

# Medical Microbiology

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# Medical Microbiology

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TENTH EDITION

## **Patrick R. Murray, PhD, F(AAM), F(IDSA)**

Emeritus Professor  
University of Maryland School of Medicine  
Baltimore, Maryland

## **Ken S. Rosenthal, PhD**

Adjunct Professor Infectious Diseases  
University of Georgia College of Veterinary Medicine;  
Professor of Immunology  
Augusta University, University of Georgia Medical Partnership  
Athens, Georgia;  
Emeritus Professor  
Northeastern Ohio Medical University  
Rootstown, Ohio

## **Michael A. Pfaller, MD, F(CAP), F(AAM), F(IDSA)**

Consultant  
JMI Laboratories  
North Liberty, Iowa;  
Professor Emeritus  
University of Iowa College of Medicine  
Iowa City, Iowa



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# Contents

## SECTION 1

### Introduction, 1

- 1** *Introduction to Medical Microbiology*, 2
- 2** *Human Microbiome in Health and Disease*, 5
- 3** *Infection Prevention and Control*, 11

## SECTION 2

### General Principles of Laboratory Diagnosis, 21

- 4** *Traditional Diagnostic Methods*, 22
- 5** *Molecular and Proteomic Diagnostic Methods*, 29

## SECTION 3

### Basic Concepts in the Immune Response, 35

- 6** *Elements of Host Protective Responses*, 36
- 7** *Innate Host Responses*, 46
- 8** *Antigen-Specific Immune Responses*, 61
- 9** *Immune Responses to Infectious Agents*, 81
- 10** *Antimicrobial Vaccines*, 103

## SECTION 4

### Bacteriology, 111

- 11** *Bacterial Classification, Structure, and Replication*, 112
- 12** *Bacterial Metabolism and Genetics*, 124
- 13** *Mechanisms of Bacterial Pathogenesis*, 134
- 14** *Role of Bacteria in Disease*, 143
- 15** *Laboratory Diagnosis of Bacterial Diseases*, 149
- 16** *Antibacterial Agents*, 155
- 17** *Staphylococcus and Related Gram-Positive Cocci*, 163
- 18** *Streptococcus and Enterococcus*, 176
- 19** *Bacillus*, 194

- 20** *Listeria and Related Gram-Positive Bacteria*, 200

- 21** *Mycobacterium and Related Acid-Fast Bacteria*, 209

- 22** *Neisseria and Related Bacteria*, 224

- 23** *Haemophilus and Related Bacteria*, 233

- 24** *Enterobacteriales*, 240

- 25** *Vibrio and Related Bacteria*, 254

- 26** *Pseudomonas and Related Bacteria*, 261

- 27** *Campylobacter and Helicobacter*, 269

- 28** *Miscellaneous Gram-Negative Rods*, 276

- 29** *Clostridium and Clostridioides*, 290

- 30** *Non-Spore-Forming Anaerobic Bacteria*, 301

- 31** *Treponema, Borrelia, and Leptospira*, 310

- 32** *Mycoplasma*, 323

- 33** *Rickettsia, Ehrlichia, and Related Bacteria*, 326

- 34** *Chlamydia*, 336

## SECTION 5

### Virology, 343

- 35** *Viral Classification, Structure, and Replication*, 344

- 36** *Mechanisms of Viral Pathogenesis*, 361

- 37** *Role of Viruses in Disease*, 372

- 38** *Laboratory Diagnosis of Viral Diseases*, 378

- 39** *Antiviral Agents and Infection Control*, 384

- 40** *Papillomaviruses and Polyomaviruses*, 392

- 41** *Adenoviruses*, 402

- 42** *Human Herpesviruses*, 409

- 43** *Poxviruses*, 432

- 44** *Parvoviruses*, 438

**45** *Picornaviruses*, 443  
**46** *Coronaviruses*, 455  
**47** *Noroviruses and Small RNA Enteric Viruses*, 463  
**48** *Paramyxoviruses*, 467  
**49** *Orthomyxoviruses*, 479  
**50** *Rhabdoviruses, Filoviruses, and Bornaviruses*, 489  
**51** *Reoviruses*, 497  
**52** *Togaviruses, Flaviviruses, and Rubivirus (Matonaviridae)*, 505  
**53** *Bunyavirales: Bunyaviridae and Arenaviridae*, 517  
**54** *Retroviruses*, 523  
**55** *Hepatitis Viruses*, 540  
**56** *Prion Diseases*, 557

