PowerPoint® Lecture Presentations

CHAPTER 11

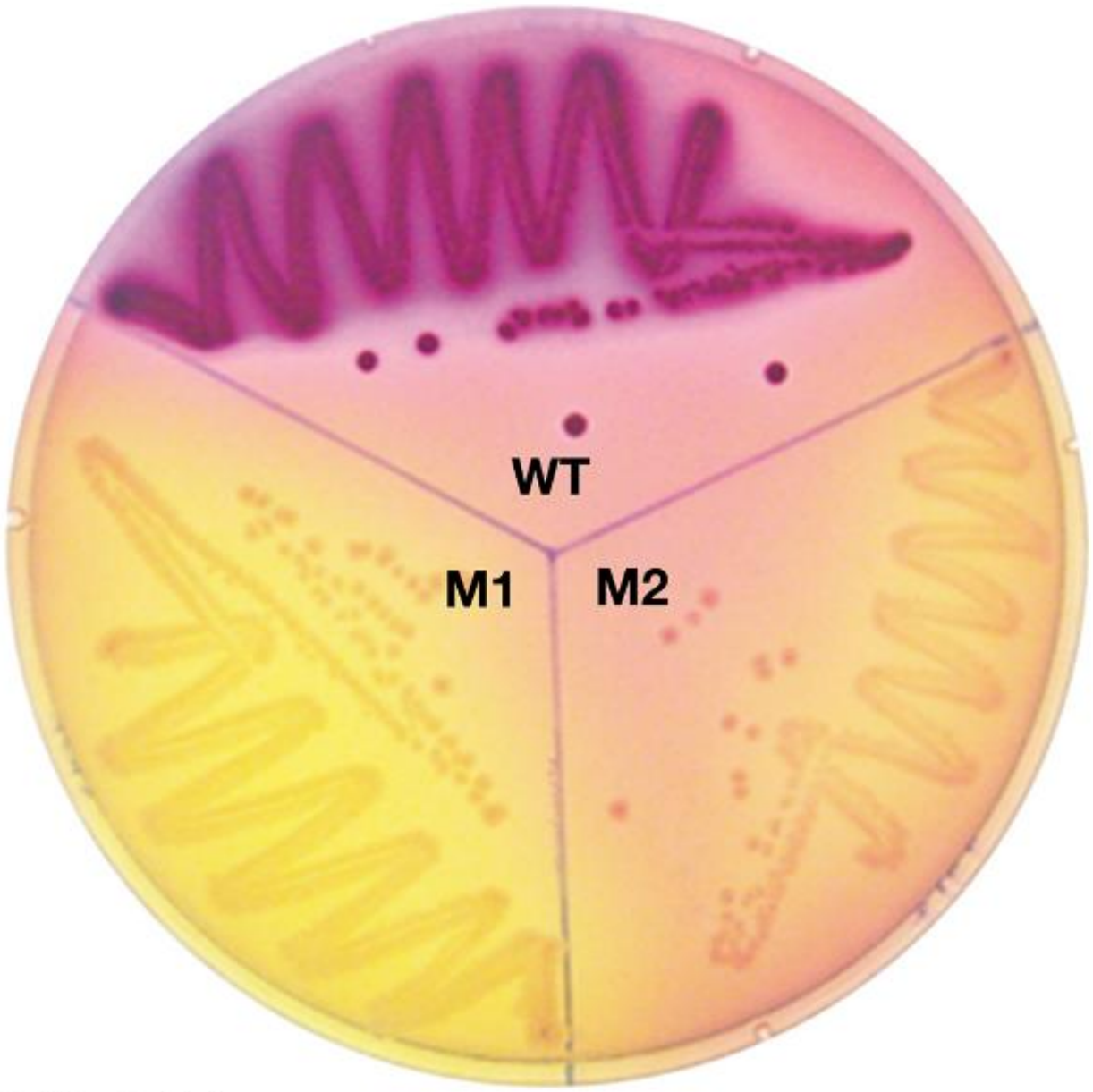
Genetics of *Bacteria* and *Archaea*

I. Mutation

- 11.1 Mutations and Mutants
- 11.2 Molecular Basis of Mutation
- 11.3 Reversions and Mutation Rates
- 11.4 Mutagenesis

