection. These lucky people are homozygous carriers of the mutated gene - meaning that they inherited a copy from both of their parents. Another 10 -15% of people with European heritage inherited one copy of the gene. Just one copy of the mutation does not prevent against infection. It does however reduce carrier's chances of infection and delays the progress of AIDS.**

**Since the CCR5-delta 32 is tied primarily to the Eurasia region, the mutation has not been found in Africans, East Asians, or Amerindians.**

# Why does the CCR5-delta 32 mutation appear in people of European descent only?



**One hypothesis suggests that the mutation originated in the Vikings.**



Researchers noticed that the mutation exhibits a north-to-south cline. The gene appears more frequently in Northern Europeans than it does in Southern Europeans. Some scientists attribute this pattern to the [Viking invasions](#).



Along this line of reasoning scientists have suggested that past epidemics were the driving force behind the prevalence of the mutation in Europeans. Scientists hypothesize that the mutation gave some sort of advantage to people against the epidemic. This gave these individuals an increased chance of survival and ability to reproduce and pass on the affected allele. Evidence dating the mutation back 700 years ago coincides perfectly with the Black Death. According to this idea, the Black Death drove natural selection in the human population. Those with the mutation were more likely to survive the plague and pass on their genes than those without which caused an increase in the percentage of people with the mutation.



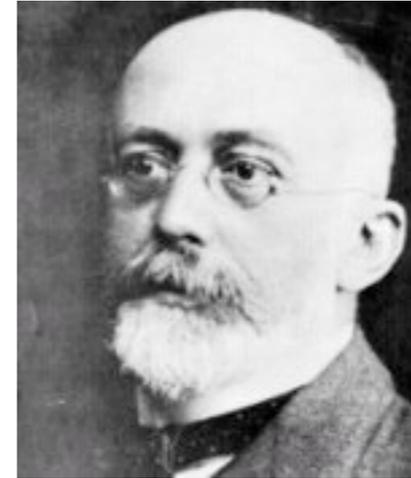
Smallpox is another epidemic that has been suggested. Those in favor of smallpox have continuity on their side. Unlike the Black Death, smallpox "has been continuous [for the last 700 years]" says Alison Galvani, a Yale University professor of epidemiology. Galvani notes that smallpox's longevity provided a reason for the mutation to continue throughout the generations. HIV and smallpox also share an important similarity. Both utilize the CCR5 receptor to infiltrate other cells. Coincidence? I don't know. But it is interesting to think that the mutation could have appeared several hundred years ago as a protective means against smallpox, survived through the generations, and then by chance have the ability to also provide HIV resistance.



*Essential knowledge 4.C.3: The level of variation in a population affects population dynamics.*

c. Allelic variation within a population can be modeled by the Hardy-Weinberg equation(s). [See also 1.A.1]

# Godfrey Hardy & Wilhelm Weinberg



- **A British mathematician and German physician asked this same question and their work tells us...**
- **Yes it is possible for allele/gene frequencies to remain unchanged in a population, Yes it is possible that a population does not evolve over time.**
- **However it is only possible provided the population meets certain criteria.**

# Hardy-Weinberg Equilibrium

- This principle describes a hypothetical non-evolving population
- In order for a population to maintain the Hardy-Weinberg equilibrium (not change) they must meet all 5 of the following conditions:

Very large population	No genetic drift can occur.
No emigration of immigration	No gene flow can occur.
No mutations	No new alleles can be added to the gene pool.
Random mating	No sexual selection can occur.
No natural selection	All traits must equally aid in survival.

**How likely is it that a population meets these conditions?**

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**How likely is it that a population meets these conditions?**

