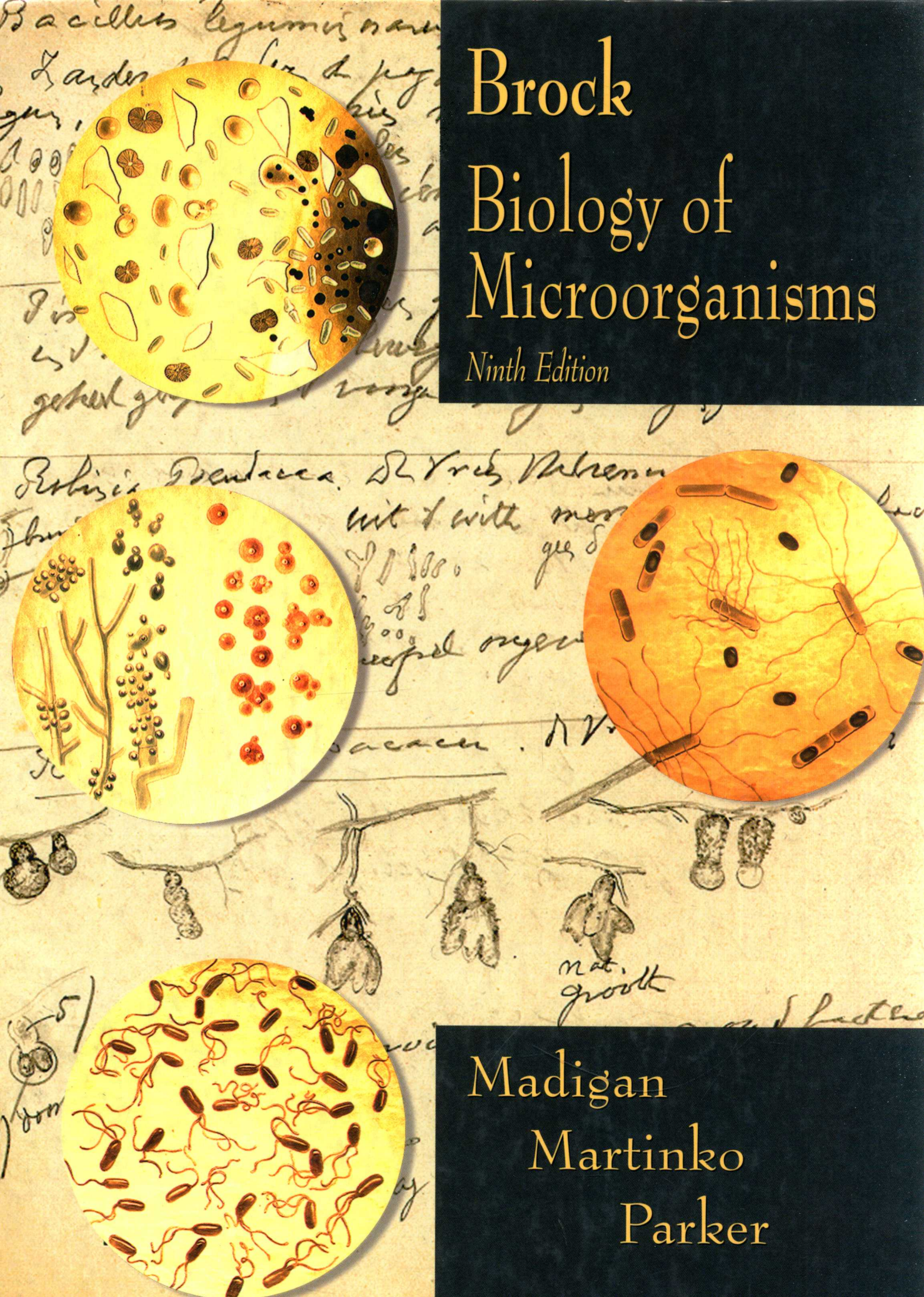
Brock Biology of Microorganisms

Ninth Edition



Madigan
Martinko
Parker

Ninth Edition

BROCK
Biology of Microorganisms

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Jack Parker

Southern Illinois University Carbondale

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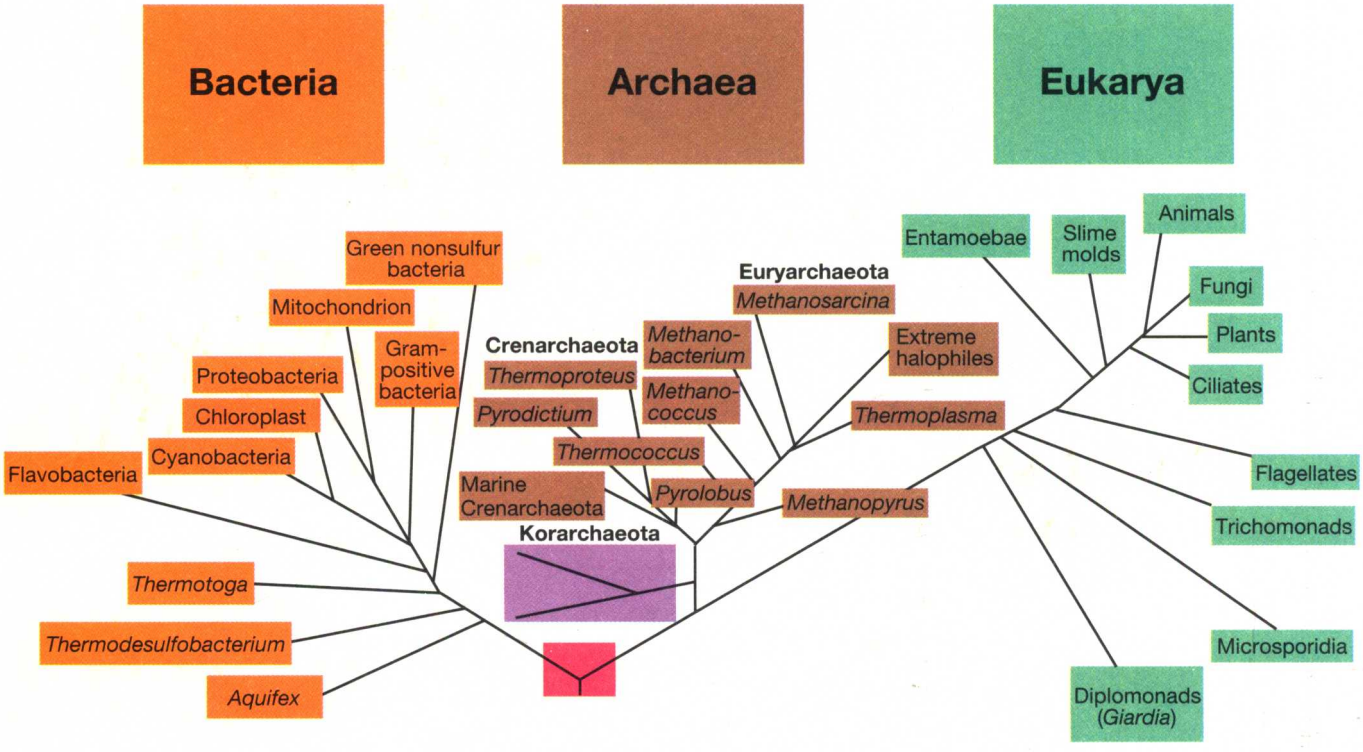
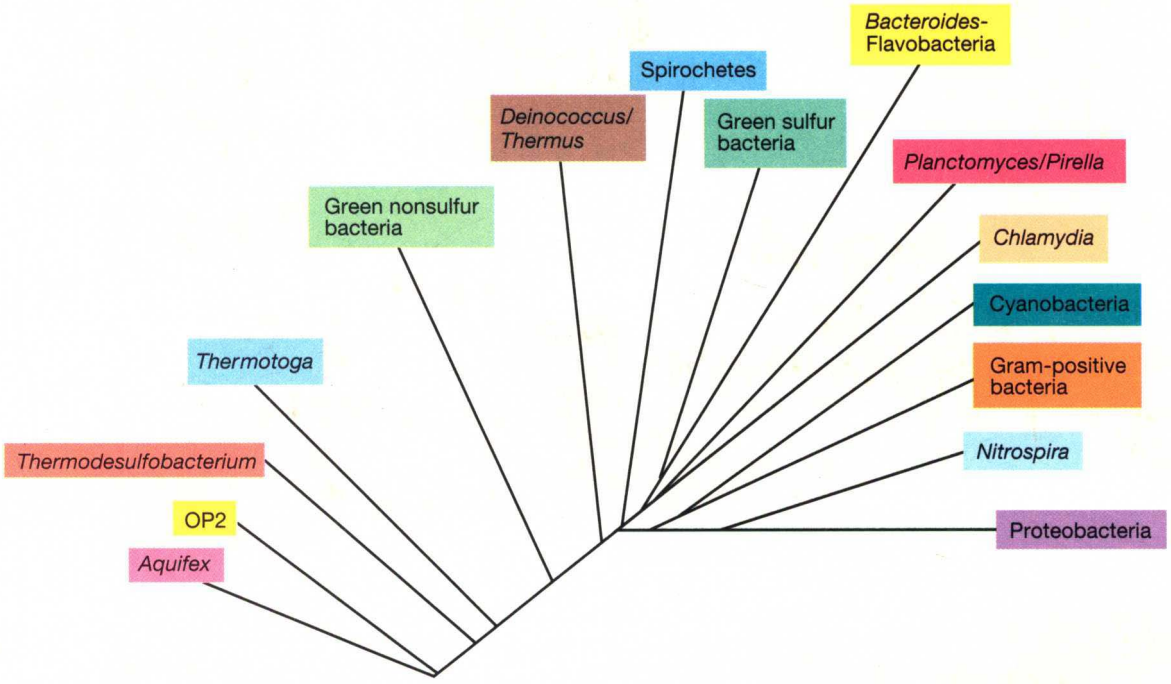
Phylogeny of the Living World–Overview