11.1 Mutations and Mutants

- Mutation
  - heritable change in DNA sequence that can lead to a change in phenotype (observable properties of an organism)
- Wild-type strain
  - typically refers to strain isolated from nature
  - “Wild-type” can also refer to just one gene.
- Mutant
  - a cell or virus derived from wild type that carries a nucleotide sequence (*genotype*) change
  - Observable properties (*phenotype*) may also be altered (Figure 11.1).
  - can be obtained from *parental strain* derived from wild-type



WT	Wild type
M1	Mutant 1
M2	Mutant 2

Howard Shuman and Thomas Silhavy

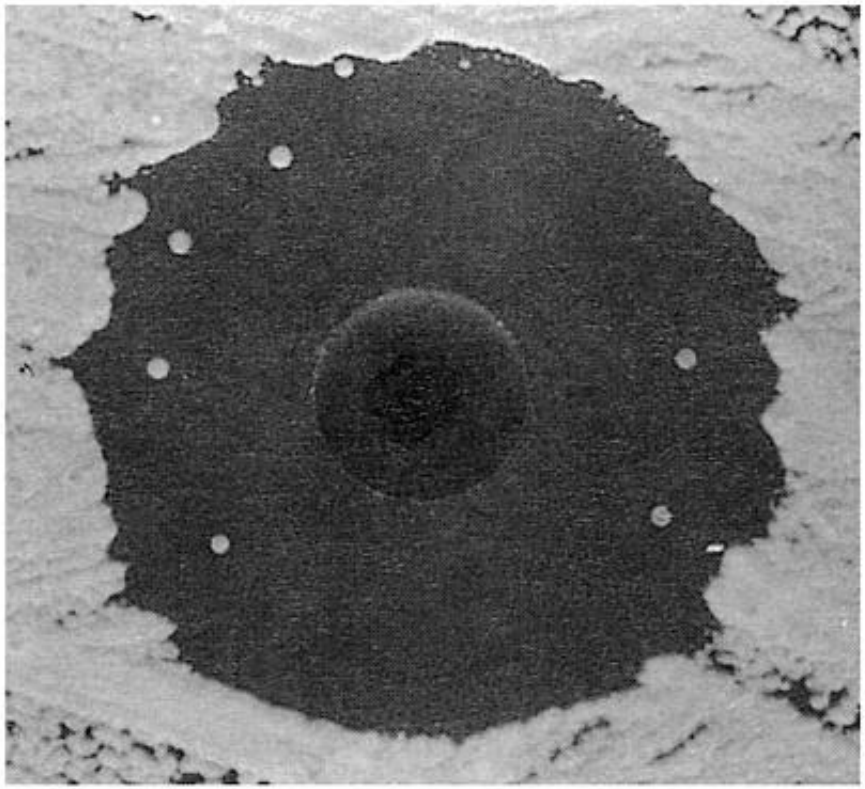
Figure 11.1

11.1 Mutations and Mutants

- Mutant strain may/may not differ in phenotype.
  - *Genotype* is designated by italicized (three lowercase letters + capital letter) for a gene (*e.g., his*C).
  - Mutations are designated with numbers referring to order of isolation (*e.g.*, *hisC1*, *hisC2*).
  - *Phenotype* is designated by capital letter + two lowercase letters and +/– to indicate presence/absence (*e.g.,* His^+ or His^-).

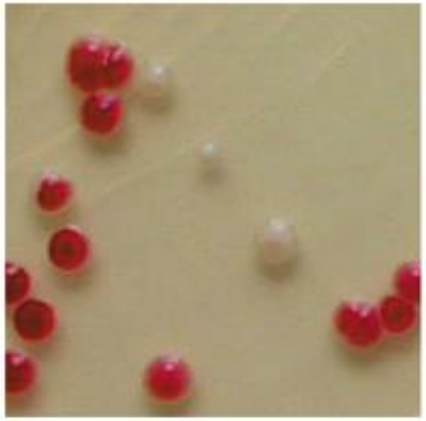
11.1 Mutations and Mutants

- Isolation of mutants: screening versus selection
  - *Selectable mutations* confer an advantage.
    - Under certain environmental conditions, progeny outgrow and replace parent.
    - example: antibiotic resistance (Figure 11.2*a*)
    - powerful genetic tool
  - Nonselectable mutations do not confer an advantage even though they may lead to a phenotypic change.
    - example: color loss in a pigmented organism (Figure 11.2*b*, *c*)
    - requires laborious, time-consuming screening (examining large numbers and looking for differences)



