"Some of the great scientists, carefully ciphering the evidences furnished by geology, have arrived at the conviction that our world is prodigiously old, and they may be right but Lord Kelvin is not of their opinion. He takes the cautious, conservative view, in order to be on the safe side, and feels sure it is not so old as they think. As Lord Kelvin is the highest authority in science now living, I think we must yield to him and accept his views." [Mark Twain quoted by Burchfield (1990)]
Part I: The Eclipse of Darwinism.

- Between Scylla and Charybdis.


  b. By 1867, Darwin’s argument had been shredded:

    1. Selection requires heritable variation, and heritable variation would be lost at a rate of ~50% per generation – due to “blending” – Hat tip: Fleeming Jenkin.

    2. Gradual evolution requires earth ancient, but both the age of the habitable earth and that of the sun ≤ 100 million years – consequence of 2nd Law – Hat tip: William Thomson, later Lord Kelvin.
• By the end of the 19\textsuperscript{th} century:

a. Many biologists confidently predicted the imminent \textit{demise}, if it was not already dead, of what by this time was called “Darwinism”.

b. There was renewed interest in both an inherent tendency to progress and IAC – so-called “\textit{Neo-Lamarckism}”.

c. What kept the idea of evolution alive was increasing evidence – anatomical, developmental and \textit{especially} paleontological – for \textit{descent with modification}.

d. But come the new century, Jenkin’s and Kelvin’s objections would be answered.

1. \textbf{Particulate inheritance} (the rediscovered legacy of Mendel) preserved heritable variation.

2. \textbf{Earth’s true age}, as ascertained by \textbf{radiometric dating}, restored time in greater abundance than even Darwin and his acolytes had imagined.
Part II. Particulate Inheritance.

- Mendelism.
  a. “Experiments in Plant Hybridization” publ. in 1866.¹
  b. No one connected his work to the evolution debate.²
  c. Mendelian inheritance rediscovered around 1900.
    1. Initially believed to contradict gradual change.
    2. Discrete characters seen as evidence for “salta-
       tion”, i.e., all at once origin of new species.
    3. Wallace imagined Mendelian traits rare in nature
       – variations of small effect the “stuff of evolution”.
  d. In 1918, R. A. Fisher paved the way for the “Modern
     Synthesis” by showing that variability effectively
     continuous when many genes involved.

¹ Available in translation at [http://www.mendelweb.org/Mendel.html](http://www.mendelweb.org/Mendel.html)

² Mendel’s opinion about evolution remains a subject of dispute. Some sug-
gest that he viewed his results as supporting limits to variability, an opinion
that would have placed him in the anti-evolution camp. Certainly, he disa-
greed with Darwin’s claim that domestication promotes variability.
• The Not So Modern Synthesis.

a. **Genotype – Pheno-type** distinction.

b. **Both** genes and environment determine

1. Phenotype & hence

2. Probabilities of reproduction & survival, *i.e.*, fitness.

c. **Genes** the sole repositories of heritable characters.

d. **Mutation** the source of heritable variation.

1. Environment can affect mutation rates, **but** mutations **independent of need**.

2. Unlike IAC – especially via **and use and disuse**.

e. According to the Synthesis, evolution **is** changing gene frequencies.
• Review of Diploid Genetics.

a. Most eukaryotes have a diploid stage – \(2n\) chromosomes and 2 copies of each autosomal gene.

b. Sex cells are haploid.

1. \(\Rightarrow\) an “alternation of generations”.

2. Both haploid and diploid phases and can be uni- or multicellular.

c. Benefits of diploidy:

1. **DNA repair** (sister chromatids used as templates);

2. Production of new genotypes (**recombination**);

3. **Dominance**: Masks deleterious mutations; preserves genetic **variability**.

