GENERAL BIOLOGY · STUDY GUIDE

Exam 4 Study Guide

This guide covers chapters 7 and 8. First you see how cells decide which genes to use, how that builds an embryo, and how the tools of biotechnology read and rewrite DNA. Then you step back to populations and learn how evolution works and how to measure it with the Hardy-Weinberg equation. Work one chapter at a time. Read the "what to know" paragraphs, copy the tables out by hand, then try each worked case with the steps covered. Finish each chapter with its ten self-test items. The cram sheet at the end holds every switch, tool and formula.

WHAT THIS GUIDE COVERS

- Chapter 7 · the lac and trp operons, cis and trans control, chromatin and histone marks, DNA methylation, enhancers and combinatorial control, microRNAs, epigenetics, development and Hox genes, stem cells, restriction enzymes, cloning, gels, PCR, sequencing, CRISPR and ethics.
- Chapter 8 · Darwin and the thinkers before him, natural selection, evidence for evolution, homology and analogy, Hardy-Weinberg and its five conditions, carrier frequencies, drift, gene flow, modes of selection, sexual and balancing selection, and the limits of selection.

CHAPTER 7

Gene Regulation and Biotechnology

Read it in full: **Chapter 7 in the textbook**. See it in motion: **Gene Regulation Lab** and **Biotechnology Lab**.

What to know

Cells save resources by making proteins only when needed. Bacteria control most genes at the start of transcription. An **operon** is a promoter, an **operator** (the on and off switch) and several structural genes read as one mRNA. A **repressor** made by a separate regulatory gene binds the operator and blocks RNA polymerase. The repressor is **trans-acting** (a diffusible protein); the operator is **cis-acting** (it controls only the genes attached to it).

The **lac operon** is **inducible**. With no lactose, the repressor sits on the operator. When lactose arrives, some becomes allolactose, the **inducer**, which pulls the repressor off. Strong expression also needs **cAMP-CAP**, an activator that binds only when glucose is low and cAMP is high. So transcription is high only when lactose is present and glucose is absent. With both sugars present, output is low and the cell uses glucose first (diauxie). Mutants revealed the logic: $lacI^-$ and $lacO^c$ are always on, and a super repressor can never be induced.

The **trp operon** is **repressible**. It is normally on. Tryptophan acts as a **corepressor**, switching the repressor on so it binds the operator. **Attenuation** adds a second dial: when tryptophan is plentiful, a ribosome speeds through the leader, regions 3 and 4 form a terminator and transcription stops early. Inducible operons usually serve catabolic pathways; repressible ones serve anabolic pathways. Bacteria also use alternative sigma factors, riboswitches and quorum sensing.

Eukaryotes can regulate at every step: chromatin, transcription, RNA processing, mRNA transport and stability, translation, and protein processing and destruction. **Histone acetylation** neutralizes lysine charges and opens chromatin; deacetylases close it. Histone methylation can activate or repress depending on the site. **DNA methylation** at CpG sites usually silences genes, and the pattern is copied at each replication.

RNA polymerase II needs general transcription factors at the promoter (TFIID binds the TATA box). **Activators** bound to **enhancers**, which may be far away, bend the DNA, contact the mediator complex and recruit acetyltransferases. **Combinatorial control** lets a few thousand factors run about 20,000 genes: the lens cell and liver cell both carry the crystallin gene, but only the lens cell has the full set of activators for its enhancer. After transcription, alternative splicing, mRNA lifetime, **microRNAs** (about 22 nucleotides, which block translation or cut targets) and ubiquitin tagging for the proteasome all tune the output.

Epigenetics is heritable change in gene expression without a change in the DNA sequence. X inactivation (XIST RNA and the Barr body), genomic imprinting (Prader-Willi and Angelman syndromes) and diet effects in agouti mice are examples. Cancer cells often silence tumor suppressors by promoter methylation, and drugs that block methyltransferases or deacetylases can switch genes back on.