T. D. Brock

(a)



Steven R. Spilatro

(b)

Shiladitya DasSarma, Priya Arora, Lone Simonsen

(c)

Figure 11.2

11.1 Mutations and Mutants

- Isolation of nutritional auxotrophs
  - *Replica plating* screens for nutritionally defective mutants. (Figure 11.3)
    - transfer colonies from master plate
    - Inability of colony to grow medium lacking a nutrient indicates mutation.
    - Colony on master plate is picked, purified, characterized.
  - Auxotroph has an additional nutritional requirement for growth (*e.g.,* His^-).
  - Parental strain is *prototroph* (*e.g.,* His^+).

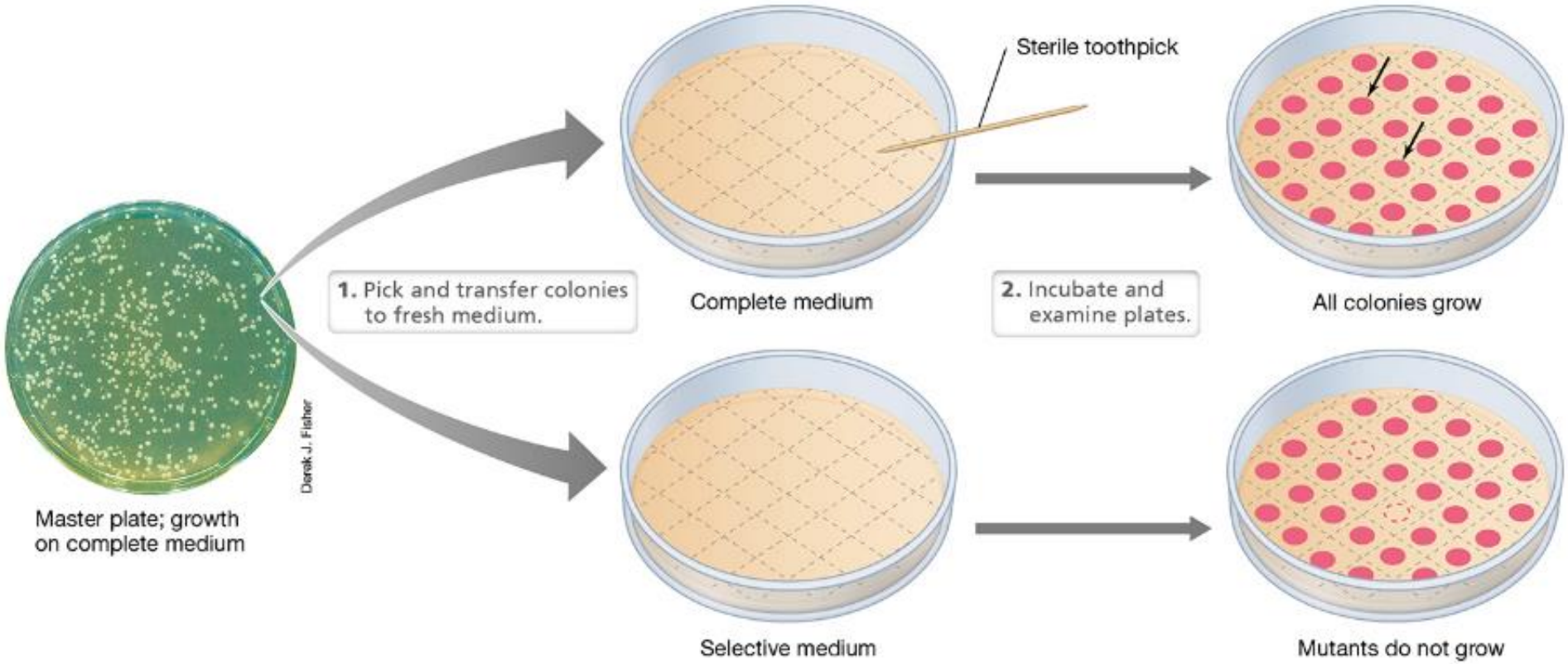


Figure 11.3

11.2 Molecular Basis of Mutation

- Spontaneous mutations
  - those that occur without external intervention
  - Most result from occasional errors by DNA polymerase during replication.
- Induced mutations
  - those made environmentally and deliberately
  - can result from exposure to natural radiation or chemicals that chemically modify DNA
- Point mutations
  - mutations that change only one base pair
  - can lead to single amino acid change in a protein, an incomplete protein, or no change at all

11.2 Molecular Basis of Mutation

- Base-pair substitutions: Missense, nonsense, and silent mutations (Figure 11.4)
  - Not all mutations change polypeptides due to degeneracy.
  - Silent mutations do not affect sequence of encoded polypeptide or phenotype.
    - almost always third base of codon
  - Missense mutation changes sequence of amino acids in polypeptide (*e.g.,* UAC to AAC).
    - if at a critical location, especially active site, could alter activity
    - Not all missense mutations lead to dysfunction.
  - Nonsense mutation leads to stop codon.
    - typically results in *truncated* (incomplete) protein that lacks normal activity