UNIVERSAL PHYLOGENETIC TREE. This tree is derived from comparative sequencing of 16S or 18S RNA. Note the three major domains of living organisms: the Bacteria, the Archaea, and the Eukarya. The evolutionary distance between two groups of organisms is proportional to the cumulative distance between the end of the branch and the node that joins the two groups. See Sections 12.4–12.8 for further information on ribosomal RNA-based phylogenies. *Data for the tree obtained from the Ribosomal Database Project* **http://www.cme.msu.edu/RDP/**

Phylogeny of the Living World–Bacteria



PHYLOGENETIC TREE OF BACTERIA. This tree is derived from 16S ribosomal RNA sequences. Fifteen major groups of Bacteria can be defined as indicated. See Sections 12.4–12.8 for further information on ribosomal RNA-based phylogenies. *Data for the tree obtained from the Ribosomal Database Project* http://www.cme.msu.edu/RDP/

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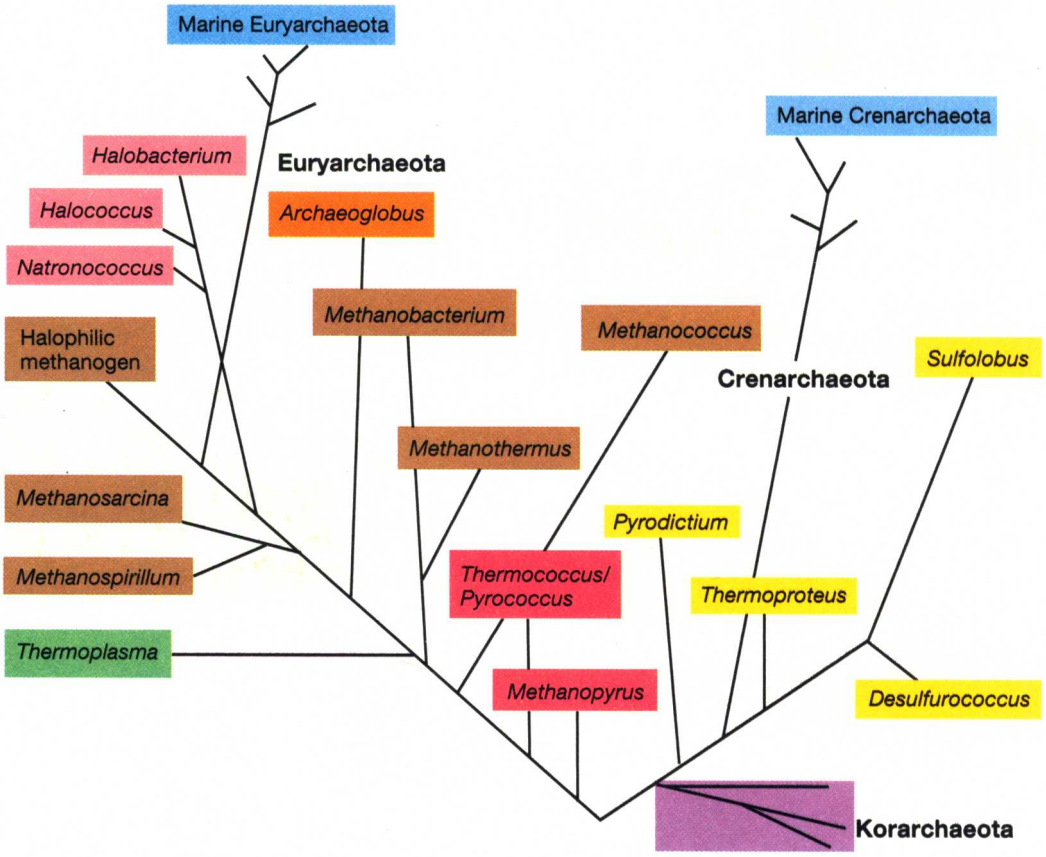
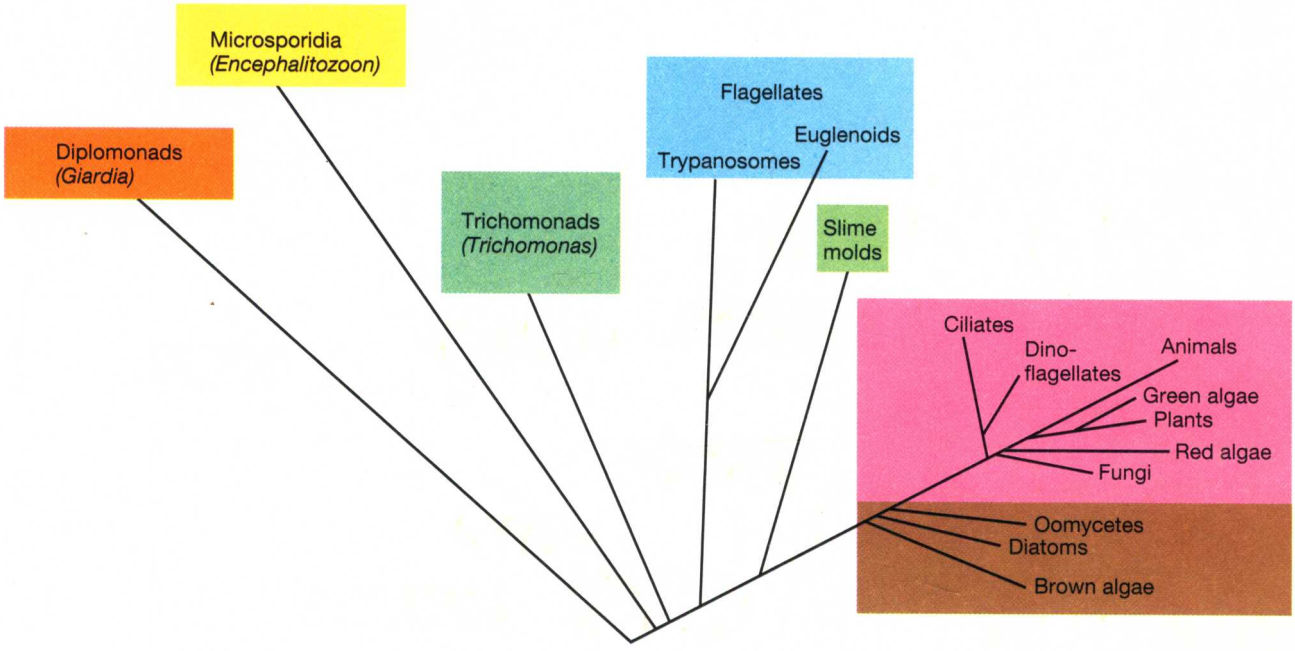
Phylogeny of the Living World–Archaea



