SPECIAL COMMUNICATION

Design and Implementation of Core Knowledge Objectives for Medical Microbiology and Immunology

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ABSTRACT

Academic curriculum subcommittees of the Association of Medical School Microbiology and Immunology Chairs (AMSMIC) have developed a series of core knowledge objectives for courses in medical microbiology and immunology. Detailed and specific objectives were created by separate subcommittees on Fundamental Microbiology, Host Defenses and Pathogenesis. The academic subcommittees consisted of meeting conferees and distinguished faculty that met at biennial meetings. In 2006 the faculty developed a practical wiki site for membership guidance and revision of the objective documents, allowing changes, contributions and corrections to the core objectives. The wiki afforded the identification of problematic areas and provided a process for ranking objectives, using a numerical rating scale, which provided quantifiable information. The wiki site greatly facilitated the evaluation of core knowledge objectives and was formulated into a condensed set of parameters listing specific academic areas of importance. The final documents contain core competency objectives and provide a format for academic medical microbiology and immunology departments on a national and international level.

INTRODUCTION

In 1986 the Association of Medical Schools Microbiology and Immunology Chairs (AMSMIC) hosted an inaugural educational conference at the Ocean Creek Conference Center in Myrtle Beach, South Carolina. The meeting brought together a wide range of plenary sessions and provided a number of afternoon workshops, covering specific topics on General and Molecular Microbiology, Immunology, Pathogenic Bacteriology, Virology, Parasitology and Mycology. The purpose of these sessions was to provide a format to discuss teaching modalities in medical education. The meetings have continued on a biennial basis since the inaugural session, with the concept of similar workshops facilitated by a variety of speakers and the objective to share experiences in teaching and implementing educational outcomes in medical microbiology and immunology. Additional evening
workshops were also conducted on “Optimal Course Content.” In 1991 Cantor and coworkers, in a survey of 1369 medical educators, observed a strong endorsement of the need for “fundamental changes” or “thorough reform” in medical education. In the mid to late 1990’s a major shift in the modes of medical student education was underway. The distinct levels of cognition, organized into a taxonomy of general objectives by Bloom, provided a basis for higher education. Other avenues of concern, modifications and changes in the medical curriculum have occurred more recently. As modification of the medical curriculum occurred, the “Myrtle Beach” meetings focused on curriculum change, innovative techniques and evaluation formats in medical education. At the 1998 meeting the former course categories were condensed to Pathogenesis/Infectious Disease and Immunology/Host Defenses. Since that time the meetings have, in addition to curriculum discussions, centered on bioterrorism, computerization techniques and up-to-date innovations. The fields of medical microbiology and immunology are rapidly evolving with new research and the appearance of unrecognized pathogens and discovery of new immunological diseases. There is a need therefore to provide a regularly updated resource for core knowledge objectives to aid in the development and improvement of existing discipline-based and integrated medical school curriculums. At the 2006 meeting the curriculum sessions were divided into three distinct areas: (1) Fundamental [Basic] Microbiology, (2) Host Defenses [Immunology] and (3) Pathogenesis [Infectious Disease]. Following the meeting a wiki website was created and made available for the membership to establish core knowledge objectives in these areas. This paper describes the outcome and current status of the medical microbiology and immunology core knowledge objectives project. However, the project is a continuum and the objectives are continuously open for modification at the wiki website and at biennial AMSMIC meetings.

**MATERIALS AND METHODS**

Beginning in 1998, three groups of faculty at the Myrtle Beach meeting were charged to provide a computerized listing of core knowledge objectives for the disciplines medical microbiology and immunology, including the broad areas of virology, medical parasitology and mycology. In May 2006, at the 11th Educational Strategies Meeting in Myrtle Beach, the learning objectives were further refined to three main academic areas, namely Fundamental Microbiology, Host Defenses and Pathogenesis. Each section subcommittee, directed by faculty facilitators, was responsible for compiling and documenting core knowledge objectives and a wiki website was created following the meeting to facilitate revision of the proposed objectives. The website carried the following general instructions:

> “After you have logged in, click on your name next to the logoff link and change your password. Please remember to click

"Update Password" link to change your password. You may then enter and exit the website.

To make corrections or changes in any of the Workshop articles click on the button located on the left side. If you then click on "Edit" you can add your changes to the page. You must scroll down and click the "Save" button for changes to become effective. For major changes to the page please deselect the "minor changes" option before you click "Save."

To create sub pages within each article enclose the word you wish to become a link to a sub page with double brackets e.g. [[link]]. After you save the page a new link will appear in the article. Click on the link to create the new page and to start adding content. This same technique can be used to create links to other sub pages within each article. A complete list of acceptable syntax within the wiki is located below the save button.

The Education Committee requests that you not change information in any more than two Workshops. If you have questions or need additional information please contact the Workshop Director.

Thank you for your efforts and assistance in designing the knowledge objectives for Medical Microbiology & Immunology.”