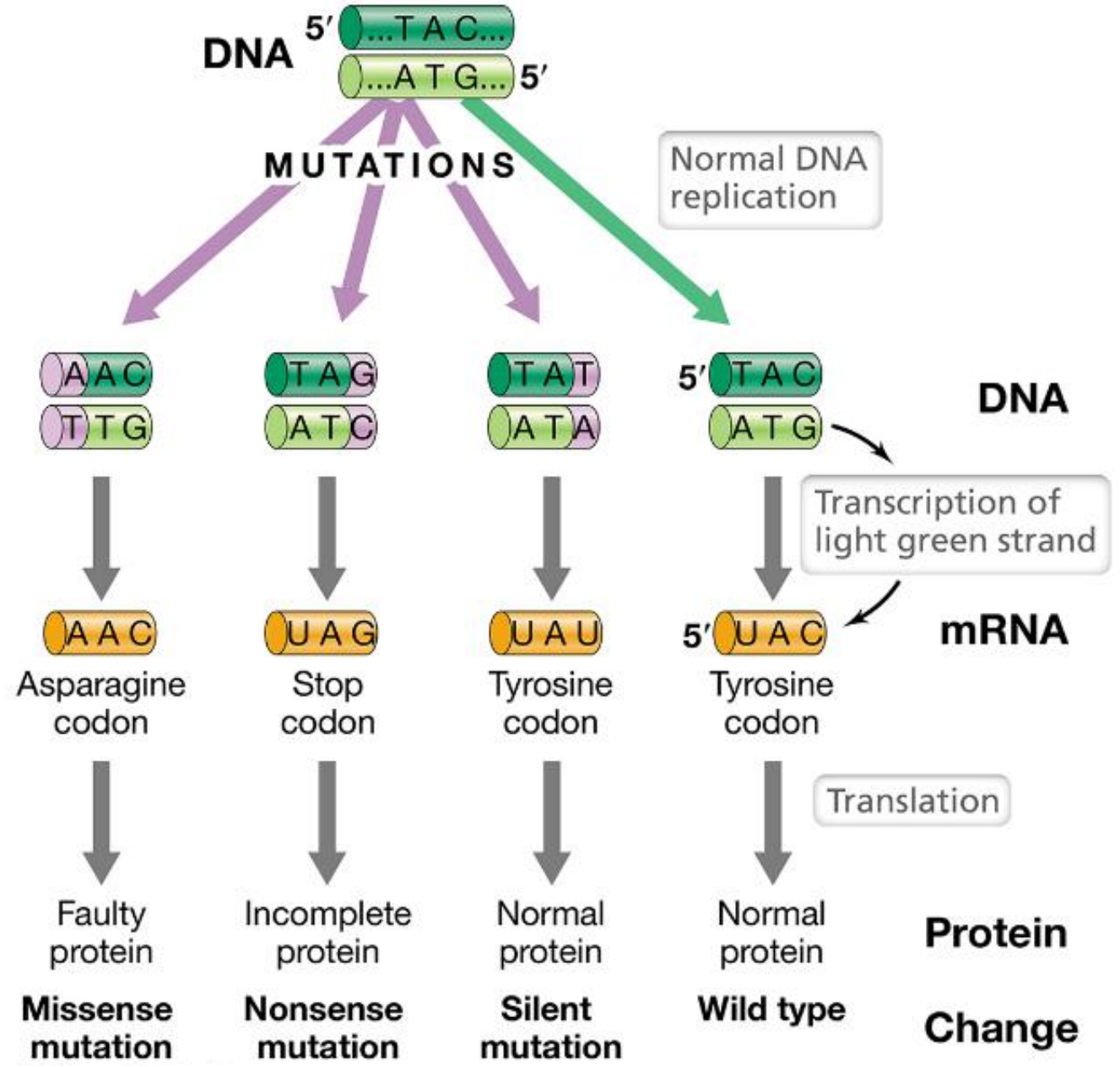


Figure 11.4

11.2 Molecular Basis of Mutation

- Base-pair substitutions: Missense, nonsense, and silent mutations (Figure 11.4)
  - Transitions are purines (A/G) substituted for other purines.
  - Transversions are purines substituted for pyrimidines (C/T) or vice versa.

11.2 Molecular Basis of Mutation

- Frameshifts and other insertions or deletions
  - frameshift mutations (Figure 11.5)
    - deletions or insertions that result in a shift in the reading frame
    - Insertion/deletion of three base pairs adds/deletes an amino acid, which usually is not as bad.
    - scrambles entire polypeptide sequence downstream
  - Insertions/deletions can result in gain/loss of hundreds to thousands of base pairs.
    - often result in complete loss of gene function
    - may arise from errors during genetic recombination
    - Large insertions may be due to *transposable elements.*