PHYLOGENETIC TREE OF ARCHAEA. This tree is derived from 16S ribosomal RNA sequences. Three major kingdoms of Archaea can be defined: the Crenarchaeota, which consists of both hyperthermophiles and cold-dwelling species; the Euryarchaeota, which contains methanogenic and extremely halophilic prokaryotes; and the Korarchaeota, which are, as far as is known, hyperthermophiles. See Sections 12.4–12.8 for further information on ribosomal RNA-based phylogenies. *Data for the tree obtained from the Ribosomal Database Project* http://www.cme.msu.edu/RDP/

Phylogeny of the Living World–Eukarya



PHYLOGENETIC TREE OF EUKARYA, THE EUKARYOTES. This tree is based on 18S rRNA sequences obtained from the small subunit of cytoplasmic ribosomes. Note the close phylogenetic relationship between animals and plants but the distant relationship between these groups and organisms such as *Giardia*. See Sections 12.4–12.8 for further information on ribosomal RNA-based phylogenies.
Data for the tree obtained from the Ribosomal Database Project
http://www.cme.msu.edu/RDP/

BROCK

Biology of Microorganisms

MICHAEL T. MADIGAN dedicates this book to all of his microbiology professors who revealed to him, each in their own way, the fascinating world of microorganisms. These include in particular Joseph Harris and Robert Simpson at UW-Stevens Point, Thomas Brock and Jerry Ensign at UW-Madison, and Howard Gest at Indiana University. A very special acknowledgement goes to Tom Brock, who besides guiding me through my graduate studies and introducing me to the world of bacterial diversity, taught me the value of hard work and instilled in me a love of writing. And I will never forget Tom's willingness nearly 20 years ago to give a young, untested assistant professor a chance to contribute to a book that has turned out to be the experience of a lifetime.

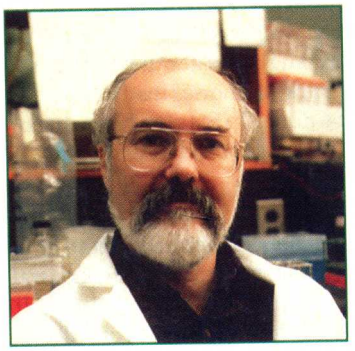
JOHN M. MARTINKO dedicates this book to the inspiring teachers and mentors he had throughout his education. To Peter Baker, Jerry Senturia, and Richard Dickerson at Cleveland State University, you shared your enthusiasm as well as your knowledge. To Willard Schmidt at Case Western Reserve University, you taught me about practical science. To Richard Bankert and George Mayers at Roswell Park Memorial Institute and SUNY at Buffalo, you showed me how research should be done. To Stanley Nathenson at Albert Einstein College of Medicine, you showed me the single-minded dedication and focus necessary to be successful.

JACK PARKER dedicates this edition to the wonderful teachers he has had. There have been too many to list them all, but in particular there was Wesley Stieg at North Central College, Ed Umbarger at Purdue University, Frederck C. Neidhardt at Purdue and at the University of Michigan, and James D. Friesen at York University. Thank you all for the time you spent, and for your love of learning.

About the Authors

Michael T. Madigan (in photo with Willie and Plum) received a bachelor's degree in biology and education from Wisconsin State University at Stevens Point in 1971 and M.S. and Ph.D. degrees in 1974 and 1976, respectively, from the University of Wisconsin, Madison, Department of Bacteriology. His graduate work involved study of the biology of hot spring photosynthetic bacteria under the direction of Thomas D. Brock. Following three years of postdoctoral training in the Department of Microbiology, Indiana University, where he worked on photosynthetic bacteria with Howard Gest, he moved to Southern Illinois University at Carbondale, where he is now Professor of Microbiology. He has been a coauthor of *Biology of Microorganisms* since the fourth edition (1984) and teaches courses in introductory microbiology and bacterial diversity. In 1988 he was selected as the outstanding teacher in the College of Science, and in 1993 its outstanding researcher. His research has dealt almost exclusively with anoxygenic phototrophic bacteria, especially those species that inhabit extreme environments. He has published nearly 85 research papers, has coedited a major treatise on photosynthetic bacteria, and is Chief Editor for North America of the journal *Archives of Microbiology*. His nonscientific interests include reading, hiking, tree planting, and caring for his dogs and horses. He lives aside a quiet lake about five miles from the SIU campus with his wife, Nancy, two dogs, Willie and Plum, and King and Feenkönig (horses).

John M. Martinko attended The Cleveland State University and majored in biology with a chemistry minor. As an undergraduate student he participated in a cooperative education program, gaining research experience in several microbiology and immunology laboratories. He then worked for two years at Case Western Reserve University as a laboratory manager, continuing his cooperative education research on the structure, serology, and epidemiology of *Streptococcus pyogenes*. He next went to the State University of New York at Buffalo where he did research on antibody specificity and idiotypes for his M.A. and Ph.D. (1978) in Microbiology. As a postdoctoral fellow, he worked at Albert Einstein College of Medicine in New York on the structure of major histocompatibility complex proteins. Since 1981, he has been in the Department of Microbiology at Southern Illinois University at Carbondale where he is currently the Chair and Associate Professor. His research interests include the effects of growth hormone on the immune response and the immunological identification of soybean brown stem rot disease. His teaching interests include undergraduate and graduate courses in immunology and a team-taught general microbiology course, where he is responsible for immunology, host defense, and infectious diseases. He lives with his wife Judy, a junior high school science teacher, and their daughters, Martha and Helen, in Carbondale where he has been active in coaching his daughters' soccer and softball teams. He tries to find time to play soccer and golf.