4. **Heterozygote advantage**.
• **Review of Mendelian Inheritance.**

a. Inheritance **particulate** – genes (Mendel’s “elementen”) pass unchanged from generation to generation.

b. In diploid species, two copies of each gene.

c. Genes **segregate**: each parent passes one copy to each offspring.

d. In accompanying example, T (tall) is **dominant**. All F1 individuals **tall**. In F2 generation, short individuals reappear. Tall: short ratio is 3:1.

e. If heterozygote phenotype intermediate, all F1 individuals **intermediate**; F2 ratios, 1:2:1.

f. Multiple characters **assort independently** – now known to be the result of genes being on **different chromosomes** or of **crossing over** during meiosis.
Mitosis vs. meiosis. Products of mitosis (left) are diploid and genetically identical to parental cells. Products of meiosis (right) are haploid and not genetically identical to parental cells. Lack of identity is due to crossing over.
Crossing over produces recombination. The probability of recombination between genes varies inversely with the distance between them. Guessing that this was the case allowed Thomas Hunt Morgan and his students to produce the first genetic maps.
1. **Independent Assortment** illustrated by the “dihybrid” cross at the right.

2. In the case shown, *R* (round) and *Y* (yellow) are both dominant.


• Questions.

1. Contrast the predictions of blending inheritance for F1 and F2 generations with those observed under Mendelian inheritance.

2. When would you expect independent assortment to break down?
g. Preceding can be summarized as Mendel’s Laws:\(^3\)

1. **Law of Segregation:** Each hereditary characteristic is controlled by two ‘factors’ (Mendel’s *Elementen*), which pass into separate germ cells.

2. **Law of Independent Assortment:** Pairs of ‘factors’ segregate independently of each other when germ cells are formed.

• Mendel’s Motivation.

a. **Hybridization** had long been suggested as a way of producing **new species**.

b. Mendel interested in the **stability of hybrids**.

c. Calculated that continued selfing leads to **elimination** of heterozygotes, *i.e.*, hybrids **unstable** – see Table next page.

1. Elimination of hybrids by selfing an extreme example of **finite population effects** – see below.

2. **Contrary** to the idea that new species can be produced by hybridization as had been advocated by Lamarck.

3. **Historical note:** Mendel’s experiments were **massive** – the entire monastery recruited to help. Not the work of an amateur frolicking in the pea patch one summer.
### Selfing Leads to Heterozygote Elimination.

(Assume Four (4) Seeds per Cross)

<table>
<thead>
<tr>
<th>Cross / Genotype</th>
<th>AA</th>
<th>Aa</th>
<th>aa</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1st Generation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aa x Aa</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><strong>1st Generation Total</strong></td>
<td>1 (.25)</td>
<td>2 (.50)</td>
<td>1 (.25)</td>
</tr>
<tr>
<td><strong>2nd Generation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA x AA</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2 (Aa x Aa)</td>
<td>2</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>aa x aa</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td><strong>2nd Generation Total</strong></td>
<td>6 (.375)</td>
<td>4 (.25)</td>
<td>6 (.375)</td>
</tr>
<tr>
<td><strong>3rd Generation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 (AA x AA)</td>
<td>24</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4 (Aa x Aa)</td>
<td>4</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>6 (aa x aa)</td>
<td>0</td>
<td>0</td>
<td>24</td>
</tr>
<tr>
<td><strong>3rd Generation Total</strong></td>
<td>28 (.4375)</td>
<td>8 (.125)</td>
<td>28 (.4375)</td>
</tr>
<tr>
<td><strong>4th Generation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>28 (AA x AA)</td>
<td>112</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>8 (Aa x Aa)</td>
<td>8</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>28 (aa x aa)</td>
<td>0</td>
<td>0</td>
<td>112</td>
</tr>
<tr>
<td><strong>4th Generation Total</strong></td>
<td>120 (.46875)</td>
<td>16 (.0625)</td>
<td>120 (.46875)</td>
</tr>
</tbody>
</table>

- **Question.**

3. Continue preceding table into the 5th generation.
Population Genetics.

- Hardy-Weinberg Law.

  a. If mating random, genotype frequencies determined by gene frequencies.

  b. 1 locus; 2 alleles: $A$, $a$.

  1. Three genotypes: $AA$, $Aa$, $aa$.

  2. If gene frequencies $p$ and $q$. H-W genotype frequencies are:

      
      
      \[ p_{AA} : p_{Aa} : p_{aa} = p^2 : 2pq : q^2 \]  

      

  3. Follows from the fact that the joint probability of two independent events is given by the product of the individual probabilities.