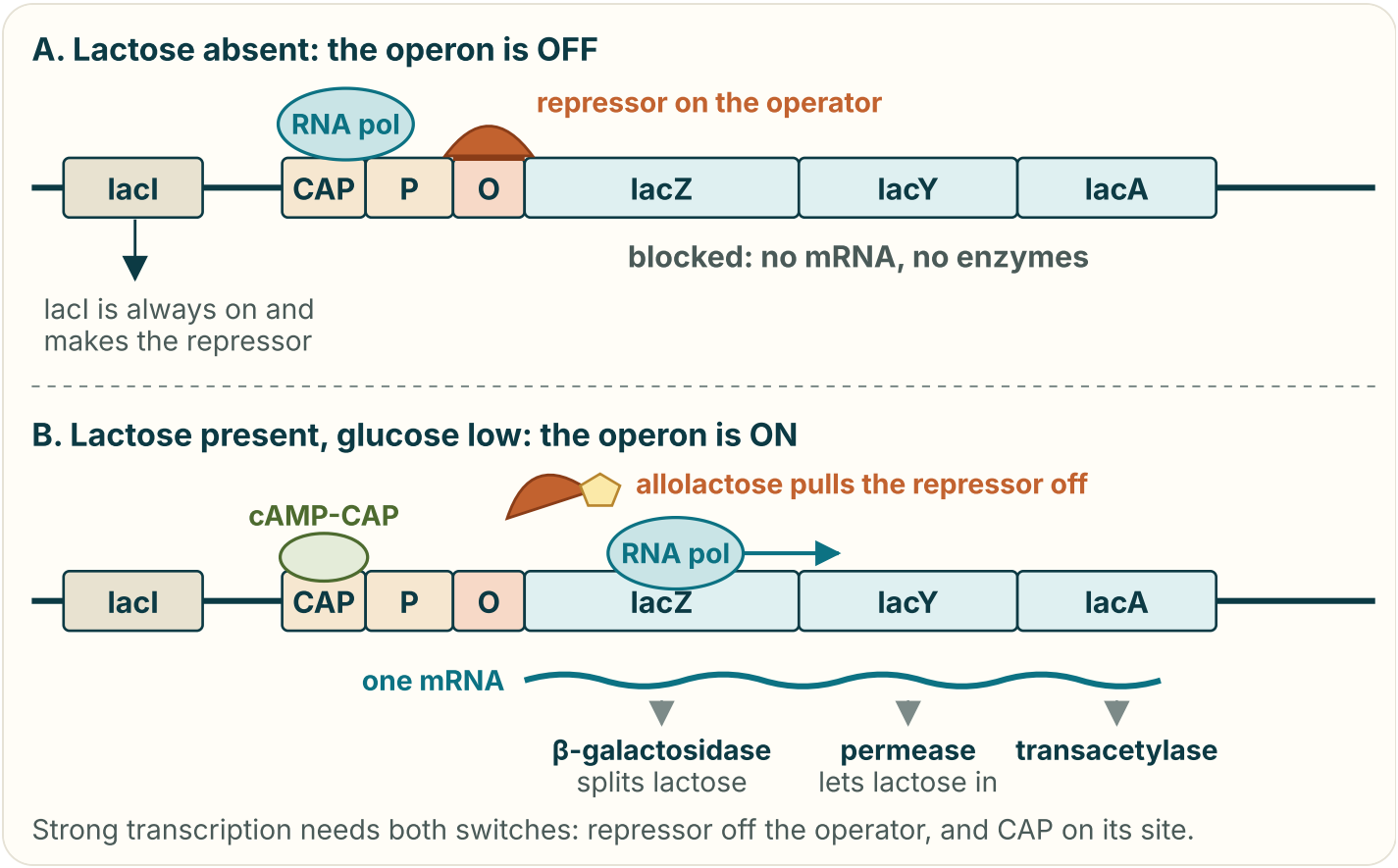
Development uses one genome to make many cell types by **differential gene expression**. Cloned frogs and Dolly the sheep showed that differentiated nuclei keep all their genes. **Determination** commits a cell; **differentiation** makes it look specialized. MyoD, a master regulator, switches itself on and locks in a muscle fate. Cells learn position from **cytoplasmic determinants** in the egg and from **induction** by neighbors. A **morphogen** gradient gives positional information.

In flies, maternal **bicoid** mRNA at the front makes a protein gradient. It switches on gap genes in broad bands, then pair-rule genes (seven stripes), segment polarity genes (fourteen stripes) and finally **Hox genes**, which give each segment its identity. Hox mutants have the right number of segments with the wrong identity: Antennapedia puts legs on the head. Every Hox gene has a 180 nucleotide homeobox, and Hox genes are lined up head to tail on the chromosome in flies and mammals (colinearity).

A **totipotent** cell (zygote) can make a whole organism, including the placenta. A **pluripotent** cell (embryonic stem cell) can make every body cell type. A **multipotent** cell (blood stem cell) makes a limited set. Adding four factors (Oct4, Sox2, Klf4, c-Myc) turns adult cells into **iPS cells**.

Biotechnology tools: **restriction enzymes** cut palindromic sites (EcoRI cuts GAATTC, leaving sticky ends) and **ligase** joins pieces. Plasmid vectors carry an origin, an antibiotic resistance gene and a cloning site; blue-white screening finds inserts. Eukaryotic genes go into bacteria as **cDNA** from reverse transcriptase. In **gel electrophoresis**, negatively charged DNA runs to the positive pole and short pieces run farthest. **PCR** repeats denaturation (94 to 98 °C), annealing (50 to 65 °C) and extension (72 °C) with Taq polymerase; n cycles give up to 2^n copies. **Sanger sequencing** uses dye-labeled ddNTPs that lack a 3′ OH and stop the chain.