Jack Parker received his bachelor's degree in biology and also received his doctoral degree in a biology program (Ph.D., Purdue University, 1973). However, his research project dealt with bacterial physiology and he completed his Ph.D. research while in the microbiology department at the University of Michigan. Following this he spent four years studying bacterial genetics at York University in Toronto, Ontario. He has taught courses in bacterial genetics, general genetics, human genetics, molecular biology, and molecular genetics, and has participated in courses in introductory microbiology, medical microbiology, and virology primarily at Southern Illinois University at Carbondale, where he is now a Professor in the Department of Microbiology and Dean of the College of Science. His research has been in the broad area of molecular genetics and gene expression and has been focused most specifically on studies of how cells control the accuracy of protein synthesis. He is the author of approximately 50 research papers. His home is on the edge of the Shawnee National Forest in deep southern Illinois where he lives with his wife, Beth, and three children, Justine, D'Arcy, and Grant.

Overview

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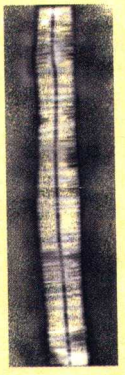
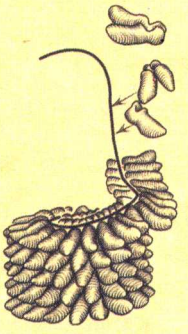
Tobacco Mosaic Virus (TMV), whose structure is shown here, was the first virus discovered. Work began on it in 1886 and the Russian microbiologist Ivanowsky showed that it passed through filters that could retain all known bacteria. The Dutch microbiologist Martinus Beijerinck proposed in 1899 that the tobacco mosaic agent was a new type of microorganism that we now call a virus. TMV was also the first virus purified and crystallized (by Wendell Stanley in 1935), and later the first large biological structure whose subunits could be shown to spontaneously reassemble. Interestingly, TMV is also a virus whose genetic material is RNA and gave early evidence of the incredible variety of strategies that viruses use to replicate themselves.





CHAPTER 8 Viruses

The CHAPTER OUTLINE provides an overview of the chapter's main concepts.

SECTION NUMBERS keyed to page numbers provide easy reference points

The WORKING GLOSSARY provides definitions of important terms within each chapter.

WORKING GLOSSARY

Anticodon a sequence of three bases in a tRNA molecule that base-pairs with a codon during protein synthesis
Aminoacyl-tRNA synthetases a group of enzymes each one of which catalyzes the attachment of the correct amino acid to a tRNA
Antiparallel in reference to double-stranded DNA, one strand runs 5′ → 3′ and other 3′ → 5′
Chromosome a genetic element, usually circular in prokaryotes and linear in eukaryotes, carrying genes essential to cellular function
Codon a sequence of three bases in mRNA that encodes an amino acid
DNA polymerase an enzyme that synthesizes a new strand of DNA in the 5′ → 3′ direction using an antiparallel DNA strand as a template
Exon the coding DNA sequences in a split gene (contrast with intron)
Gene a segment of DNA specifying a protein (via mRNA), a tRNA, or an rRNA
Genome the total complement of genes contained in a cell or virus
Hybridization formation of a duplex nucleic acid molecule with strands derived from different sources by complementary base pairing
Intron the intervening noncoding DNA sequences in a split gene (contrast with exon)
Messenger RNA (mRNA) an RNA molecule that contains the genetic information necessary to encode a particular protein
Molecular chaperones a group of proteins that help other proteins fold or refold from a partially denatured state
Operon a cluster of genes whose expression is controlled by a single operator
Primary transcript an unprocessed RNA molecule that is the direct product of transcription
Primer a molecule (usually a polynucleotide) to which DNA polymerase can attach the first deoxyribonucleotide during DNA replication
Promoter a site on DNA to which RNA polymerase can bind and begin transcription
Replication synthesis of DNA using DNA as a template
Restriction enzyme an enzyme that recognizes and makes double-stranded breaks at specific DNA sequences
Ribosomal RNA (rRNA) types of RNA found in the ribosome; some participate actively in the process of protein synthesis
Ribosome a cytoplasmic particle composed of ribosomal RNA and protein, which is a central part of the protein-synthesizing machinery of the cell
Ribozyme an RNA molecule that can catalyze chemical reactions
RNA polymerase an enzyme that synthesizes RNA in the 5′ → 3′ direction using an antiparallel DNA strand as a template
RNA processing the conversion of a precursor RNA to its mature form
Semiconservative replication DNA synthesis yielding new double helices, each consisting of one parental and one progeny strand
Transcription the synthesis of RNA using a DNA template
Transfer RNA (tRNA) an adaptor molecule used in translation that has specificity for both a particular amino acid and for one or more codons
Translation the synthesis of protein using the genetic information in messenger RNA as a template

We now begin a study of the flow of information in microorganisms that will extend over the next five chapters. As we noted in Chapter 1, two hallmarks of life are *energy transformation* and *information flow*. In Chapter 4 we dealt with the problem of energy transformation: *metabolism*. Now we deal with the problem of information flow: *genetics*. **Genetics** is the discipline that deals with the mechanisms by which traits are passed from one organism to another and how they are expressed.

Since biological information flow is the basis o cellular function, genetics is a major research tool i attempts to understand the molecular mechanisms b which cells function. The study of genetics at the mo ecular level is also central to an understanding of th variability of organisms and the evolution of specie

Understanding how information is transmitte through biological systems also has tremendous pra tical applications. For instance, genetics provides u with techniques to specifically modify organisms fo

TABLES have been redesigned to make key information even more accessible to students.

CONCEPT CHECKS summarize each section and provide quiz questions, so students can evaluate their understanding as they progress through the chapter.

CONCEPT LINKS alert students to material that builds on previous concepts and provides a useful cross-referencing system for the entire book.

Outstanding MICROGRAPHS are included throughout.

TABLE 9.1 Kinds of mutants

Description	Nature of change	Detection of mutant
Auxotroph	Loss of enzyme in biosynthetic pathway	Inability to grow on medium lacking the nutrient
Cold-sensitive	Alteration of an essential protein so it is inactivated at low temperature	Inability to grow at a low temperature (for example, 20°C) that normally supports growth
Drug-resistant	Alteration of permeability to drug or drug target or detoxification of drug	Growth on medium containing a growth-inhibitory concentration of the drug
Noncapsulated	Loss or modification of surface capsule	Small, rough colonies instead of larger, smooth colonies
Nonmotile	Loss of flagella; nonfunctional flagella	Compact colonies instead of flat, spreading colonies
Pigmentless	Loss of enzyme in biosynthetic pathway leading to loss of one or more pigments	Presence of different color or lack of color
Rough colony	Loss or change in lipopolysaccharide outer layer	Granular, irregular colonies instead of smooth, glistening colonies
Sugar fermentation	Loss of enzyme in degradative pathway	Lack of color change on agar containing sugar and a pH indicator
Temperature-sensitive	Alteration of an essential protein so it is more heat-sensitive	Inability to grow at a temperature normally supporting growth (for example, 40°C) but still growing at a lower temperature (for example, 30°C)
Virus-resistant	Loss of virus receptor	Growth in presence of large amounts of virus

Some of the most common kinds of mutants and the means by which they are detected are listed in Table 9.1.