  4. Since $p + q = 1$, Eq(1) can also be written as

      
      
      \[ p_{AA} : p_{Aa} : p_{aa} = p^2 : 2p(1 - p) : (1 - p)^2 \]  

      

H-W frequencies result from random mating between male and female parents.
c. In **infinitely large**, random-mating (**panmictic**) populations, H-W frequencies established in **one** generation.

d. Observation of H-W frequencies suggests that **no other forces** affecting gene frequencies operative.

H-W genotypic frequencies vs. gene frequency. Here \( p \) is the frequency of gene \( A \), and \( q = 1 - p \) is the frequency of gene \( a \).
Questions.

4. Suppose there are three alleles, $A_1$, $A_2$, $A_3$, with gene frequencies $p$, $q$, $r$. How many genotypes are there? What are their H-W frequencies?

5. Suppose the genotypic frequencies, $p_{AA}$, etc., are 0.10, 0.50 and 0.40. a. What are the gene frequencies? b. Is the population in Hardy-Weinberg equilibrium?

6. What might one conclude if a population is not in H-W equilibrium?
• Equilibria and their Stability.

a. A system is at equilibrium if, *absent external disturbances*, its state *doesn’t* change.

b. Kinds of equilibria.

1. **Stable:** Following a perturbation, the system **returns to equilibrium**.

2. **Unstable:** Following a perturbation, the system **moves further away** from equilibrium.

3. **Neutrally Stable:** Following a perturbation, system **neither** returns to nor **moves further away** from equilibrium – *e.g.*, a **frictionless** pendulum.

c. Hardy-Weinberg equilibria **neutrally stable**. Changing gene frequencies =>
   1. New genotype frequencies.
   2. No tendency to return to or diverge further from old.
• **Forces that Affect Gene Frequencies.**

a. **Selection.** Different genotypes differentially represented in the next generation due to different probabilities of reproduction / survival.

b. **Mutation.** Changes in DNA base pair sequence – sometimes reversible \((A \rightleftharpoons a)\); sometimes not.

c. **Migration.** Gene frequency among immigrants can differ from those among residents.

d. **Genetic Drift.** Gene frequency change due to sampling error.

1. *E.g.*, 50% chance an offspring of a heterozygote mating is homozygous.

2. Result is loss of alleles – esp. in small populations, and especially if selection is weak. – See **Announcements**

Simulated genetic drift in small (top) and large (bottom) populations.
e. **Temporary** small population size effects:

1. **Founder effect.**

2. Periodic **bottlenecks** of small population size.

Low genetic diversity in cheetahs is conjectured to have resulted from population size bottlenecks From O’Brian *et al.* 1987. *PNAS.* 84:508-511.
Finite Population Size, i.e., $N < \infty$, Reduces Heterozygosity by Promoting Inbreeding.

a. Even if gene frequencies don’t change.

b. Recall Mendel’s selfing calculations.

c. More generally, random mating implies crossings between individuals of differing degrees of relatedness – brother-sister, cousin-cousin, etc.

1. More closely related individuals, more likely to share genes that are same by descent.

2. In infinite populations, everyone unrelated $\Rightarrow$ H-W genotypic frequencies.

3. In finite populations, all individuals related and therefore more likely to have same genes. Frequency of heterozygotes, $p_{\text{het}} \to 0$ as $t \to \infty$.

4. $\Rightarrow$ more frequent expression of recessive, often deleterious, alleles – think hemophilia.

d. In real world, loss of alleles and heterozygosity countered by mutation and migration.
• Selection: How it Works.

a. Assign **fitness values**, $w_{AA}, w_{Aa}, w_{aa}$ (usually scaled on $[0,1]$, and **always** $\geq 0$) to each genotype.

b. If the **initial value** of $\rho$ is **neither 0 nor 1**, the following results obtain.