**Not at all likely, close to impossible!**

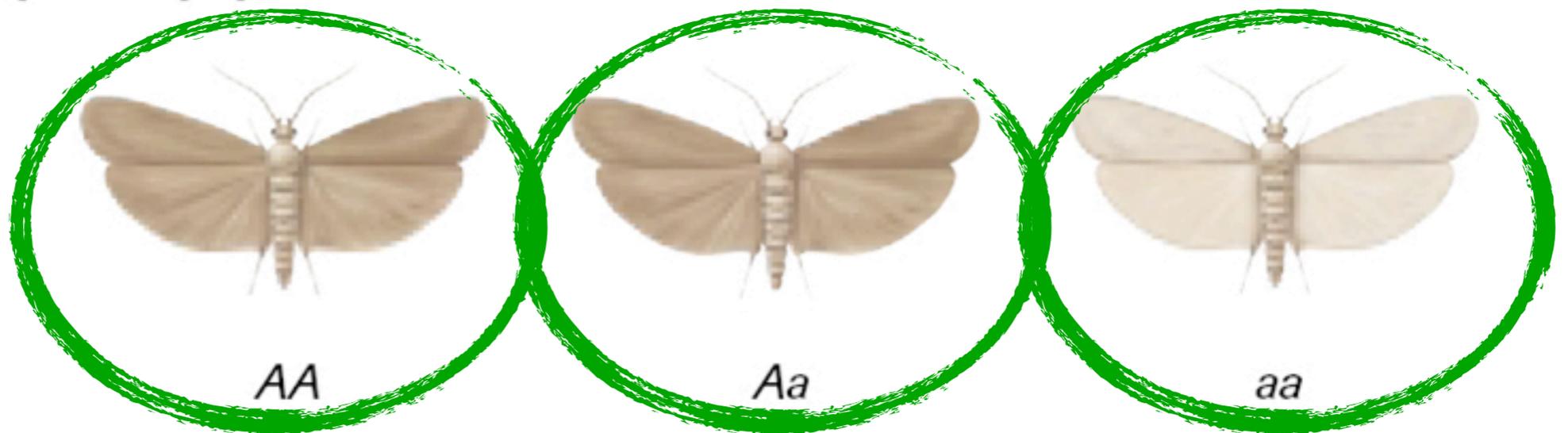
# Measuring Allele Frequency

- **To determine that evolution has occurred we must know the allele(s) frequency in a population at a point in time and compare it to the allele(s) frequency at a later time.**
- **This implies/requires that we can measure allele(s) frequencies...but HOW?**
  - *First we must know the total number of individuals in a population*
  - *Next we must know the frequency of a trait in a population.*
  - *Lastly we need to know which alleles control the trait we are examining*

# Genetic structure of parent population

*which alleles control the trait*

phenotypes



genotypes

AA

Aa

aa

number of moths  
(total = 500)

320

160

20

*population size*

*frequency of a trait in a population*

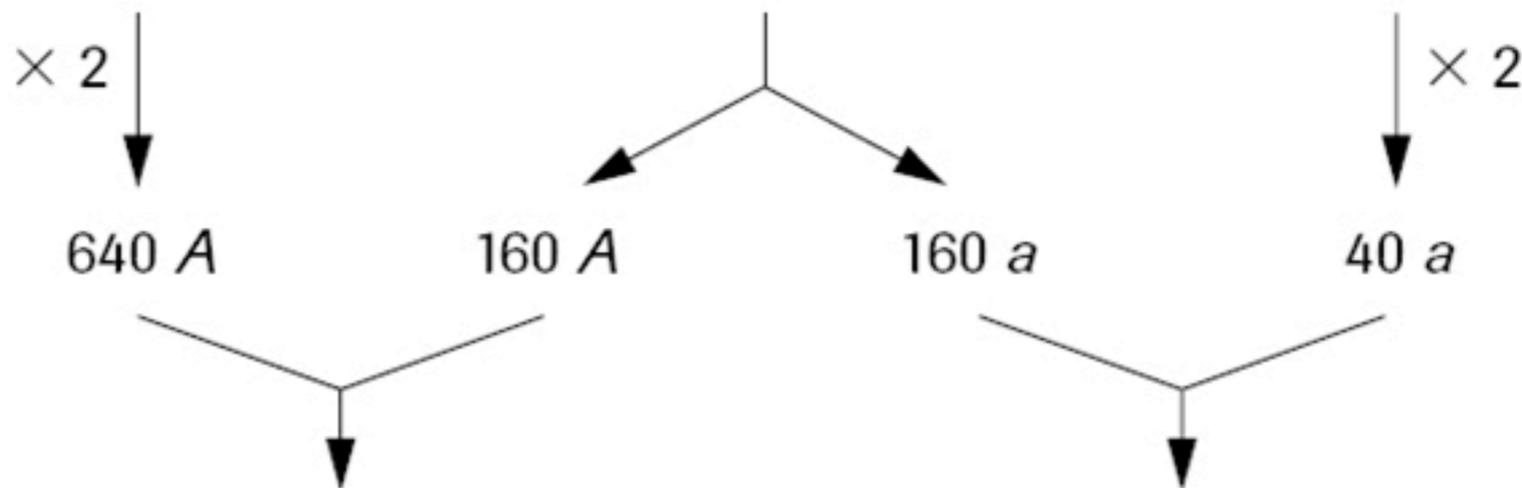
genotype frequencies

$$\frac{320}{500} = 0.64 \text{ AA}$$

$$\frac{160}{500} = 0.32 \text{ Aa}$$

$$\frac{20}{500} = 0.04 \text{ aa}$$

number of alleles  
in gene pool  
(total = 1000)