CRISPR-Cas9 uses a guide RNA with a 20 nucleotide spacer. Cas9 needs a PAM (NGG) and cuts both strands about 3 bp upstream of it. Nonhomologous end joining leaves small indels that knock out the gene; homology-directed repair copies a supplied template to make a precise edit. Uses include recombinant insulin (1982), gene therapy, CAR T cells, the 2023 CRISPR therapy for sickle cell disease, STR profiling with 20 loci in the US database, Bt crops and Golden Rice. Ethics separates three questions: is it safe, is it effective, and is it right. Somatic editing affects one patient; germline editing is inherited.



The lac operon, off and on. The repressor is the lactose switch and CAP is the glucose switch. Both must be in the right place for strong transcription.

LOCK THESE IN

- Operator = cis switch; repressor = trans protein.
- lac: high only with lactose present AND glucose absent (repressor off, cAMP-CAP on).
- Inducible = catabolic, default off. Repressible = anabolic, default on.
- Acetylation opens chromatin; DNA methylation silences.
- Enhancers plus activators plus mediator; combinatorial control sets cell type.
- Bicoid to gap to pair-rule to segment polarity to Hox.
- PCR: denature, anneal, extend; 2^n copies. Sanger: ddNTPs lack a 3′ OH.
- CRISPR: guide RNA plus NGG PAM; NHEJ knocks out, HDR rewrites.

Key terms and comparisons: the lac operon by condition

Glucose	Lactose	Repressor	cAMP-CAP	Transcription
Present	Absent	On operator	Not bound	Off
Absent	Absent	On operator	Bound	Off (repressor wins)
Present	Present	Released	Not bound	Low
Absent	Present	Released	Bound	High

Key terms and comparisons: operons, development and tools

Item	What it does	Key idea
lac operon	Inducible; breaks down lactose	Allolactose is the inducer
trp operon	Repressible; builds tryptophan	Tryptophan is the corepressor; attenuation
Gap genes	Broad bands	Mutant loses a block of segments
Pair-rule genes	Seven stripes	Mutant loses every other segment
Hox genes	Segment identity	Mutant swaps one part for another
Restriction enzyme	Cuts at a palindromic site	Sticky or blunt ends
Reverse transcriptase	RNA to cDNA	No introns
Gel electrophoresis	Sorts DNA by size	Short fragments run farthest
PCR	Amplifies a target	2^n copies after n cycles
Sanger sequencing	Reads bases	ddNTP stops the chain
CRISPR-Cas9	Cuts a chosen site	20 nt guide; NGG PAM

Numbers to know

Item	Value
PCR temperatures	Denature 94 to 98 °C; anneal 50 to 65 °C; extend about 72 °C
PCR primers	About 18 to 25 nucleotides
Copies after 30 cycles	About a billion (2^{30})
Sanger read length	About 700 to 1,000 bases
MicroRNA length	About 22 nucleotides
US forensic STR panel	20 loci
trp leader	About 160 nucleotides; two Trp codons

Worked cases

WORKED CASE 1 · READING THE LAC SWITCHES

Predict lac transcription in four cultures: (a) glucose only; (b) lactose only; (c) both sugars; (d) a $lacI^-$ mutant with glucose only.

STEP 1 · ASK ABOUT LACTOSE FIRST

In (a) there is no lactose, so the repressor sits on the operator: **off**.

STEP 2 · THEN ASK ABOUT GLUCOSE

In (b) lactose removes the repressor and low glucose lets cAMP-CAP bind: **high**.

STEP 3 · BOTH SUGARS

In (c) the repressor is off but glucose keeps cAMP low, so CAP is not bound: **low**.

STEP 4 · THE MUTANT

In (d) no working repressor exists, so the operator is free, but glucose keeps CAP off: **low but constitutive**, with no need for lactose.

WORKED CASE 2 · HOW MANY PCR COPIES?

A single double-stranded target goes through 25 PCR cycles. About how many copies are there, ideally? How many cycles give about a billion?

STEP 1 · USE 2^N

2^{25} = **33,554,432**, about 34 million copies.

STEP 2 · WORK BACKWARD

2^{30} = 1,073,741,824, so **30 cycles** give about a billion.

WHY TAQ

Every cycle heats to about 95 °C, which would destroy an ordinary polymerase.

WORKED CASE 3 · READING A GEL

A 10 kb linear DNA has EcoRI sites at 3 kb and 7 kb. A 5 kb circular plasmid has EcoRI sites at positions 1,000 and 3,800. How many bands does each give, and what sizes?