<table>
<thead>
<tr>
<th>Case</th>
<th>Fitness Relations</th>
<th>Long-Term Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$w_{AA} &gt; w_{Aa} &gt; w_{aa}$</td>
<td>$p \rightarrow 1$</td>
</tr>
<tr>
<td>2</td>
<td>$w_{AA} &lt; w_{Aa} &lt; w_{aa}$</td>
<td>$p \rightarrow 0$</td>
</tr>
<tr>
<td>3</td>
<td>$w_{AA} &lt; w_{Aa} &gt; w_{aa}$</td>
<td>$p \rightarrow p^<em>, 0 &lt; p^</em>&lt; 1$</td>
</tr>
<tr>
<td>4</td>
<td>$w_{AA} &gt; w_{Aa} &lt; w_{aa}$</td>
<td>$p \rightarrow 0$ or $p \rightarrow 1$</td>
</tr>
</tbody>
</table>

c. In case 1, fitness **increases** with the number of copies of $A$, and the frequency of $A \rightarrow 1$.

d. In case 2, fitness **decreases** with the number of copies of $A$, and the frequency of $A \rightarrow 0$. 
e. In case 3 (heterozygote advantage), the heterozygote has greater fitness than the homozygotes, and there is a stable equilibrium

\[ p_A^* = \frac{w_Aa - w_{aa}}{(w_{Aa} - w_{AA}) + (w_{Aa} - w_{aa})} \]  

between 0 and 1, i.e.,

\[ 0 \leq p_A^* \leq 1. \]

f. In case 4 (heterozygote inferiority), the heterozygote is less fit than the homozygotes,

i. There is an unstable equilibrium, \( p_A^* \), given by Eq (6), and

ii. \( p_A \) goes to 0 or 1 depending on its initial value.

g. One can show, but we will not, that gene frequency, \( p \), changes so as to maximize the average fitness of the population defined by the equation

\[ \bar{W} = p_{AA}w_{AA} + p_{Aa}w_{Aa} + p_{aa}w_{aa}. \]
Response to selection. **Top left.** Gene $A$ replaces $a$. **Top right.** Gene $a$ replaces $A$. **Bottom left.** Case of heterozygote advantage. There is a stable equilibrium, $0 < p_A^* < 1$, and both genes are maintained in the population. **Bottom right.** Heterozygote inferiority. Either $A$ or $a$ is fixed (frequency goes to 1) depending on the initial frequency, $p$. 
Questions.

7. In the case of heterozygote *superiority*, is $p^*$ stable or unstable?

8. In the case of heterozygote *inferiority*, is $p^*$ stable or unstable?
• Malaria and Sickle Cell Anemia.

a. Hemoglobin (Hb) transports oxygen in red blood cells (RBCs).

b. Hb composed of four polypeptide chains: two $\alpha$- and two $\beta$-chains.

c. **Substitution** of one amino acid in HbS/HbS individuals causes the $\beta$-chains of **deoxygenated** HbS to polymerize, forming **rigid fibers that collapse** RBCs.

d. **Cycling** between polymerized and de-polymerized states causes the cells to aggregate into **fibrous threads**.

e. Threads obstruct small blood vessels; causes **hypoxia** (lack of oxygen), **tissue / organ damage**.

f. *E.g.*, “cerebral malaria”, which is often fatal.

"Sickling" of an RBC drawn from an individual suffering from sickle cell anemia.
g. Clinically, HbS/HbS homozygotes “present” as **sickle cell anemia**.

h. => life-long morbidity / reduced longevity (~ 45 years in the US).

i. Heterozygotes present as **sickle cell trait** – usually, but not always, **benign** – *i.e.* there is an increased **risk** of exercise-related death.

j. In **presence of malaria**, heterozygotes have **greater** life expectancy than Hb/Hb homozygotes.

k. Heterozygote advantage **maintains** the HbS gene in **malaria-exposed** populations despite low fitness of HbS/HbS homozygotes.

l. There are at least a dozen other mutations that also confer resistance to malaria – *e.g.*, pyruvate kinase deficiency, a metabolic disorder causing anemia by disrupting glycolysis.
Questions.

9. Although estimated fitness values for the three genotypes, Hb/Hb, Hb/HbS and HbS/HbS, in the presence of malaria vary, the following numbers are probably reasonable:

\[ w_{Hb/Hb} = 0.88; \quad w_{Hb/HbS} = 1.0; \quad w_{HbS/HbS} = 0.14. \]

10. Using Eq (6), compute the equilibrium frequency of HbS.

11. Do you expect the frequency of HbS to be higher or lower among American than African blacks? Give two reasons why or why not?