allele frequencies

$$\frac{800}{1000} = 0.8 \text{ A}$$

$$\frac{200}{1000} = 0.2 \text{ a}$$

**Bingo!**

$$p = \text{frequency of A} = 0.8$$

$$q = \text{frequency of a} = 0.2$$

# Measuring Allele Frequency

- To determine that the moth population is evolving we simply recalculate the allele(s) frequency in the population at a later time.
- If we find that the frequency of A is no longer 0.8 or 80% and a is no longer 0.2 or 20% then the population has evolved because its allele frequencies have changed.
- If we find that the frequency of A remains 0.8 or 80% and a remains 0.2 or 20% then the population has not evolved because its allele frequencies have not changed.

# Hardy-Weinberg Equilibrium

- **This principle describes a hypothetical non-evolving population**
- **When gametes are donated randomly and matings are also random allele frequencies will not change and we can calculate genotypic frequencies from allele frequencies:**
- **We can summarize the union of gametes in an algebraic equation**

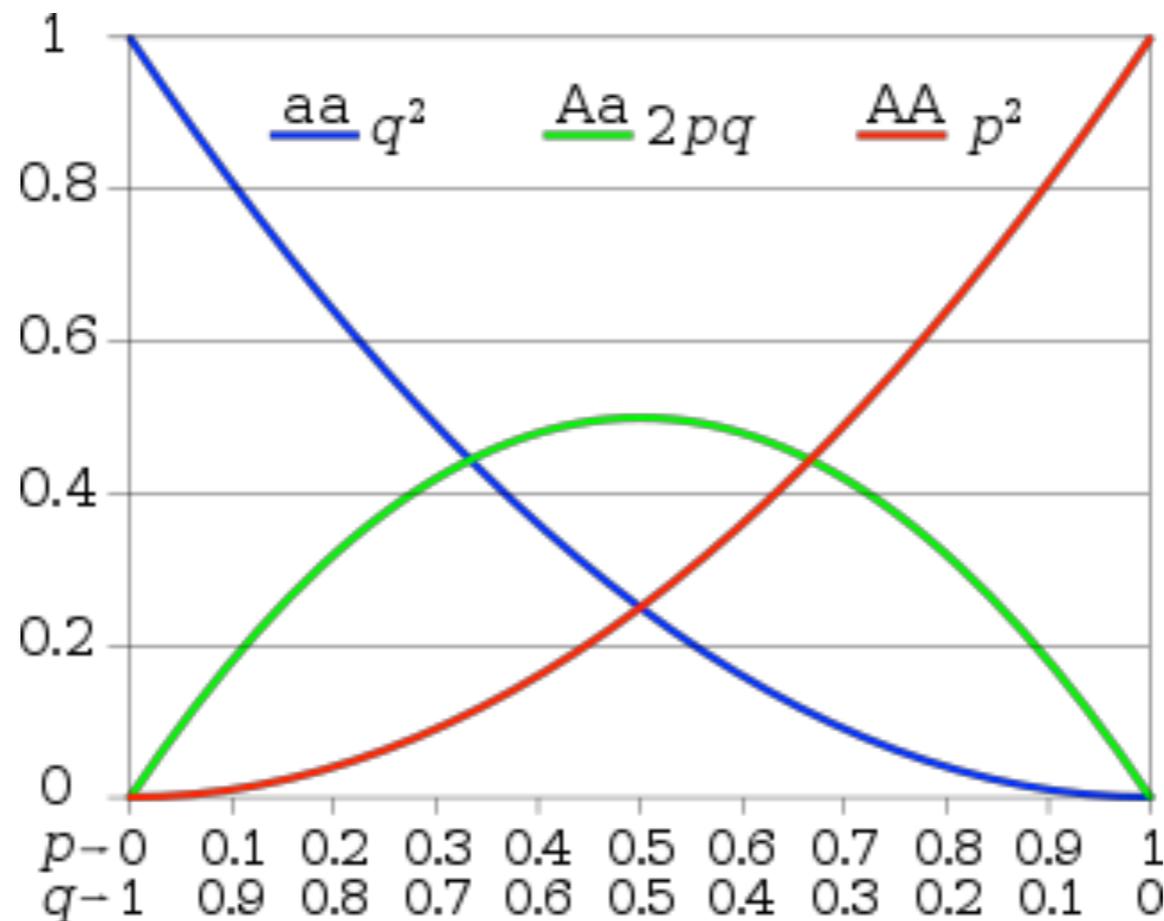
$$(p+q)(p+q) = p^2 + 2pq + q^2 = 1$$

# Hardy-Weinberg Equilibrium

Thus a locus with two alleles, the genotypes will appear in the proportions.