STEP 1 · LINEAR DNA

Two cuts make three pieces: 3, 4 and 3 kb. The two 3 kb pieces run together, so you see **two bands**: 4 kb and a brighter 3 kb.

STEP 2 · CIRCULAR PLASMID

Two cuts in a circle make two pieces: 3,800 − 1,000 = **2,800 bp** and 5,000 − 2,800 = **2,200 bp**.

STEP 3 · ORDER ON THE GEL

The 2,200 bp piece runs farther than the 2,800 bp piece, because short pieces move faster toward the positive pole.

WORKED CASE 4 · KNOCKOUT OR REWRITE?

A lab cuts a gene with Cas9 in two dishes. Dish 1 gets no repair template. Dish 2 gets a template carrying one corrected base with matching DNA on each side. Predict each.

STEP 1 · FIND THE SITE

The guide matches 20 bases next to an NGG PAM; Cas9 cuts both strands about 3 bp upstream.

STEP 2 · DISH 1

Cells use nonhomologous end joining, which often adds or removes a few bases. The frame shifts: a **knockout**.

STEP 3 · DISH 2

Some cells use homology-directed repair and copy the template: a **precise edit**.

SOMATIC OR GERMLINE

Editing body cells of one patient is somatic and not inherited; editing embryos is germline.

TRAPS PEOPLE FALL INTO

- **Thinking lactose alone gives full lac expression.** Glucose must also be low so cAMP-CAP can bind.
- **Mixing inducer and corepressor.** An inducer switches a repressor off (allolactose); a corepressor switches it on (tryptophan).
- **Expecting a plasmid operator to rescue a lacO^c chromosome.** Operators are cis-acting; they control only their own DNA.
- **Saying methylated promoters are active.** Heavy DNA methylation usually silences a gene.
- **Thinking development removes genes.** Cloning showed differentiated nuclei keep the whole genome.
- **Saying long DNA runs farthest on a gel.** Short fragments run farthest.
- **Adding instead of doubling in PCR.** Each cycle doubles: 2^n.
- **Confusing somatic and germline editing.** Only germline changes pass to children.

Chapter 7 self-test · ten items

1. In the lac operon, what does allolactose do?

A. It is split to make cAMP for CAP
B. It binds CAP and helps it reach DNA
C. It binds the operator and blocks it
D. It binds the repressor and releases it

D. It binds the repressor and releases it Allolactose is the inducer. It changes the repressor's shape so it falls off the operator.

2. A partial diploid has $lacO^c$ on the chromosome and a normal lac region on a plasmid. How is chromosomal lacZ expressed?

A. Only when glucose is present
B. Constitutively, without lactose
C. Only when lactose is present
D. Never, in any condition

B. Constitutively, without lactose The operator is cis-acting. No repressor can bind the mutant operator, so the genes beside it stay on.

3. Tryptophan is plentiful. What happens in the trp leader?

A. Regions 3 and 4 form a terminator
B. The ribosome stalls at the Trp codons
C. Regions 2 and 3 form an antiterminator
D. Polymerase reads into all five genes

A. Regions 3 and 4 form a terminator With plenty of charged Trp tRNA, the ribosome covers region 2, so 3 and 4 pair and stop transcription.

4. A drug blocks histone deacetylases. What is the likely effect?

A. Chromatin packs into heterochromatin
B. Histones are removed from DNA
C. Chromatin opens at many genes
D. DNA methylation rises sharply

C. Chromatin opens at many genes Acetyl groups stay on the lysines, which loosens their grip on DNA.

5. Lens and liver cells both carry the crystallin gene, but only lens cells express it. Why?

A. Liver cells lack RNA polymerase II
B. Lens cells read a different genetic code
C. Only lens cells hold all its activators
D. Liver cells deleted the crystallin gene

C. Only lens cells hold all its activators Combinatorial control: the enhancer needs a specific mix of activators.

6. A fly mutant is missing every other segment. Which gene class is affected?

A. A gap gene
B. A maternal effect gene
C. A Hox gene
D. A pair-rule gene

D. A pair-rule gene Pair-rule genes are expressed in seven stripes, so mutants lose alternate segments.

7. Which cell can form a whole organism, including the placenta?

A. A zygote, which is totipotent
B. A blood stem cell in marrow
C. An iPS cell made from skin
D. An embryonic stem cell (ESC)

A. A zygote, which is totipotent Only totipotent cells make extraembryonic tissue. Embryonic stem and iPS cells are pluripotent.