Malaria infects some 300 million souls and kills 1-2 million yearly. Left. Parasite distribution in the Old-World tropics. Right. Frequency of HbS in human populations in Africa and Asia. Pre-DDT, malaria was present in the U. S., Europe and northern Asia.
• Mutation–Selection Balance for Recessive Mutants.

a. Assume

1. Fitness values 1, 1, and 1-s, where 0 < s ≤ 1,

2. Wild type mutates to mutant at rate \( \mu \ll 1 \).

b. Equilibrium frequency, \( q^* \), of the recessive mutant thus determined by opposing forces:

1. Mutation increases \( q^* \).

2. Selection reduces \( q^* \).

c. At equilibrium, one can show (but we will not) that

\[
q^* \approx \sqrt{\frac{\mu}{s}}, \quad \text{if } \mu \ll 1 \quad (10)
\]

1. Reducing the fitness, of the mutant, \( i.e., \) making \( s \) larger, reduces \( q^* \).

2. If \( q \) small, \( 2q(1 - q) \approx 2q \gg q^2 \).

3. **Impossible** to eliminate mutants by selection, \( i.e., \) fraction mutant genes in heterozygotes \( \to 0 \)
d. If the mutant is **dominant**, selection more effective because **both** heterozygotes and homozygotes are exposed to selection and their numbers **reduced**.

e. Recessive mutations are **more difficult to eliminate** than dominant mutations because **only** homozygotes are exposed to selection.

f. Important implications for animal breeding and human eugenics.

g. Appreciated by 20\textsuperscript{th} century eugenists who nonetheless were enthusiastic in their advocacy for “racial hygiene”.
12. Tay-Sachs disease (TSD) occurs in frequencies of .03-.04 in Jews of Eastern European extraction. TSD is a neurodegenerative disorder caused by a single autosomal mutation. In its most common form, it is almost invariably fatal by age 4. Assume the disease only presents in homozygotes (two copies of the mutation) and a mutation rate of $2 \times 10^{-6}$.

a. Compute the equilibrium frequency of TSD according to Eq (10). Hint: What is the value of the coefficient of selection, $s$?

b. What do you conclude?
• Migration.

a. Most species divided into local populations coupled by migration. Consequence of

1. Inherent limitations to dispersal;

2. Geographic barriers.

b. Because conditions vary spatially, gene frequencies often vary along geographic transects

1. E.g., increasing body size in mammals as one goes from equator to poles (Bergmann’s rule – next page).

2. Consequence of selection for larger body size in cold climates.

c. Gene flow (consequence of migration)

1. Retards local differentiation that would otherwise result from differing selective regimes.

2. Holds species together genetically, i.e., prevents gene frequency divergence of local populations.
The range of white-tailed deer (*Odocoileus virginianus*) extends from Canada to the Amazon basin. There is a strong size gradient, with the largest animals in the north (left) and the smallest in the tropics (right).
Part III. The “Stuff” of Evolution.

- Quantitative Genetics.

  a. Studies “continuous” variation.

    1. Examples include size, crop yield, fat content in meat, IQ, blood pressure, bristle number in fruit flies. *etc.*

    2. Reflects polygenic (many genes) control of most quantitative traits. Recall Fisher’s reconciliation of Mendelism and Darwin-Wallace selection.

    3. Quantitative traits typically under both environmental and genetic control.

Distribution of a quantitative character in a population. **Left.** Raw data. **Right.** Continuous distribution inferred therefrom.
b. The Big Picture:

1. **Both** Mendelian and quantitative genetics are **models** of heredity.

2. Mendelian genetics focuses on allelic frequencies and **phenotypically discrete** genotypes;

3. Quantitative genetics focuses on **distributions** of **trait values** that are more or less **continuous**.

<table>
<thead>
<tr>
<th>Appropriate Genetic Model</th>
<th>Within Genotype Variability</th>
<th>Between Genotype Variability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mendelian</td>
<td>Small</td>
<td>Large</td>
</tr>
<tr>
<td>Quantitative</td>
<td>Large</td>
<td>Small</td>
</tr>
</tbody>
</table>

[Graphs showing frequency distribution for quantitative and Mendelian models]
Heritability of Quantitative Traits.

a. Heritability, $h^2$, is estimated by comparing traits, *e.g.*, height, of parents and offspring.

b. $h^2$ is the **slope** of the regression line: offspring trait value *vs.* mean parent trait value.

c. The following formula is used to predict offspring trait value from mean parental value:

$$T_o = \bar{T} + h^2(\bar{T}_p - \bar{T})$$  \hspace{1cm} (11)

where

$T_o$ is the expected offspring trait value.