✓ 9.1 Concept Check

Mutation, a heritable change in DNA, can lead to a change in phenotype. Selectable mutations are those that give the mutant a growth advantage under certain environmental conditions and are especially useful in genetics research.

✓ Distinguish between *mutation* and *mutant*.
✓ Distinguish between *screening* and *selection*.

9.2 Molecular Basis of Mutation

As previously mentioned, mutations arise in cells because of changes in the *base sequence* of an organism's genetic material. In many cases, mutations lead to phenotypic changes in the organism; these changes are mostly harmful or neutral although beneficial changes do occur occasionally.

Mutation can be either spontaneous or induced. **Spontaneous mutations** can occur as a result of the action of natural radiation (cosmic rays, and so on) which alters the structure of bases in the DNA. Spontaneous mutations can also occur during replication, as a result of errors in the pairing of bases, leading to changes in the replicated DNA. In fact, such errors occur at a frequency of about 10^{-7}–10^{-11} per base pair during a single round of replication. (A typical gene has about 1000 base pairs; therefore, the frequency of these errors in a normal, fully grown culture of organisms having approximately 10^8 cells/ml, there are probably a number of different mutants in each milliliter of culture.

Mutations involving one (or a very few) base pairs are sometimes referred to as **point mutations.** Point mutations can result in *base-pair substitutions* in the DNA or in the insertion or deletion of a base pair (called *microinsertions* and *microdeletions*). As is the case with all mutations, the phenotypic change that comes about because of a point mutation depends on exactly where the mutation took place in the gene, what the nucleotide change was, and what product the gene normally encodes.

Base-Pair Substitutions

If a point mutation occurs within the coding region of a gene that encodes a protein, any change in the phenotype of the cell is almost certainly the result of a change in the amino acid *sequence* of the protein being produced. Figure 9.3 shows a number of base-pair substitutions that can occur in a short region of DNA within a gene that encodes a protein. The error in the DNA is transcribed into mRNA, and this erroneous mRNA in turn is used as a template and translated into protein. (Because only one strand of the DNA is used as template for the mRNA, an AT base pair does not have the same meaning as a TA base pair.) The triplet code that directs the insertion of an amino acid via a transfer RNA will thus be incorrect. What are the consequences of base-pair substitutions?

In interpreting the results of mutation, we must first recall that the genetic code is degenerate (Section

because the genes encode proteins that can diffuse through the cytoplasm of the cell. (One cannot complement mutations in regulatory sites such as promoters because these function at the DNA level.) If each homologous DNA molecule contributes a different required gene, then the cell will have all the enzymes it requires to synthesize tryptophan. Notice that complementation *does not* involve recombination. To do the test, the mutations must be in *trans*. (If one molecule has both mutations, the other is wild type and should be sufficient itself to confer the wild-type phenotype unless one of the mutations is *dominant*. Therefore, having the mutations in cis serves as a control.)

This type of complementation test, called a *cis–trans test*, is used to define whether two mutations are in the same genetic (functional) unit. The genetic unit defined by the cis–trans test is sometimes called a **cistron** (a term essentially equivalent to a gene). As noted, two mutations in the *same* cistron *cannot* complement each other, and so when complementation is found to exist, this implies that the two mutations lie in *different* cistrons (that is, different genes). The term *cistron* is now rarely used except when describing whether an mRNA has the genetic information from one gene (monocistronic mRNA) or from more than one gene (polycistronic mRNA) (Section 6.8).

✓ 9.5 Concept Check

Homologous recombination arises when closely related sequences from two genetically distinct elements are combined together in the same element. Recombination is an important evolutionary process, and cells have specific mechanisms for ensuring that recombination takes place. In eukaryotes, genetic recombination occurs as a consequence of the sexual cycle. Mechanisms of recombination also occur in prokaryotes but involve DNA transfer during the processes of transformation, transduction, and conjugation.

✓ What protein, found in all prokaryotes, facilitates the pairing required for homologous recombination?
✓ Complementation tests do not involve recombination. Explain.

9.6 Genetic Transformation

As we have noted, genetic transformation is a process by which free DNA is incorporated into a recipient cell and brings about genetic change. The discovery of genetic transformation in bacteria was one of the outstanding events in biology, as it led to experiments demonstrating that DNA is the genetic material (see the box, Origins of Bacterial Genetics). This discovery became the keystone of molecular biology and modern genetics.

A number of prokaryotes have been found to be naturally transformable, including certain species of both gram-negative and gram-positive Bacteria and some species of Archaea. However, even within transformable genera, only certain strains or species are transformable. Since the DNA of prokaryotes is present in the cell as a large single molecule, when the cell is gently lysed, the DNA pours out (Figure 9.13). Because of its extreme length (1700 μm in *Bacillus subtilis*), the DNA molecule breaks easily; even after gentle extraction the *B. subtilis* chromosome of 4.2 megabase pairs is converted to fragments of about 15 kilobase pairs. Because the DNA that corresponds to an average gene is about 1000 nucleotides, each of the fragments of purified DNA has about 15 genes. A single cell usually incorporates only one or a few DNA fragments so only a small proportion of the genes of one cell can be transferred to another by a single transformation event.

Competence

A cell that is able to take up a molecule of DNA and be transformed is said to be **competent.** Only certain strains are competent; the ability seems to be an inherited property of the organism. Competence in most naturally transformable bacteria is regulated, and special proteins play a role in the uptake and processing of DNA. These competence-specific proteins may include a membrane-associated DNA binding protein, a cell

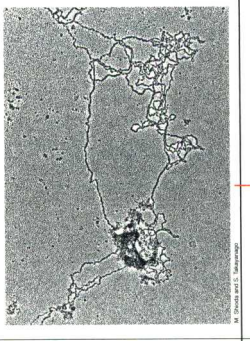


FIGURE 9.13 The prokaryotic chromosome, as shown in the electron microscope. The circular chromosome is from the hyperthermophile *Sulfolobus*, a member of the Archaea (Section 14.8). See also Figure 9.18.

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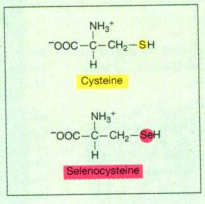
A FOCUS ON . . . Selenocysteine: The Twenty-first Amino Acid

The genetic code has codons for 20 amino acids that are assembled into proteins during translation. However, many proteins contain other amino acids. In fact, there are well over 100 different amino acids found in at least a few proteins. Until recently, it was thought that these "extra" amino acids were made by modifying one of the standard amino acids *after* it was incorporated into protein, a process called *posttranslational modification*. However, it is now clear that one of these extra amino acids is put into protein by the translational machinery itself. This one exception is *selenocysteine*.

Selenocysteine has the same structure as cysteine, but it has a selenium atom rather than a sulfur atom. It was known for some time that a few proteins contain this unusual amino acid. For example, *Escherichia coli* makes two different formate dehydrogenase enzymes and both contain a single selenocysteine residue. When the gene encoding one of these enzymes was sequenced, it was found that the codon that corresponded to the selenocysteine was a UGA. UGA is normally an efficient stop codon in *E. coli*, but it has now been demonstrated that it can be translated directly as selenocysteine in certain mRNA molecules, not only in *E. coli* but also in other prokaryotes and in eukaryotes, including humans. Therefore, selenocysteine is the twenty-first amino acid known to be encoded by the genetic code.