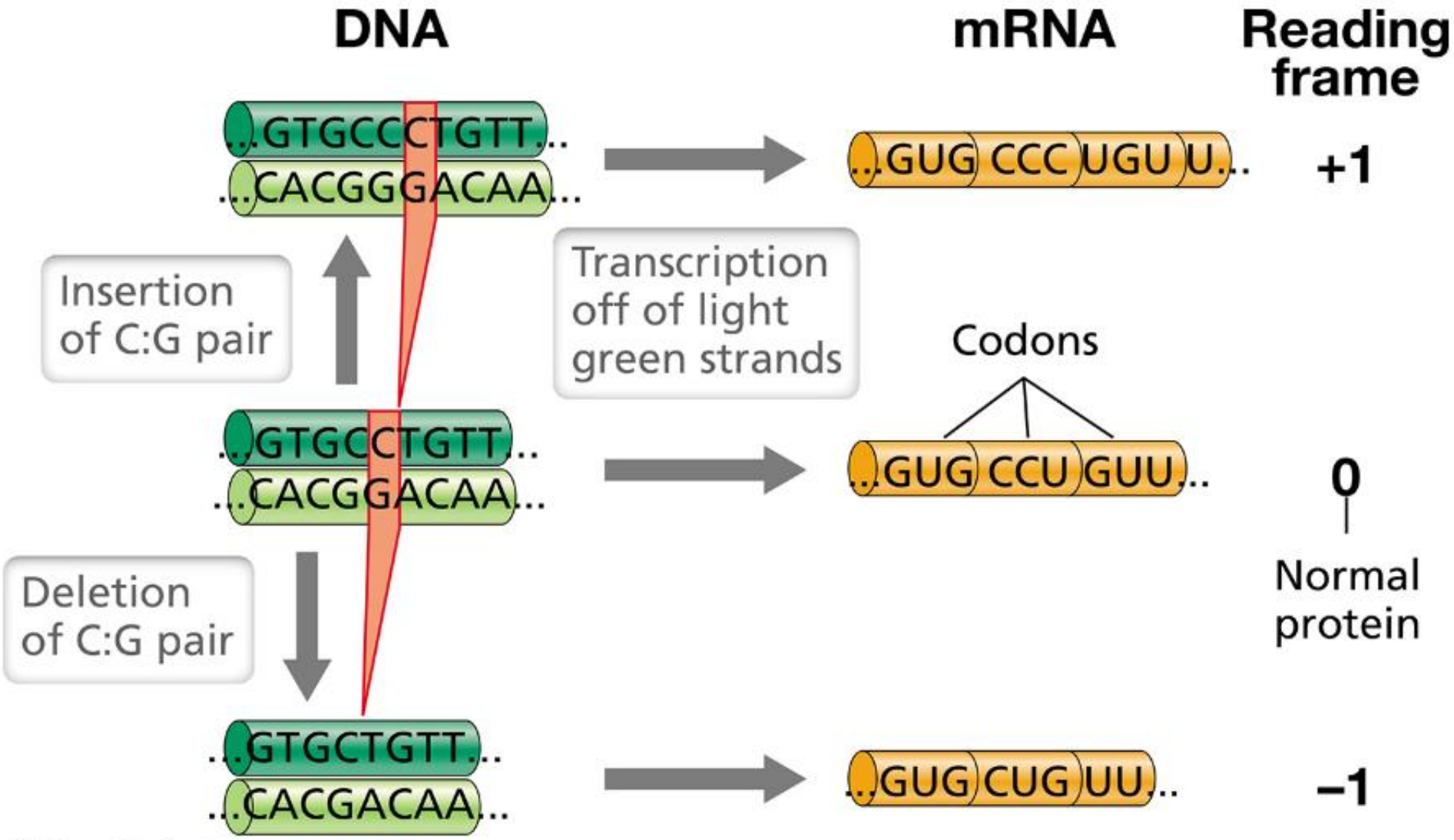


Figure 11.5

11.4 Mutagenesis

- Mutagens: chemical, physical, or biological agents that increase mutation rates
- Chemical mutagens and radiation (Table 11.2)
  - *nucleotide base analogs:* resemble nucleotides but have faulty base-pairing (Figure 11.7)
    - Replication errors occur at higher frequencies due to incorrect base pairing.
  - chemical mutagens that induce chemical modifications
    - Example: Alkylating agents such as nitrosoguanidine introduce changes in replicating or nonreplicating DNA.
  - chemical mutagens that cause frameshift mutations
    - Example: Intercalating agents such as acridines push two base pairs apart, triggering insertions or deletions.

Mutagens

11.4 Mutagenesis

- Mutagens: chemical, physical, or biological agents that increase mutation rates
- Chemical mutagens and radiation (Figure 11.8)
  - *nonionizing radiation* (*e.g.,* Ultraviolet [UV])
    - Purines and pyrimidines strongly absorb UV.
    - *Pyrimidine dimer* (two adjacent Cs or Ts on the same strand become covalently bonded) is primary effect of UV radiation.
    - Killing cells by UV is primarily due to effect on DNA.
  - *ionizing radiation* (*e.g.,* X-rays, cosmic rays, and gamma rays)
    - more powerful than UV
    - ionize water, forming free radicals such as hydroxyl radical (OH·) that damage macromolecules, leading to double- or single-stranded breaks and rearrangements or large deletions