## SECTION 6

### *Mycology*, 563

**57** *Fungal Classification, Structure, and Replication*, 564  
**58** *Pathogenesis of Fungal Disease*, 570  
**59** *Role of Fungi in Disease*, 580  
**60** *Laboratory Diagnosis of Fungal Disease*, 582  
**61** *Antifungal Agents*, 592  
**62** *Superficial and Cutaneous Mycoses*, 604  
**63** *Subcutaneous Mycoses*, 614  
**64** *Systemic Mycoses Caused by Dimorphic Fungi*, 624  
**65** *Opportunistic Mycoses*, 641  
**66** *Fungal and Fungal-Like Infections of Unusual or Uncertain Etiology*, 667

## SECTION 7

### *Parasitology*, 679

**67** *Parasitic Classification, Structure, and Replication*, 680  
**68** *Pathogenesis of Parasitic Diseases*, 687  
**69** *Role of Parasites in Disease*, 691  
**70** *Laboratory Diagnosis of Parasitic Disease*, 693  
**71** *Antiparasitic Agents*, 702  
**72** *Intestinal and Urogenital Protozoa*, 710  
**73** *Blood and Tissue Protozoa*, 724  
**74** *Nematodes*, 745  
**75** *Trematodes*, 764  
**76** *Cestodes*, 775  
**77** *Arthropods*, 787

## SECTION 8

### *Infectious Diseases by Organ System*, 805

**78** *Sepsis and Cardiovascular Infections*, 806  
**79** *Ear, Nose and Throat Infections*, 809  
**80** *Lower Respiratory Tract Infections*, 812  
**81** *Eye, Brain and Nervous System Infections*, 815  
**82** *Genitourinary Tract Infections*, 820  
**83** *Gastrointestinal Infections*, 823  
**84** *Musculoskeletal, Skin, and Soft Tissue Infections*, 825  
**85** *Hematopoietic and Lymphatic System Infections*, 830  
*Index*, 835

# Video Contents

## SECTION 3

### Basic Concepts in the Immune Response

#### Chapter 6 Elements of Host Protective Responses

Video 6.1 Cells and organs of the immune system.  
From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

#### Chapter 7 Innate Host Responses

Video 7.1 Pathways of complement activation.  
From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

Video 7.2 Cell Migration and phagocytosis.  
From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

Video 7.3 Inflammation.  
From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

Video 7.4 Capture and presentation of protein antigens by dendritic cells.  
From Murray, P.R., Rosenthal, K., Pfaller, M.A., 2021. Medical Microbiology, ninth ed. Elsevier.

#### Chapter 8 Antigen-Specific Immune Responses

Video 8.1 Steps in maturation of lymphocytes.  
From Murray, P.R., Rosenthal, K., Pfaller, M.A., 2021. Medical Microbiology, ninth ed. Elsevier.

Video 8.2 T cell Education.

From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

Video 8.3 Antigen Presentation.

From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

Video 8.4 Antibody Classes.

From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

#### Chapter 9 Immune Responses to Infectious Agents

Video 9.1 Anti-bacterial defenses.

From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

Video 9.2 Antibody mediated opsonization and phagocytosis of microbes.