$\bar{T}_p$ is the parental mean.

$\bar{T}$ is the population mean.

Offspring vs. parental height as determined by Darwin’s cousin, Francis Galton in 1889.
d. Implications of Eq (11):
1. \( h^2 = 0 \Rightarrow T_o = \bar{T} \)
2. \( h^2 = 1 \Rightarrow T_o = \bar{T}_p \)
3. \( \bar{T}_p = \bar{T} \Rightarrow T_o = \bar{T} \)

e. High heritability does not necessitate a genetic basis.

f. **Threshold selection.**

1. *E.g.*, animal breeding.
2. Only individuals above threshold allowed to reproduce.
3. Distribution more or less reestablished the next generation.
4. Mean is shifted.

Selection for a quantitative trait. In this case, only those individuals for which the trait exceeds some threshold are permitted to reproduce.
Questions.

13. Parent-offspring comparisons in humans would yield high heritability for life-time earning and religion – *i.e.*, the children of the rich tend to be rich, *etc.* Does this necessitate the existence of “poverty genes”? Explain.

14. In assessing whether or not intelligence has a heritable basis, twin studies are often used. Design such a study.

15. Assume the average IQ in a population is 100 and $h^2 = 0.4$. What is the expected IQ of the daughter of parents whose IQs are 120 and 110? What about the IQ of a child born to parents with IQs of 90?

16. Comparing the offspring’s expected IQ to that of the parents and to the population mean, what do you conclude?
• Types of Selection on Quantitative Characters.

Stabilizing (left), directional (center) and disruptive (right) selection. Blue arrows indicate phenotypes selected against.
• **Directional Selection in Cliff Swallows.**

a. Mammals and birds maintain **constant** body temperature.

b. In cold environments, heat lost to the environment **must be balanced** by heat generated metabolically.

c. Small individuals have a heat loss problem due to **large surface area to volume ratios**.

d. In the case of **insectivorous** birds, cold weather further **reduces food availability** and therefore the rate at which heat can be generated.

e. Cold weather should select for **larger** body size.

f. Following a series of severe breeding season storms (cold, rainy weather) in Nebraska, **differential mortality** was observed with small birds being more likely to wind up dead below the nests.
Foot length in dead (black) and surviving (shaded) cliff swallows following a cold-snap in Nebraska during the breeding season.
• Stabilizing Selection in Humans.

Infant mortality (pre- and post-partem) superimposed on frequency distribution of birth weights. The mean birth weight is about half a pound less than the optimum as judged by infant survival.
• **Disruptive Selection in Finches.**

a. Selection for **small and large bills** in an African finch. (black bellied seed cracker)

b. Feed principally on **seeds** of two species of grass-like plants (sedges) that live in wetlands.

   1. Seeds about the same size.

   2. Differ dramatically in **hardness**.

   3. Smaller bills **better** for manipulating seeds but can’t crack hard ones.

   4. Larger bills **better** for cracking hard seeds.

c. Result is selection **for** small and large bills and **against** bills of intermediate size.

d. **Supported** by observation of differential juvenile survival depending on bill size – see figure on next page.
Selection for small and large bills in black bellied seed crackers. Light orange bars represent all juveniles; dark orange bars, those that survived to adulthood.
• Questions.

17. Which of the three modes (stabilizing, directional, disruptive) of selection always increases phenotypic variance? Which mode shifts trait frequency distributions?

18. Which of the three modes of selection is often a consequence of trade-offs and could be responsible for evolutionary stasis?

19. How might you account for the fact that the observed mean birth weight of humans is slightly less than the optimum?

20. The following species nest in Alaska where summer nights can be cold: golden eagle, great horned owl, kingbird, rufous hummingbird, yellow-shafted flicker. One of these goes torpid (reduced metabolic rate and body temperature) at night. Which species do you imagine does this? Explain.

21. Small cliff swallows are more agile fliers than large individuals and therefore more likely to be able to capture insects on the fly. How does this affect selection for body size in this species?