8. Why are eukaryotic genes cloned into bacteria as cDNA?

A. cDNA is double the gene length
B. Bacteria cannot remove introns
C. cDNA carries its own ribosomes
D. Bacteria cannot copy plasmid DNA

B. Bacteria cannot remove introns cDNA is copied from mature mRNA, so it has no introns.

9. Why does a ddNTP stop a growing DNA chain?

A. It cuts the template strand apart
B. It carries a fluorescent dye group
C. It pairs with the wrong base each time
D. It has no 3′ hydroxyl to join

D. It has no 3′ hydroxyl to join Without a 3′ OH, the next nucleotide cannot form a bond.

10. What must sit next to the target for the common Cas9 to cut?

A. An NGG PAM sequence
B. A TATA box promoter
C. A poly-A signal sequence
D. A GAATTC restriction site

A. An NGG PAM sequence Cas9 scans for the protospacer adjacent motif NGG before checking the guide match.

CHAPTER 8

Evolution, Natural Selection and Population Genetics

Read it in full: **Chapter 8 in the textbook**. See it in motion: **Evolution and Natural Selection Lab** and **Population Genetics Lab**.

What to know

Evolution is descent with modification. In populations it is a change in allele frequencies over generations. Microevolution is change within a population; macroevolution is the origin of new species and larger groups. The pattern (species changed and share ancestors) is settled; scientists research the relative weight of the processes.

Darwin built on others. Linnaeus gave binomial names and nested groups. Cuvier showed fossils change by layer and that extinction happens, though he favored catastrophism. Hutton (gradualism) and Lyell (uniformitarianism) showed Earth is old and slow processes add up. Lamarck proposed use and disuse and inheritance of acquired traits; the mechanism is wrong, but he saw that species change. Malthus showed populations outgrow resources.

On the Beagle voyage (1831 to 1836), Darwin saw that Galápagos species resembled mainland species and varied island to island. Wallace reached the same idea, and they presented together in 1858; the Origin came in 1859. The logic: individuals vary, many traits are inherited, more offspring are born than survive, so individuals with helpful heritable traits leave more offspring and those traits build up. That is **natural selection**, and it produces **adaptations**. Artificial selection (wild mustard to cabbage, kale and broccoli) showed what selection can do.

Remember three points. Individuals are selected, but **populations evolve**. Only heritable traits count. Selection is local and has no goal. A "needed to" explanation is a Lamarckian error; the Darwinian version says resistant variants already existed and the others died.

The evidence agrees across fields. **Direct observation**: soapberry bug beaks shortened on small fruit, finch beaks deepened after the 1977 drought, and MRSA appeared within years of methicillin. **Homology**: the same forelimb bones in humans, cats, whales and bats; shared embryonic tails and pharyngeal arches; one genetic code. **Vestigial structures**: whale pelvic bones and the human coccyx, plus the broken GULO pseudogene shared by humans, apes and monkeys. **Convergent evolution** gives analogous traits, such as insect and bird wings. The **fossil record** has transitional forms (Tiktaalik; Pakicetus to Dorudon in whales). **Biogeography** shows island species related to the nearest mainland.

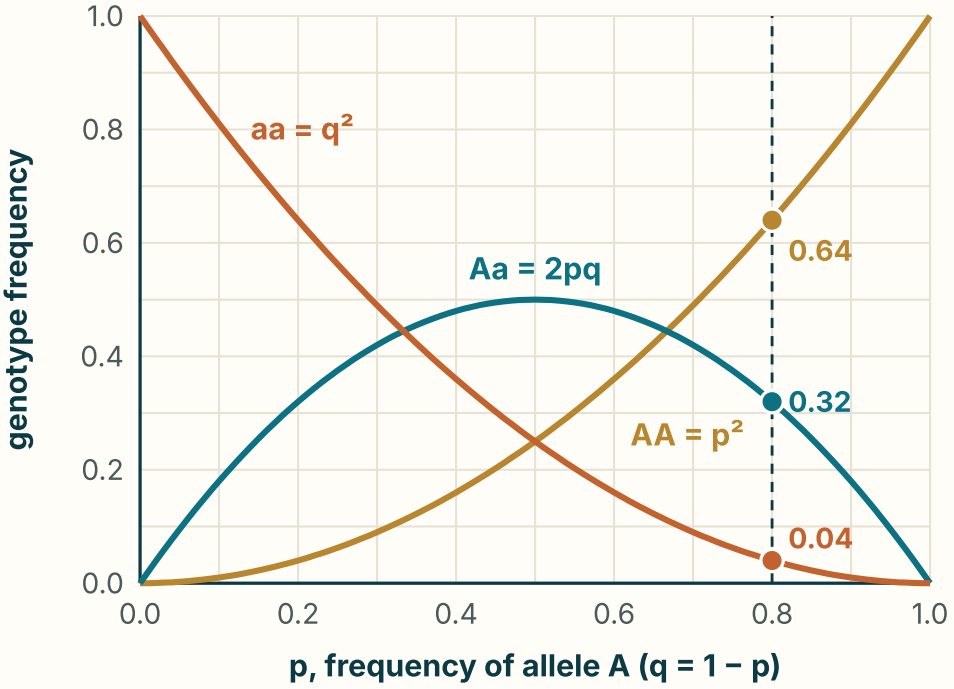
A population's **gene pool** is described by allele frequencies p and q, with p + q = 1. The **Hardy-Weinberg** principle says allele and genotype frequencies stay constant unless something changes them. Genotype frequencies are p^2 (AA), 2pq (Aa) and q^2 (aa), and they add to 1. Five conditions are needed: no mutation, random mating, no selection, a very large population and no gene flow. It is a null model: a departure shows that something is acting.