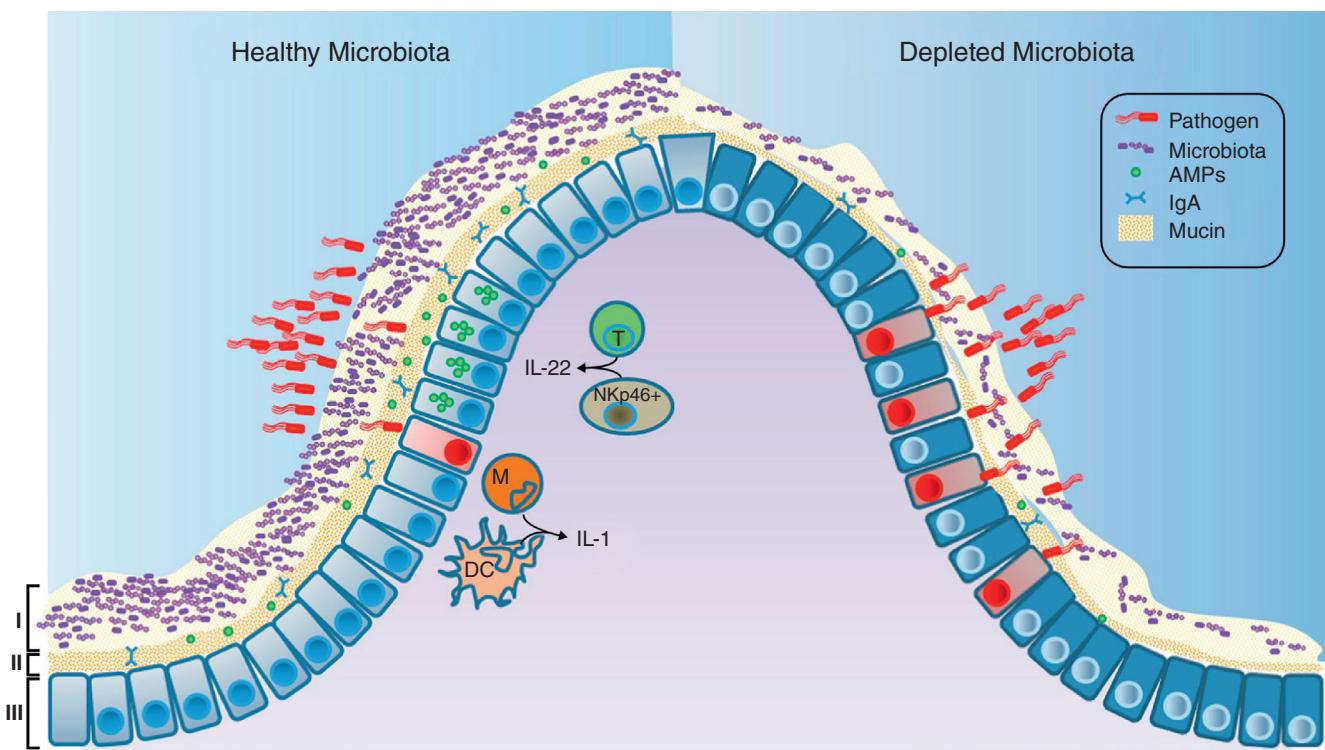
From Murray, P.R., Rosenthal, K., Pfaller, M.A., 2021. Medical Microbiology, ninth ed. Elsevier.

Video 9.3 Anti-viral defenses.

From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.

Video 9.4 Immunopathogenesis.

From Male, D., Stokes Peebles Jr., R., Male, V., 2020. Immunology, ninth ed. Elsevier.



**Fig. 2.2** Intestinal microbiota protection against enteric infections. (I) Saturation of colonization sites and consumption of nutrients limit pathogen access to host tissues; (II) the microbiota prime innate immunity by stimulating mucin production, immunoglobulin (Ig)A, and antimicrobial peptides (AMPs); and (III) the microbiota stimulate interleukin (IL)-22 expression, which increases epithelial resistance, and IL-1 $\beta$  production, which promotes recruitment of inflammatory cells. (From Khosravi, A., Mazmanian, S., 2013. Disruption of the gut microbiome as a risk factor for microbial infections. *Curr. Opin. Microbiol.* 16, 221–227.)