How can a codon sometimes be a stop codon and sometimes a sense codon *in the same chromosome?* The answer apparently lies in the *context* of the codon, the sequence of the bases surrounding the UGA codon and in their secondary structure. In certain contexts, the translational machinery interprets UGA as "selenocysteine." In all other contexts, UGA means "stop translation." Selenocysteine has its own tRNA (as do all the standard amino acids) and also has a special protein factor that brings only this tRNA to the ribosome.

Selenocysteine is even more readily oxidized than cysteine. Therefore, enzymes that contain this amino acid must be protected from oxygen. It has been proposed that UGA might once have been a normal sense codon, calling only for selenocysteine, but that the increase in oxygen in our environment following the evolution of photosynthesis (Chapter 12) selected for proteins that contain cysteine (whose codons are UGU and UGC). This allowed the coding assignment of UGA to be altered except in a few special cases. ■



Other Genetic Codes

When the genetic code was cracked during the 1960s, all the prokaryotes and eukaryotes examined were found to use the same code. When the mRNA for mammalian hemoglobin was given to the *Escherichia coli* protein-synthesizing machinery (such as ribosomes and tRNA), mammalian hemoglobin was synthesized. Therefore, the genetic code appeared to be a **universal code** in that the exact same code was used by all living systems. Once techniques became available to sequence DNA easily, genes from many organisms began to be sequenced. However, comparison of these DNA sequences to the amino acid sequence of the proteins they encode has led to a few unexpected surprises. One of these, the discovery of introns in eukaryotic genes, was very surprising but did not change our ideas about the genetic code. However, it has also been discovered that are slight variations of the "universal" genetic code (see the box, Selenocysteine: The Twenty-first Amino Acid).

The original findings of these alternative codes were in the genomes of mitochondria. So far as is known, only the mitochondria of plants use the universal code without change. The other organelles in plants, the chloroplasts, also use this standard code. The mitochondria of all other eukaryotes use codes with one or a few slight differences. A few of these variations are shown in Table 6.7. Note that there is *not* simply a mitochondrial code, although there are a few common themes, such as the general use of UGA as a tryptophan codon. It is also clear that these alternate codes are very closely related to the un
tainly derived from it e
of cells are now known
slightly different codes,
also given in Table 6.7.

FEATURE BOXES provide additional, relevant information. Some take an historical perspective, some focus on techniques and applications, and others explore a text topic in greater depth.

BOLDFACED TERMS are defined in the Glossary.

The ART in the ninth edition has been thoroughly revisd, but still maintains the use of consistent color coding.

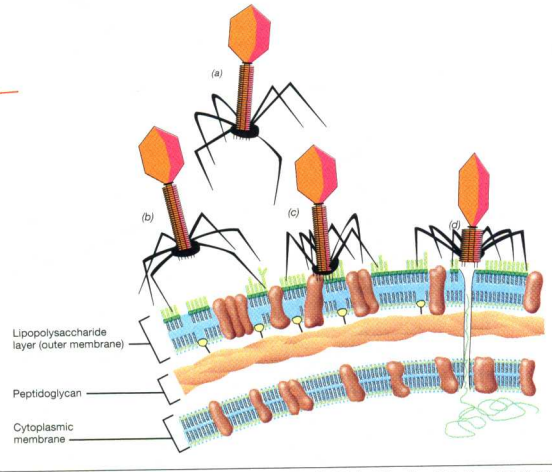


FIGURE 8.10 Attachment of T4 bacteriophage particle to the cell wall of *Escherichia coli* and injection of DNA: (a) Unattached particle. (b) Attachment to the wall by the long tail fibers interacting with core polysaccharide. (c) Contact of cell wall by the tail pins. (d) Contraction of the tail sheath and injection of the DNA. For a detailed description of the gram-negative cell wall see Section 3.8.

✓ 8.6a Concept Check

The attachment of a virion to a host cell is a highly specific process, involving the interaction of receptors on the host with proteins on the surface of the virus particle. Only after attachment has occurred can the virus or its nucleic acid penetrate the host cell.

Virus Restriction and Modification by the Host

We have already seen that one form of host resistance to virus arises when there is no receptor site on the cell surface to which the virus can attach. Another and more specific kind of host resistance occurs in prokaryotes and involves destruction of the double-stranded DNA genome of a virus after it has been injected. This destruction is brought about by host enzymes that cleave the viral DNA at one or several places, thus preventing its replication. This phenomenon is called *restriction* and is part of a general host mechanism to prevent the invasion of foreign nucleic acid. We have already discussed *restriction enzymes* and their action in some detail (Section 6.5) and noted that their cellular role is in defending against foreign DNA. Restriction enzymes are highly specific, attacking only certain sequences (generally four or six base pairs). The host protects its own DNA from the action of restriction enzymes by *modifying* its DNA at the sites where the restriction enzymes act. Modification of host DNA is brought about by methylation of purine or pyrimidine bases (in such a way that their base-pairing properties are not altered).

Some viruses can overcome host restriction mechanisms by modifications of their nucleic acids so that they are no longer subject to enzymatic attack. Two kinds of chemical modifications of viral DNA have been recognized, glucosylation and methylation. For instance, the T-even bacteriophages (T2, T4, and T6) have their DNA glucosylated to varying degrees, and the glucosylation prevents or greatly reduces endonuclease attack. Many other viral nucleic acids have been found to be modified by methylation, but glucosylation has been

REVIEW QUESTIONS

1. If an enzyme can be effectively inhibited by feedback inhibition, why would cells also have mechanisms to regulate its synthesis?
2. Compare the processing of proinsulin and the product of the *gyrA* gene of *Mycobacterium leprae* (see box, Protein Processing). In both cases an "interior" peptide is removed. However, only the latter case is considered protein splicing. Explain.
3. Describe why a protein that binds to a specific sequence of double-stranded DNA is unlikely to bind to the same sequence if the DNA is single-stranded.
4. Describe the regulation of two different operons, one having an effector that is an *inducer* and the other having an effector that is a *corepressor*.
5. The maltose regulon is inducible and is regulated by an activator protein. The lactose operon is inducible but is regulated by a repressor protein. Explain how induction can be brought about by either positive control (activator protein) or negative control (repressor protein).
6. In most cases operators are very close to the promoters they control, while activator binding sites can be some distance away. Explain why this should be so.
7. Describe how transcriptional attenuation works. What is actually being "attenuated"? Why hasn't the type of attenuation that controls several different amino acid biosynthetic pathways in *Escherichia coli* also been found in eukaryotes?
8. Describe the mechanism by which catabolic activator protein (CAP), the regulatory protein for catabolite repression, functions using the lactose operon as an example. For this operon the CAP protein is not a repressor. Describe the regulatory region of a gene for which the CAP protein *is* a repressor. (*Hint:* Think about your answer to Question 6.)
9. What are the two components that give the name to signal transduction regulation in prokaryotes? What is the function of each of the components?
10. One of the members of a two-component system is typically located in the cell membrane. What reason can you think of why this might be so?
11. Many genes are under multiple control systems. In the lactose operon, there is lactose-specific regulation and regulation by a global control system. Describe how each of the controls on the lactose operon actually functions. Why do you think both systems are necessary?