Start Hardy-Weinberg problems from q^2, the only class you can see. For cystic fibrosis at about 1 in 2,500 births, q = 0.02 and carriers are 2pq, about 1 in 25. For PKU at about 1 in 10,000, q = 0.01 and carriers are about 1 in 50. Carriers far outnumber affected people, which is one reason harmful recessive alleles persist.

Mutation is the source of new alleles but works slowly. Recombination reshuffles alleles every generation. Three forces change allele frequencies most directly. **Natural selection** is the only one that is consistently adaptive. **Genetic drift** is chance change, strongest in small populations; the **founder effect** (Ellis-van Creveld syndrome in an Amish community) and the **bottleneck effect** (elephant seals cut to about 20) are its classic cases. Drift erodes variation and can fix harmful alleles. **Gene flow** moves alleles between populations and makes them more alike; it rescued Illinois prairie chickens, whose egg hatching rose from under half to above 90 percent.

Relative fitness is reproductive contribution compared with others, not strength. **Directional** selection shifts the mean (deeper finch beaks). **Stabilizing** selection favors the middle and narrows variation (human birth weight). **Disruptive** selection favors both extremes (seedcracker finches). **Sexual selection** can be intrasexual (males fighting) or intersexual (mate choice, as with long-calling tree frogs). **Balancing selection** keeps variation: heterozygote advantage (sickle cell carriers resist severe malaria) and frequency-dependent selection (left and right mouthed cichlids).

Not every trait is an adaptation; it could be a byproduct, a leftover or the result of drift. Guppies moved to low-predation streams became more colorful within about 15 generations, as predicted. An **exaptation** evolved for one role and was co-opted for another, as feathers were. Selection cannot make perfect organisms: it works only on existing variation, is bound by history (the recurrent laryngeal nerve loops under the aorta), balances trade-offs and is pushed around by chance and changing environments. **Coevolution** keeps the target moving, which is why combination drug therapy is used for HIV and tuberculosis.



Wildflower example

p = 0.8 red, q = 0.2 white

AA red $0.8^2 = 0.64$

Aa pink $2(0.8)(0.2) = 0.32$

aa white $0.2^2 = 0.04$

Sum: 0.64 + 0.32 + 0.04 = 1

Of 500 plants: 320, 160, 20

Heterozygotes peak at 0.5 when p = q = 0.5. When a recessive allele is rare, most copies hide in carriers.

Hardy-Weinberg, drawn to scale. Pick any allele frequency p, read up, and the three curves give the genotype frequencies. They always add to 1.

LOCK THESE IN

- Individuals are selected; populations evolve. Only heritable traits count.
- Homology = shared ancestry. Analogy = convergent evolution.
- $p + q = 1$; $p^2 + 2pq + q^2 = 1$.
- Start from q^2 (the recessive phenotype), take the square root, subtract for p.
- Five Hardy-Weinberg conditions: no mutation, random mating, no selection, huge population, no gene flow.
- Drift is random and strongest in small populations (founder, bottleneck).
- Directional shifts the mean; stabilizing narrows; disruptive splits.
- Heterozygote advantage keeps the sickle cell allele where malaria is common.

Formulas and numbers

Item	Formula or value	Example
Allele frequencies	$p + q = 1$	$p = 0.8$, $q = 0.2$
Genotype frequencies	$p^2 + 2pq + q^2 = 1$	0.64 + 0.32 + 0.04
Allele count from genotypes	p = (2 × AA + Aa) ÷ (2 × total)	(640 + 160) ÷ 1,000 = 0.8
Carriers from incidence	q = √(incidence); carriers = 2pq	CF: about 1 in 25
Relative fitness	Offspring of a genotype ÷ offspring of the best genotype	2 ÷ 4 = 0.5
Cytochrome c differences	Human and chimp 0; rhesus 1; yeast about 40%	More time, more differences

Key terms and comparisons: forces of evolution

Mechanism	What happens	Adaptive?	Effect on variation
Natural selection	Favored alleles increase	Yes	Directional lowers it; balancing keeps it
Genetic drift	Chance sampling changes	No	Lowers it; can fix harmful alleles
Gene flow	Alleles move between populations	Either	Populations become more alike
Mutation	New alleles appear	No	Source of all new variation; slow
Nonrandom mating	Inbreeding or choosing similar mates	No	More homozygotes; allele frequencies unchanged