## Evolution of the Microbiome and Normal Flora

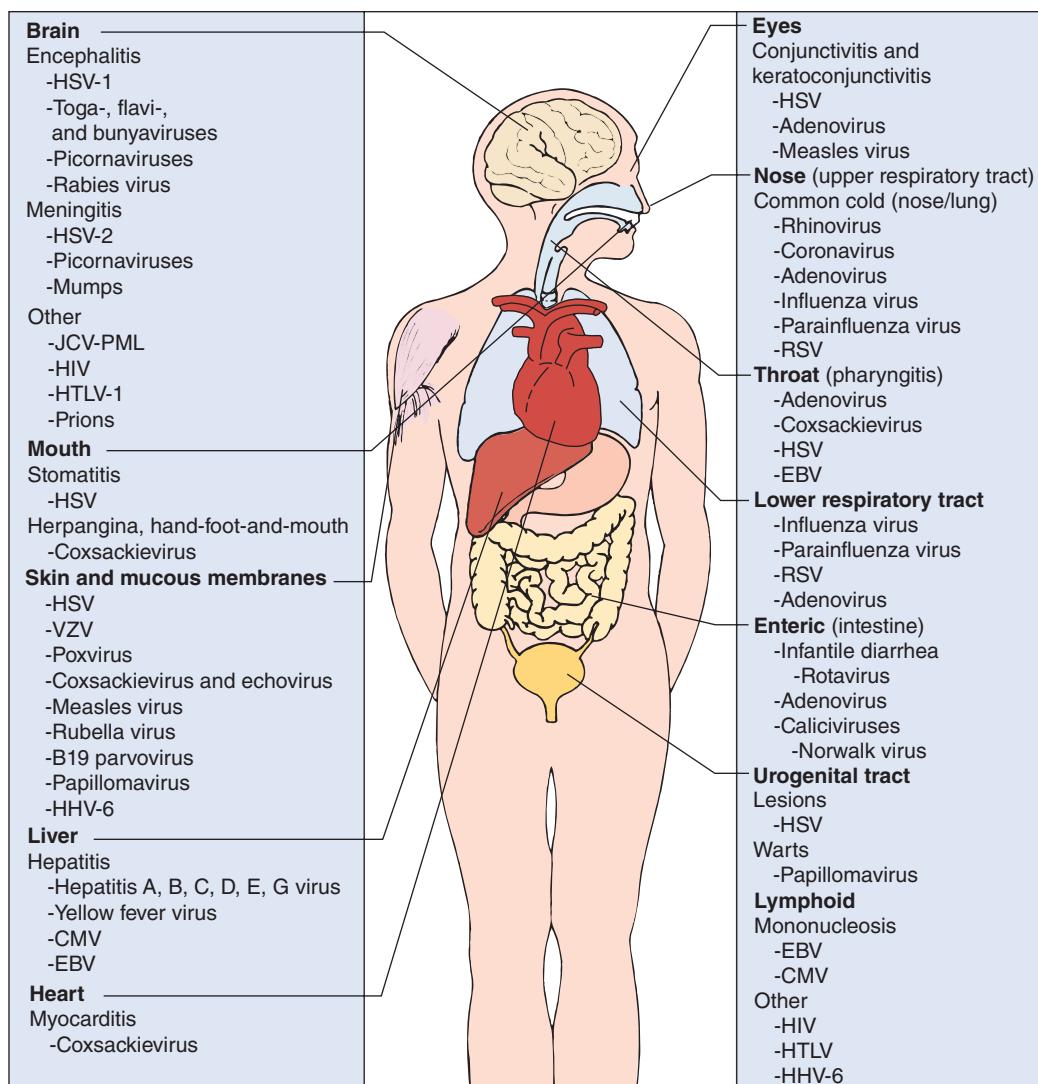
The **normal flora** or microbial population of a particular site of the body consists of a unique community of core and secondary microbiota that evolved through a symbiotic relationship with the host and a competitive relationship with other species. The host provides a place to colonize, nutrients, and some protection from unwanted species (innate immune responses). The microbes provide needed metabolic functions, stimulate innate and regulatory immunity, and prevent colonization with unwanted pathogens (Fig. 2.2). The ability to tolerate the amount of oxygen or lack thereof (redox state) and the pH and salt concentration, as well as to scavenge essential minerals and harvest and metabolize the available nutrients, determines the numbers and nature of the species that populate a site of the body. Anaerobic or facultative anaerobic bacteria colonize most of the sites of the body because of the lack of oxygen in sites such as the mouth, intestine, and genitourinary tract.