$$p^2 + 2pq + q^2 = 1$$

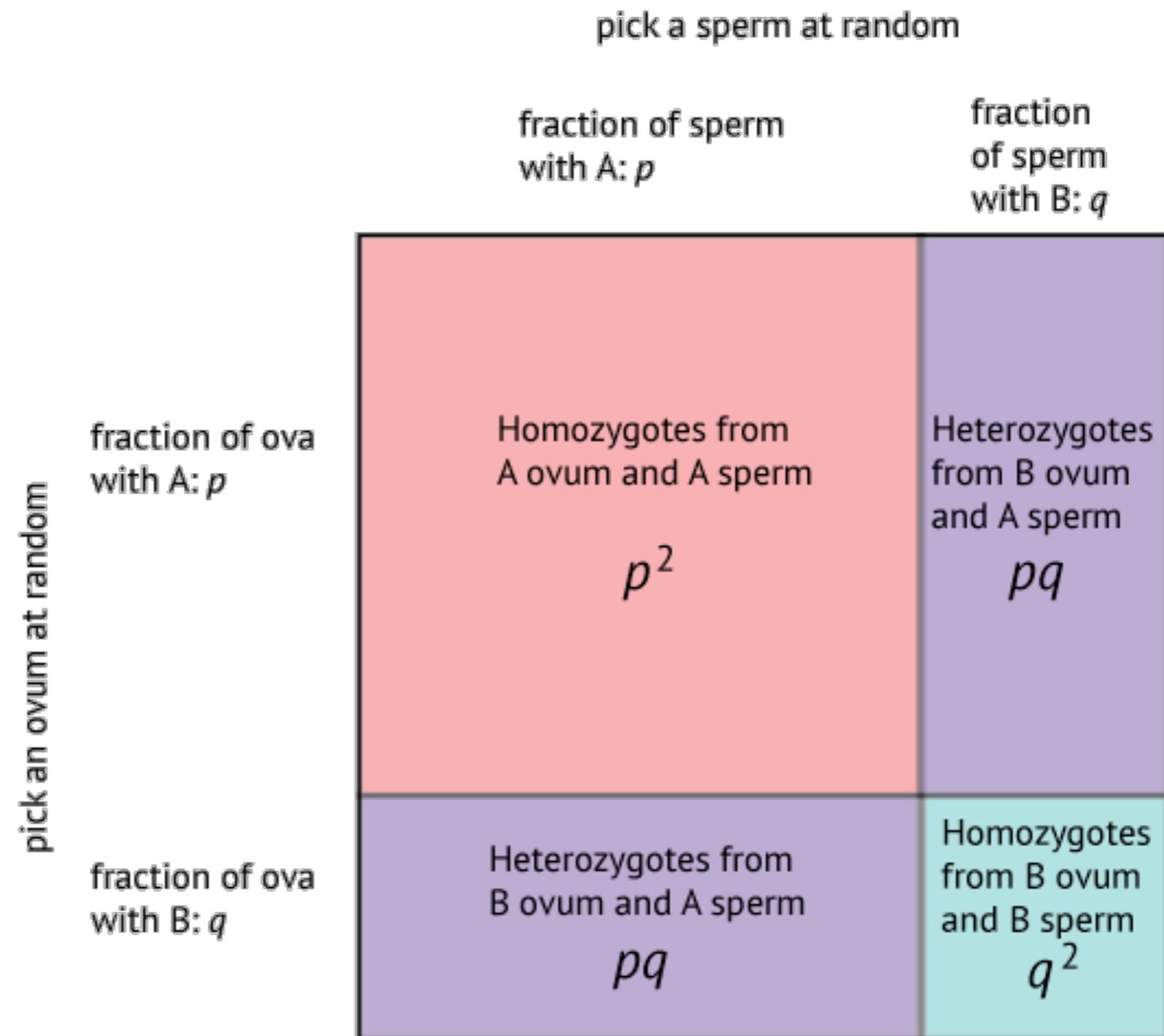
Here is graphic representation of allele and genotypic frequencies.