Key terms and comparisons: thinkers before Darwin

Thinker	Idea	What Darwin took
Linnaeus	Binomial names, nested groups	Nesting reflects branching descent
Cuvier	Fossils, extinction (catastrophism)	Species are not fixed
Hutton and Lyell	Gradualism, uniformitarianism	Deep time for slow change
Lamarck	Use and disuse; acquired traits	Species change (mechanism rejected)
Malthus	Populations outgrow resources	A struggle in every generation

Worked cases

WORKED CASE 1 · CARRIERS FROM INCIDENCE

About 1 in 10,000 babies is born with PKU, an autosomal recessive condition. Assuming Hardy-Weinberg equilibrium, what share of people are carriers?

STEP 1 · START FROM Q^2

$q^2 = 1/10{,}000 = 0.0001$.

STEP 2 · SQUARE ROOT

$q = 0.01$.

STEP 3 · SUBTRACT

$p = 1 - 0.01 = 0.99$.

STEP 4 · CARRIERS

$2pq = 2 \times 0.99 \times 0.01 = 0.0198$, about **1 in 50**.

COMPARE

Carriers (1 in 50) outnumber affected babies (1 in 10,000) about 200 fold. Most copies of the allele hide in heterozygotes.

WORKED CASE 2 · IS THIS POPULATION IN EQUILIBRIUM?

Population X has 490 AA, 420 Aa and 90 aa. Population Y has 400 AA, 200 Aa and 400 aa. Test each against Hardy-Weinberg.

STEP 1 · X ALLELE FREQUENCY

A copies = 2(490) + 420 = 1,400 of 2,000, so p = 0.7 and q = 0.3.

STEP 2 · X EXPECTED

p^2 = 0.49, 2pq = 0.42, q^2 = 0.09, so 490, 420 and 90. **Matches**: equilibrium.

STEP 3 · Y ALLELE FREQUENCY

A copies = 800 + 200 = 1,000 of 2,000, so p = q = 0.5.

STEP 4 · Y EXPECTED

250 AA, 500 Aa, 250 aa. Observed has only 200 Aa: a big **heterozygote shortage**.

INTERPRET

Too few heterozygotes points to inbreeding, assortative mating or two populations counted as one.

WORKED CASE 3 · THE RECESSIVE TRAP

In a flower population, 96 percent of plants are purple (dominant) and 4 percent are white. Find p, q and the share of purple plants that are heterozygous.

STEP 1 · USE THE WHITE PLANTS

q^2 = 0.04, so q = 0.2 and p = 0.8.

STEP 2 · GENOTYPES

AA = 0.64; Aa = 2(0.8)(0.2) = 0.32.

STEP 3 · SHARE OF PURPLE

0.32 ÷ 0.96 = **1/3** of purple plants are heterozygous.

THE ERROR TO AVOID

Taking the square root of 0.96 is wrong: the purple fraction is p^2 + 2pq, not p^2.

WORKED CASE 4 · NAMING THE MODE OF SELECTION

Describe the pattern: (a) after a drought, mean beak depth rises; (b) babies near average weight survive best; (c) seedcracker finches with small or large bills both do well, while intermediate bills do poorly.

STEP 1 · (A)

The mean moved toward one extreme: **directional**.

STEP 2 · (B)

The middle wins and the spread narrows: **stabilizing**.

STEP 3 · (C)

Both extremes win and the middle drops: **disruptive**, which can be a step toward two species.

TRAPS PEOPLE FALL INTO

- **Saying an individual evolved.** Selection acts on individuals, but only populations evolve.
- **Writing "the bacteria became resistant because they needed to."** Resistant variants already existed; the drug removed the others.
- **Taking the square root of the dominant fraction.** The dominant fraction is $p^2 + 2pq$. Start from q^2.
- **Calling drift adaptive.** Drift is random; it can even fix harmful alleles.
- **Thinking nonrandom mating changes allele frequencies.** On its own it changes genotype frequencies only.
- **Reading fitness as strength.** Fitness is reproductive success relative to others.
- **Calling a bat wing and bird wing simply homologous.** Homologous as forelimbs, analogous as wings. Ask "homologous as what?"
- **Assuming selection makes perfect organisms.** It works on existing variation within historical limits and trade-offs.

Chapter 8 self-test · ten items

1. Which statement reflects Darwin's view rather than Lamarck's?

A. Traits gained during life pass to offspring
B. Organisms change because they need to survive
C. Body parts that are used grow and are inherited
D. Existing heritable variants leave more offspring

D. Existing heritable variants leave more offspring Darwin's mechanism is unequal reproduction among existing heritable variants.