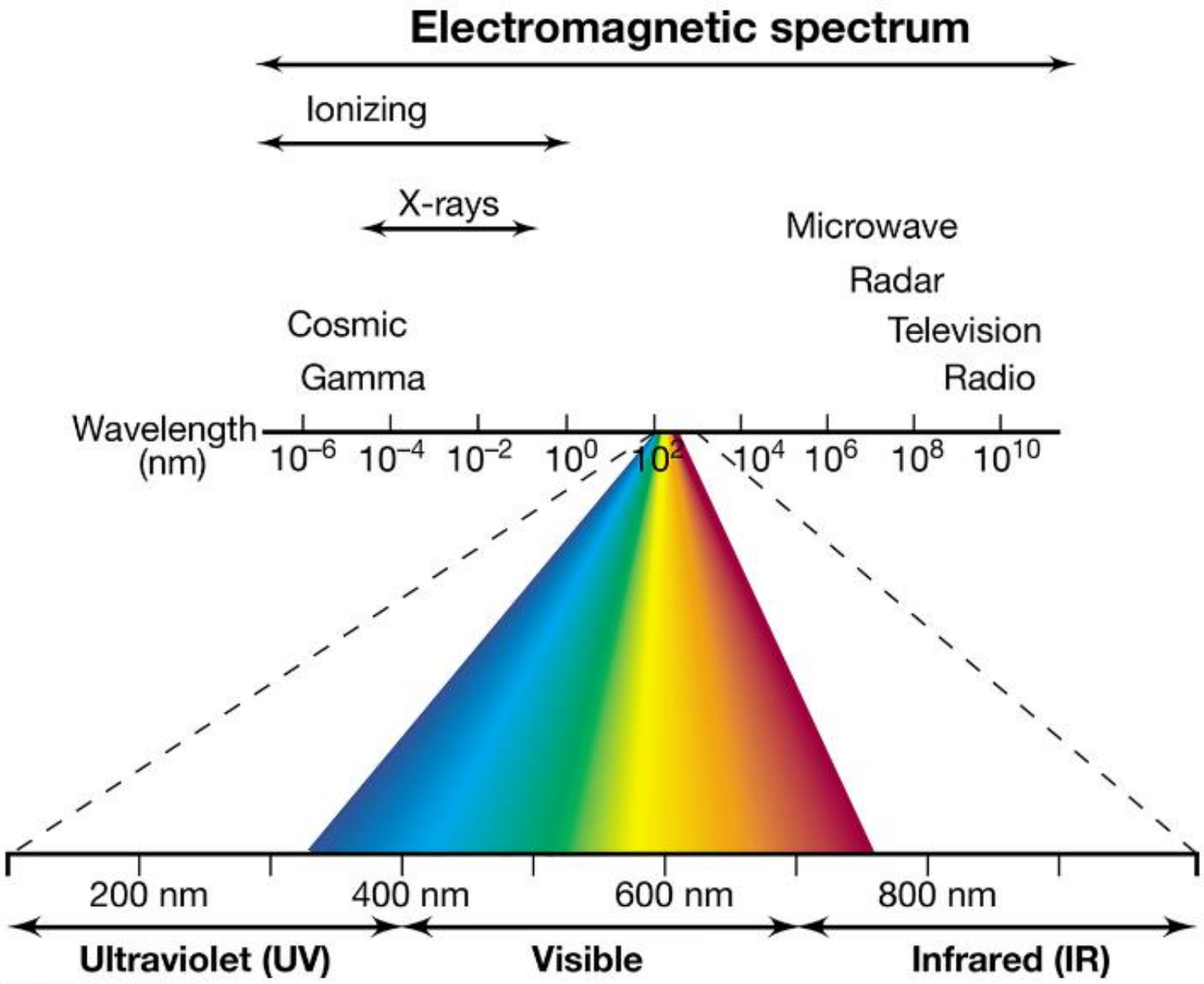


Figure 11.8

11.4 Mutagenesis

- DNA repair and the SOS system
  - In *Bacteria*, stalled replication or major DNA damage activates the SOS repair system. (Figure 10.9)
    - initiates many DNA repair processes, some of which are error-free
    - also allows DNA repair to occur without a template and with random incorporation of dNTPs (*translesion synthesis*), resulting in many errors and mutations
    - In *Escherichia coli,* SOS controls transcription of ~40 genes (regulon). (Figure 11.9)
    - Regulators are LexA (repressor) and RecA (normally for recombination) proteins.

II. Gene Transfer in *Bacteria*

- 11.5 Genetic Recombination
- 11.6 Transformation
- 11.7 Transduction
- 11.8 Conjugation
- 11.9 The Formation of Hfr Strains and Chromosome Mobilization