APPLICATION QUESTIONS

1. The amino acids isoleucine and valine share a common pathway for most steps in their biosynthesis. In *Escherichia coli* the first common step can be subject to feedback inhibition by valine but not by isoleucine. In most strains, though, the addition of valine does not cause isoleucine deprivation. However, in other strains it does (that is, adding valine causes isoleucine starvation and the cells stop growing). What explanation can you give for the difference between the normal "valine-resistant" strains and those whose growth is sensitive to valine?
2. What would happen to regulation from a promoter under negative control if the region where the regulatory protein binds were deleted? What if the promoter were under positive control?
3. Promoters from *Escherichia coli* under positive control are not close matches to the DNA consensus sequence for *E. coli* (Section 6.7). Why?
4. Interestingly, the attenuation control of some of the pyrimidine biosynthetic pathway genes in *Escherichia coli* actually involves coupled transcription and translation. Can you describe a mechanism whereby the cell could somehow make use of translation to help it measure the level of pyrimidine nucleotides?

REVIEW QUESTIONS challenge the student's mastery of chapter concepts.

APPLICATION QUESTIONS allow students to test their analytical and problem-solving skills.

APPENDICES provide useful tutorial and reference information.

APPENDIX 1 Energy Calculations in Microbial Bioenergetics

The information in Appendix 1 is intended to help students calculate changes in free energy accompanying chemical reactions carried out by microorganisms. It begins with definitions of the terms required to make such calculations and proceeds to show how knowledge of redox state, atomic and charge balance, and other factors are necessary to calculate free-energy problems successfully.

Definitions

1. ΔG^0 = standard free-energy change of the reaction at 1 atm pressure and 1 *M* concentrations; ΔG = free-energy change under the conditions specified; $\Delta G^{0\prime}$ = free-energy change under standard conditions at pH 7.
2. Calculation of ΔG^0 for a chemical reaction from the free energy of formation, G_f^0, of products and reactants:

$$\Delta G^0 = \Sigma\, \Delta G_f^0 \text{ (products)} - \Sigma\, \Delta G_f^0 \text{ (reactants)}$$

That is, sum the ΔG_f^0 of products, sum the ΔG_f^0 of reactants, and subtract the latter from the former.

3. For energy-yielding reactions involving H^+, converting from standard conditions (pH 0) to biochemical conditions (pH 7):

$$\Delta G^{0\prime} = \Delta G^0 + m\, \Delta G'_f(H^+)$$

where m is the net number of protons in the reaction (m is negative when more protons are consumed than formed) and $\Delta G'_f(H^+)$ is the free energy of formation of a proton at pH 7 = −39.83 kJ at 25°C.

4. Effect of concentrations on ΔG: with soluble substrates, the concentration ratios of products formed to exogenous substrates used are generally equal to or greater than 10^{-2} at the beginning of growth and equal to or less than 10^{-2} at the end of growth. From the relation between ΔG and the equilibrium constant (see item 8), it can be calculated that ΔG for the free-energy yield in practical situations differs from the free-energy yield under standard conditions by at most 11.7 kJ, a rather small amount, and so for a first approximation, standard free-energy yields can be used in most situations. However, with H_2 as a product, H_2-consuming bacteria present may keep the concentration of H_2 so low that the free-energy yield is significantly affected. Thus, in the fermentation of ethanol to acetate and H_2 ($C_2H_5OH + H_2O \rightarrow C_2H_3O_2^- + 2\,H_2 + H^+$), the $\Delta G^{0\prime}$ at 1 atm H_2 is +9.68 kJ, but at 10^{-4} atm H_2 it is −36.03 kJ. With H_2-consuming bacteria present, therefore, the ethanol fermentation becomes useful. (See also item 9.)
5. Reduction potentials: by convention, electrode equations are written in the direction, oxidant + $ne^- \rightarrow$ reductant (that is, as reductions), where n is the number of electrons transferred. The standard potential (E_0) of the hydrogen electrode, $2\,H^+ + 2\,e^- \rightarrow H_2$ is set by definition at 0.0 V at 1.0 atm pressure of H_2 gas and 1.0 *M* H^+, at 25°C. E_0' is the standard reduction potential at pH 7. See also Table A1.2.
6. Relation of free energy to reduction potential:

$$\Delta G^{0\prime} = -nF\,\Delta E_0'$$

where n is the number of electrons transferred, F is the Faraday constant (96.48 kJ/V), and $\Delta E_0'$ is the E_0' of the electron-accepting couple minus the E_0' of the electron-donating couple.

7. Equilibrium constant, K. For the generalized reaction $a\text{A} + b\text{B} \rightleftharpoons c\text{C} + d\text{D}$,

$$K = \frac{[\text{C}]^c\,[\text{D}]^d}{[\text{A}]^a[\text{B}]^b}$$

where A, B, C, and D represent reactants and products; a, b, c, and d represent number of molecules of each; and brackets indicate concentrations. This is true only when the chemical system is in equilibrium.

8. Relation of equilibrium constant, K, to free-energy change. At constant temperature and pressure,

$$\Delta G = \Delta G^0 + RT \ln K$$

where R is a constant (8.29 J/mol/°K) and T is the absolute temperature (in °K).

9. Two substances can react in a redox reaction even if the standard potentials are unfavorable, provided that the concentrations are appropriate.

Assume that normally the reduced form of A would donate electrons to the oxidized form of B. However, if the concentration of the reduced form of A were low and the concentration of the reduced form of B were high, it would be possible for the reduced form of B to donate electrons to the oxidized form of A. Thus, the reaction would proceed in the direction opposite that predicted from standard potentials. A practical example of this is the utilization of H^+ as an electron acceptor to produce H_2. Normally, H_2 production in fermentative bacteria is not extensive because H^+ is a poor electron acceptor; the E_0' of the $2\,H^+/H_2$ pair is −0.41 V. However, if the concentration of H_2 is kept low by continually removing it (a process done by methanogenic prokaryotes, which use $H_2 + CO_2$ to produce methane, CH_4, or by many other anaerobes capable of consuming H_2 anaerobically), the potential will be more positive and then H^+ will serve as a suitable electron acceptor.

Preface

As we enter the new millennium the rapid pace of basic research and the enormous possibilities for applications have thrust the field of microbiology into the forefront of the biological sciences. Through the years the science of microbiology has kept its roots firmly planted in fundamental principles. But now with the explosion of new molecular methods for both field and laboratory studies microbiologists can ask questions and do experiments that they could only dream of years ago. Indeed, as one notable microbiologist put it recently, the science of microbiology is "on a roll." It is during this exciting time that we present to students and instructors of microbiology a snapshot of microbiology today in the form of the ninth edition of *Brock Biology of Microorganisms (BBOM).*

What's New in Organization?

In the 30 years since the first edition of this book, originally titled *Biology of Microorganisms,* was published, many things have changed in the field of microbiology. The new edition of *BBOM* has also seen many changes—some minor, some not so minor—from that of the previous edition. In terms of chapter organization, the ninth edition follows along the lines of the eighth with two exceptions: (1) The chapter on "Microbial Evolution and Systematics" has been moved forward to Chapter 12 to set the stage for later chapters on microbial diversity, metabolic diversity, and ecology. With this move, every chapter in the book that deals with organisms and their activities in nature (including medical microbiology) can be seen, as it should be, within a phylogenetic context. And (2), the chapter entitled "Microbial Growth Control" has been moved back to Chapter 18 in this edition to immediately precede the medical/immunology block of chapters where it fits better as an introduction to this material.