The composition of the microbiota is influenced by personal hygiene (e.g., use of soap, deodorants, mouthwash, skin peels, enemas, vaginal douches), diet, water source, medicines (especially antibiotics), and exposure to environmental toxins. Drinking well water versus chlorinated city water or a diet consisting of fiber, sugar, or fats can select for different intestinal bacteria based on their ability to use the essential minerals (e.g., iron) and nutrients. Alteration

of the environment with foods or medicines can also alter the microbiota (Fig. 2.3). These changes can be acceptable if the core microbiome and critical functional properties of the microbiome are maintained but can result in disease if these functions are lost. Historically, the greatest concern with the use of broad-spectrum antibiotics was the selection of resistant bacteria; however, a greater concern should be the disruption of the microbiome and loss of essential functions. Because antibiotics are not completely selective for the targeted pathogen, the use of antibiotics is always associated with some degree of toxicity.

Of the more than 150 species of bacteria that colonize the gut of a healthy adult individual, most are members of Bacteroidetes (e.g., *Bacteroides*) and Firmicutes (e.g., *Clostridium*, *Ruminococcus*, *Faecalibacterium*, *Lactobacillus*), with Actinobacteria (e.g., *Bifidobacterium*) present in smaller numbers. Interestingly, the importance of many of these bacteria was not appreciated before gene sequencing was used to identify and quantitate the gut microbiota. Within the colon, some bacteria establish their niche by waging interspecies warfare with bacteriocins (e.g., colicins produced by *Escherichia coli*), other antibacterial proteins, and metabolites that deter other species from growing. These molecules also benefit the host by eliminating invading bacteria including *Salmonella*, *Shigella*, *Clostridiooides difficile*, *Bacillus cereus*, and other pathogens. The bacteria must also resist antimicrobial peptides and immunoglobulin (Ig) A produced by the host and released into the bowel.

Metabolism of nutrients plays a major role in the symbiotic relationship between the human host and microbe.



**Fig. 37.1** Major target tissues of viral disease. CMV, Cytomegalovirus; EBV, Epstein-Barr virus; HHV-6, human herpesvirus 6; HIV, human immunodeficiency virus; HSV, herpes simplex virus; HTLV, human T-cell lymphotropic virus; JCV-PML, JC virus induction of progressive multifocal leukoencephalopathy; RSV, respiratory syncytial virus; VZV, varicella-zoster virus.

Although enteroviruses (picornaviruses) are spread by the fecal-oral route, they usually cause only mild or no gastrointestinal symptoms. Instead, these viruses establish a viremia, spread to other target organs, and then cause clinical disease.

Enveloped viruses, including CMV and HIV, may infect the colon from the blood stream or by anal sex.

## SKIN INFECTIONS AND MANIFESTATIONS

Skin is a barrier to virus infection but small cuts or abrasions in the skin allow infection by direct contact with lesions or contaminated fluids (HSV) or fomites (e.g., towels). Oral and genitourinary mucop epithelium are prominent targets of viruses. Most of these infections present at the site of infection (e.g., papillomaviruses, HSV, molluscum contagiosum). Many skin infections and the rashes that result from virus infections result from systemic spread

of the virus to the skin or hypersensitivity reactions elicited by the infection.

## INFECTIONS OF OTHER ORGANS AND TISSUES

Viremic spread, either free in blood or cell associated in monocytes, brings viruses to the liver where the immunotolerance and hepatocyte biosynthetic machinery foster viral replication. The liver is often a source for a secondary viremia but can also be damaged by the infection. Cell-mediated immunity is necessary to control liver infections by hepatitis A, B, D, C, G, and E viruses and yellow fever virus but also causes the symptoms of hepatitis.

The heart and other muscles are also susceptible to viral infection and damage causing myocarditis or pericarditis. Infection of secretory glands, accessory sexual organs, and mammary glands by CMV generates virus to spread the virus and the inflammatory response to **mumps** causes the