# Hardy-Weinberg Health Applications

## Hardy-Weinberg proportions in a geometric view

- The Hardy-Weinberg equation can be used to estimate the percentage of the population that carries an allele for a disease (among other things as well)



# Solving Hardy-Weinberg Problems

- Every Hardy-Weinberg problem is essentially the same, you will be given one (or more) of the 5 variables below, you will then use simple algebra to solve for the unknown.

## Know This

**p = dominant allele**

**q = recessive allele**

**p<sup>2</sup> = AA genotype**

**2pq = Aa genotype**

**q<sup>2</sup> = aa genotype**

## Follow these steps

First- determine the unknown (which of the variables on the left do need to solve for)

Next- determine the known (which of the variables on the left do we have)

Last- using the equations on the right and simple algebra solve for the unknown

## Use These

$$p^2 + 2pq + q^2 = 1$$

$$p + q = 1$$

# Hardy-Weinberg Equilibrium

**Let's try a tough problem together.**

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**There is a hypothetical population with 100 individuals. There is a trait with two alleles, B and b. 12 people are normal homozygous dominant (BB), 78 people are carriers of a disease (Bb) and 10 people have the disease (bb). Is this population evolving?**

# Hardy-Weinberg Equilibrium

Let's try a tough problem together.

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OK Where do we start? Well we just learned that the Hardy-Weinberg equation can help us determine (predict) if a population is evolving.  $p^2 + 2pq + q^2 = 1$

# Hardy-Weinberg Equilibrium

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OK Where do we start? Well we just learned that the Hardy-Weinberg equation can help us determine (predict) if a population is evolving.  $p^2 + 2pq + q^2 = 1$

Before we can predict what a non-evolving population looks, we need to know what the actual population looks like.

# Hardy-Weinberg Equilibrium

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**Start by finding alleles frequencies “p” and “q”.**

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**Population with 100 individuals will have 200 alleles.**

**12 people are normal homozygous dominant (BB) so  $(12)(2) = 24$  B's**

**78 people are carriers of a disease (Bb) so  $(78)(1) = 78$  B's and 78 b's**

**10 people have the disease (bb) so  $(10)(2) = 20$  b's**

# Hardy-Weinberg Equilibrium

**Start by finding alleles frequencies “p” and “q”.**

Population with 100 individuals will have 200 alleles.

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78 people are carriers of a disease (Bb) so  $(78)(1) = 78$  B's and 78 b's

10 people have the disease (bb) so  $(10)(2) = 20$  b's

Now  $24B + 78B = 102$  total B's out of 200 or  $102/200 = 0.51$  or 51% B's “p”

And  $78b + 20b = 98$  total b's out of 200 or  $98/200 = 0.49$  or 49% b's “q”

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**Remember “p” = dominant allele (B)**

**Remember “q” = dominant allele (b)**

**And since there are only two alleles  $p + q = 1$**

# Hardy-Weinberg Equilibrium

OK Now we have  $p$  and  $q$  let's plug them into the Hardy-Weinberg equation and see what a non-evolving population would look like.



# Hardy-Weinberg Equilibrium

OK Now we have p and q let's plug them into the Hardy-Weinberg equation and see what a non-evolving population would look like.

$$p^2 + 2pq + q^2 = 1$$

$$(0.51)^2 + 2(0.51)(0.49) + (0.49)^2 = 1$$

↓  
0.26BB

↓  
0.50Bb

↓  
0.24bb

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$$\begin{array}{ccc} \downarrow & \downarrow & \downarrow \\ 0.26BB & 0.50Bb & 0.24bb \end{array}$$

Now that we know what a non-evolving population looks like. We have to compare our “predicted” non-evolving population with our “actual” population

# Hardy-Weinberg Equilibrium

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Predicted Non-Evolving

Actual Population

# Hardy-Weinberg Equilibrium

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## Predicted Non-Evolving

$$(0.26)(100) = 26 \text{ BB}$$

$$(0.50)(100) = 50 \text{ Bb}$$

$$(0.24)(100) = 24 \text{ bb}$$

## Actual Population

$$12 \text{ BB}$$

$$78 \text{ Bb}$$

$$10 \text{ bb}$$

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If they are different then population **IS** evolving.

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**What genotype is being selected for?**

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## Actual Population

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**What genotype is being selected for?**

**Bb**

# Learning Objectives:

LO 4.25 The student is able to use evidence to justify a claim that a variety of phenotypic responses to a single environmental factor can result from different genotypes within the population. [See SP 6.1]

LO 4.26 The student is able to use theories and models to make scientific claims and/or predictions about the effects of variation within populations on survival and fitness. [See SP 6.4]