Because medical microbiology is such an important area of our science, the disease chapter has been restructured and split into two chapters. Chapter 23 is now entitled "Person-to-Person Microbial Diseases" and exclusively covers microbial diseases transmitted in this fashion. Here one will find up-to-the-minute coverage of airborne and sexually transmitted pathogens and the diseases they cause. In Chapter 24, entitled "Animal-Transmitted, Vectorborne, and Common-Source Microbial Diseases," one finds coverage of important diseases transmitted by insects or animals such as malaria, Lyme, hantavirus and rabies, and diseases caused by a common vehicle such as foodborne and waterborne diseases. Those instructors who structure their introductory courses around a theme of medical microbiology will now find more than enough coverage of diseases and the disease process in the ninth edition.

Finally, it should also be mentioned that the new organization in *BBOM 9/e* conforms very closely to the recommendations of the Education Division of the American Society for Microbiology (ASM) for teaching introductory microbiology courses. This includes the content outline of the new ASM telecourse in microbiology, whose 12-part series produced by Oregon Public Broadcasting and funded by the Annenberg Corporation closely mirrors the organization of material in the ninth edition.

What's New in Content?

What *isn't* new? Every chapter in this book has seen revision, in some cases, very extensive revision. The authors' goal in producing this new edition was to maintain the breadth and depth of coverage that users have come to expect from this book while at the same time keeping things within bounds, both in terms of what students should be expected to master in one semester, and the overall length of an "introductory" textbook. As for previous editions of this book, the ninth has tried to strike a balance between concepts and details such that the beginning student is not overwhelmed while the more experienced student can still use the book as a general resource.

A very important point for instructors to note is the numbered head system used in this book. As for all eight previous editions, *BBOM 9/e* is constructed in a modular fashion, using numbered heads to group off major topics within each chapter. Instructors should take advantage of this system by assigning to their students as much or as little (depending on the depth and breadth desired) of the material of a given chapter as necessary. Instructors know that most textbooks can not (and should not) be covered in a single semester course, and *BBOM 9/e* is no exception. While some chapters of this book should be covered in their entirety in the introductory course, others need only be sampled. The numbered head system makes sampling convenient for

instructors and at the same time partitions concepts into more digestible portions for students.

Highlights of the revision include: (1) a more streamlined chapter on cell chemistry (now entitled "Macromolecules") to put the focus on the structure of the cells' major molecules; (2) a major new section on prokaryotic genomics (Chapter 9) to reflect the great excitement and advances occurring in this area today; (3) a major reorganization of the material on prokaryotes (Chapters 13 and 14) along phylogenetic lines to better integrate the evolutionary material in Chapter 12 with the properties of the organisms themselves; (4) extensive coverage of the exciting areas of phylogenetic probes and other fluorescent probes and their use in clinical medicine and microbial ecology (Chapters 16 and 21); (5) an expanded treatment of eukaryotic microorganisms and eukaryotic cell structure (Chapter 17); (6) a heavily reorganized immunology chapter which, following the pattern of the successful "nucleic acids" box in Chapter 6, separates out the molecular details of immunology into a box ("Molecular Biology of the Immune Response") for those instructors that wish to cover the field at the molecular level, while retaining the basic concepts of immunology in the text for those who wish to take a more practical approach to teaching this important area; and (7) an exciting description of the recent emergence of hantavirus pulmonary syndrome (in the *new* Chapter 24).

In summary, long-time users of this book will find a number of old friends between the covers and will meet a number of new ones as they go along. New users of this book will likely find it to be a refreshing, modern approach to teaching the science of microbiology. Either way, the book itself, coupled with the helpful pedagogical and instructional aids that make up the complete package (see below), should make the ninth edition of *BBOM* the most "teachable" for the instructor and the most useful to the student, of them all.

Pedagogical Aids

Previous users of this text will quickly notice the entirely new art program in *BBOM 9/e*. Every piece of art has been re-done, with special care to introduce more three dimensionality where pedagogically useful, and with a better eye for color usage. As before, color is a learning tool in *BBOM 9/e* and instructors will find that students quickly grasp the significance of color for reinforcing important concepts.

High quality photos and photomicrographs have been a mainstay of *Biology of Microorganisms* since the first edition appeared in 1970. *BBOM 9/e* is no exception and contains over 75 *new* color and black and white photos, most of which, like in previous editions, were supplied by top researchers in the field. Indeed, the combination of a more pleasing art style and superb photos should be well received by today's "visual" generation of students.

As has become a tradition with this book, *BBOM 9/e* incorporates several study aids into the body of each chapter. *Concept links* (signaled by the blue "chain link" icon) are the ties between what has just been read and related material found elsewhere in the book; *Concept Checks* (found at the end of each numbered head) are a brief overview of the previous material followed by several short questions designed to ensure understanding of critical concepts; and the *Working Glossary* (that begins each chapter) is a dictionary of the essential terms to be encountered therein. With regular use of these study aids students will (1) know where to go within the book to read more about a particular topic; (2) instantly know whether they have mastered a given concept; and (3) have a fingertip reference to the key terminology. In addition to these study aids built into each chapter, *BBOM 9/e* contains an extensive list of end of chapter study questions designed to both recall important concepts and apply them to solving problems.

This new edition of *Brock Biology of Microorganisms* comes complete with a companion Website (www.prenhall.com/brock). On this site you will find supplementary materials for each chapter, an advanced readings list, and a new learning aid introduced with the ninth edition, the "Testing Center." Instructors are well aware that students nowadays like to prepare for upcoming examinations by taking "practice exams." In the *BBOM 9/e* Testing Center students can do this by taking a "Virtual Exam" for each unit of material, testing their preparedness with thousands of on-line questions designed to help them succeed. The questions themselves have been taken from authentic examinations given in introductory microbiology courses taught at universities throughout the United States that use *BBOM* as their textbook resource. The instant feedback available on these exams will allow students to assess just how ready they are for exam day in their own classes.

Supplements

A number of supplements for instructor use accompany the ninth edition. A set of 275 full color transparencies, far more than is available with any other textbook of microbiology, accompany every adoption. Although classroom instruction is going more and more toward computer-generated presentation, the transparency is still the visual aid workhorse in many courses in microbiology; in recognition of this, the publishers are pleased to include 25 more in this edition than its predecessor. But for those instructors whose classrooms are wired for the use of laptop computers, we offer a CD-ROM containing virtually all of the art, photos, and

tables from *BBOM 9/e*. This CD, driven by a new version of the Prentice Hall "Presentation Manager" software system, gives instructors instant access to almost any visual aid in the book and allows them to be organized to fit an instructor's particular needs.

Students were very receptive to the free Website that accompanied the eighth edition of *BBOM*. Based on their comments and other reviews, we have revised the Website to be even more relevant and helpful. Along with the "Testing Center" mentioned previously, the quiz section of the Website allows students to take self-graded quizzes to test their knowledge, but the number of questions has been expanded and now contains much of the content formerly in the Study Guide. Finally a new feature called "Advances" encourages students to look ahead to the future in microbiology and to research current events using Weblinks we provide.

Between the book itself, the instructors' supplements, and the enhanced Website, we feel we offer an authoritative yet readable account of microbiology as we know it in the twenty-first century and have made available the means to teach it in a first-class way; indeed, *BBOM 9/e* is a winning package for both students and instructors alike.

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