2. Why did Lyell's ideas matter to Darwin?

A. They implied enough time for slow change
B. They explained extinction by catastrophes
C. They proved species were fixed forever
D. They described inheritance of acquired traits

A. They implied enough time for slow change Uniformitarianism implied an old Earth where slow processes can add up to large change.

3. A dolphin flipper and a shark fin both help in swimming. How are they related?

A. Homologous, through a shared fin ancestor
B. Analogous, through convergent evolution
C. Identical, built from the same bones
D. Vestigial, left over from land ancestors

B. Analogous, through convergent evolution The flipper is a modified mammalian forelimb; the shark fin is built differently. The shape reflects convergence.

4. Humans and apes share the same broken GULO gene. What does this best support?

A. Lamarckian use and disuse
B. A shared need for vitamin C
C. Inheritance from a common ancestor
D. Convergent evolution in each species

C. Inheritance from a common ancestor The same disabling mutation is very unlikely to arise separately in each species.

5. In a population, 9 percent show a recessive trait. What share is heterozygous?

A. 30 percent
B. 49 percent
C. 21 percent
D. 42 percent

D. 42 percent $q^2 = 0.09$, so q = 0.3 and p = 0.7. 2pq = 2(0.7)(0.3) = 0.42.

6. Which change would NOT by itself alter allele frequencies?

A. Migration from a distant population
B. A storm killing half the plants at random
C. Inbreeding within the population
D. Selection against one genotype

C. Inbreeding within the population Nonrandom mating raises homozygosity but does not change allele frequencies on its own.

7. A few birds blown to a new island start a population with a rare allele now common. What is this?

A. The founder effect
B. Disruptive selection
C. Heterozygote advantage
D. The bottleneck effect

A. The founder effect A small group starting a new population carries a chance sample of the source gene pool.

8. Hunting once left about 20 northern elephant seals. Why is their variation still low today?

A. Heterozygote advantage at every locus
B. A past bottleneck cut variation
C. Strong gene flow from other seals
D. Very high mutation rates since 1890

B. A past bottleneck cut variation The survivors carried only part of the original variation, and mutation restores it slowly.

9. A tough, long-lived deer leaves no offspring. What is its relative fitness?

A. Undefined without its age
B. Zero, since it left no young
C. High, because it lived long
D. Average for the whole herd

B. Zero, since it left no young Fitness is reproductive contribution, so no offspring means zero.

10. Why do feathers count as an exaptation for flight?

A. They evolved only once flight existed
B. They are vestigial in modern birds
C. They are a byproduct of flying muscles
D. They first served insulation or display

D. They first served insulation or display Feathers appeared in dinosaurs that could not fly and were later co-opted for flight.

Cram sheet

Everything from chapters 7 and 8 in one place. Cover the right column and see if you can rebuild each row.

Numbers and formulas

Item	Value	Note
Hardy-Weinberg	$p + q = 1$; $p^2 + 2pq + q^2 = 1$	Start from q^2
CF carriers	About 1 in 25 (incidence 1 in 2,500)	$q = 0.02$
PKU carriers	About 1 in 50 (incidence 1 in 10,000)	$q = 0.01$
PCR	2^n copies; 30 cycles about a billion	94 to 98, 50 to 65, 72 °C
CRISPR	20 nt guide; NGG PAM; cut about 3 bp upstream	NHEJ or HDR
STR profile	20 US loci	Two alleles per locus
E. coli genes	About 4,300	Human about 20,000
Hox	180 nt homeobox; 60 aa homeodomain	Mammals: 4 clusters, 39 genes
C. elegans	959 somatic cells; 131 die by apoptosis	Ced-9, Ced-4, Ced-3
Elephant seals	About 20 in the 1890s	Now over 100,000, low variation
Guppies	Brighter within about 15 generations	Low predation streams

Key terms and comparisons

Pair	How to tell them apart
Inducible vs repressible	Default off, catabolic; default on, anabolic.
Cis vs trans	DNA site acting on its own molecule; diffusible product.
Determination vs differentiation	Committed fate; visible specialization.
Totipotent vs pluripotent	Whole organism with placenta; every body cell type.
NHEJ vs HDR	Indels and knockout; template-copied edit.
Homology vs analogy	Shared ancestry; convergence.
Founder vs bottleneck	Few colonists; sudden crash.
Intrasexual vs intersexual	Competition within a sex; mate choice.
Adaptation vs exaptation	Favored for current role; co-opted from another.

Rules that win points

Topic	The rule
lac questions	Lactose first, then glucose.
Gels	Short runs far; count pieces from cuts (linear n + 1, circle n).
Hardy-Weinberg	Recessive class first; check that frequencies add to 1.
Selection graphs	Did the mean move, the spread shrink or the middle drop?
Explaining a trait	Check vestige, byproduct and drift before calling it an adaptation.

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