In addition, instructions for each academic area, including Fundamental [Basic] Microbiology, Host Defenses [Immunology] and Pathogenesis [Infectious Diseases], are available at:


**RESULTS**

To date 117 responses have been obtained, representing 56 different medical schools in the United States, Canada, Dominica and Grenada. Individual responses totaled 1,847, resulting in 63, 15 and 55 revisions for the sections on Fundamental [Basic] Microbiology, Host Defenses [Immunology] and Pathogenesis [Infectious Diseases], respectively. For the section on Fundamental Microbiology, the working group ranked each item based on importance in the curriculum using a scale of ‘3’ for
knowledge that was essential for inclusion in the curriculum, ‘2’ for important knowledge that should be included in the curriculum if time is available, and ‘1’ for information that was deemed trivial and therefore not required in the curriculum. This version of the core knowledge objectives is shown in Table 1 (see Appendix), with an average of the individual rankings. In addition, Table 1 represents the four major divisions of microbiology, including Basic Bacteriology, Basic Mycology, Basic Parasitology and Basic Virology. The division within Basic Bacteriology is divided into 9 subdivisions that represent (A) Structure and Function of Bacteria, (B) Nutrition and Growth, (C) Microbiological Techniques, (D) Physiology and Metabolism of Bacteria, (E) Microbial Genetics, (F) Antibiotic Susceptibility Testing, (G) Antibiotics, (H) Physical and Chemical Agents for Control of Microbial Growth and (I) Host-Parasite/Pathogen Relationships. The second division entails Basic Mycology and includes subdivisions on (A) Principles, (B) Fungal Classification and (C) Antifungal Agents. The third division entails Basic Parasitology and includes subdivisions on (A) Principles and (B) Classification. The fourth and last division entails Basic Virology and includes subdivisions on (A) Principles of Structure and Function, (B) Virus Multiplication and Infectivity and (C) Antiviral Agents.

A second section on Host Defenses Core Knowledge Objectives is represented in Table 2 (see Appendix). The rankings, far right-hand column, are similar to Table 1, with a value of ‘3’ for essential knowledge, ‘2’ for important knowledge and ‘1’ for information that was found to be trivial and not an absolute requirement for the curriculum. As indicated, Table 2 is composed of three major divisions, including Division I: Basic Concepts in Immunology, Division II: The Immune System and Disease and Division III: Applied Immunology. Division I on Basic Concepts in Immunology consists of 9 sections that represent (A) General Principles, (B) Development of Cells and Function of Organs, (C) Innate Immunity, (D) Antibodies and Antigens, (E) Antigen Receptor Diversity, (F) MHC, Antigen Processing and Presentation, (G) B and T Lymphocyte Activation, (H) Regulation of the Immune Response and (I) Cell Mediated Immunity. Division II on The Immune System and Disease consists of 6 sections that represent (A) Hypersensitivities, Allergy and Asthma, (B) Autoimmunity, (C) Transplantation Immunology, (D) Immunodeficiencies – Congenital and Acquired, (E) Tumor Immunology and (F) Immunity to Microbes and Vaccines. The final division, Division III: Applied Immunology, is subdivided into 2 sections that represent (A) Immunotherapeutics and (B) Immunodiagnostics. The final major division on core knowledge objectives for medical microbiology and immunology, Pathogenesis, is represented in Table 3 (see Appendix). The right-hand column represents values of ‘3’ for information that is essential knowledge to be included, ‘2’ for information that is important knowledge to be included if there is time in the curriculum and ‘1’ for information that is trivial knowledge not required in a curriculum on Pathogenesis/Infectious Diseases. In addition, Table 3 includes two major divisions, Essential Concepts in Infectious Pathogenesis and Systems-Based Diseases. The division on Essential Concepts in Infectious Pathogenesis is subdivided into 4 subdivisions that represent (A) Encounter with Pathogen, (B) Invasion and Dissemination, (C) Outcomes of Infection, (D) Treatment and Prevention. The second major division on Systems-Based Diseases consists of eleven subdivisions that represent (A) Upper Respiratory Tract Infections, (B) Lower Respiratory Tract Infections, (C) Cardiac Infections, (D) Gastrointestinal Infections, (E) Genitourinary Infections, (F) Genital Tract, (G) Musculoskeletal Infections, (H) Infections of the Nervous System, (I) Degenerative Brain Diseases, (J) Zoonotic Diseases and (K) Opportunistic Infections.

DISCUSSION

Over the past two decades the professional curriculum for medicine and other allied health professions has continued to change. In the specialty of medicine the Liaison Committee for Medical Education (LCME), which accredits complete and independent M.D.-granting programs, is recognized as the reliable authority by the nation’s medical schools and the U.S. Department of Education for this purpose. In effect, all U.S. and Canadian medical schools operate under the auspices of the LCME accreditation program. Since the LCME inception in 1942, numerous changes have altered and shaped the medical curriculum. In recent years, periodic review and amendment of the standards for the institutional setting, educational program for the M.D. degree, medical students, faculty and educational resources have all played a significant role in modification of the modern day medical school. In addition, new and progressive methods of teaching and changes in the curriculum over the past century have led to a variety of approaches. In general, didactic lectures and paper examinations have been replaced in favor of problembased learning, team-based learning, e-based small group, simulation-based learning and a shift to the use of computerized and block testing modalities, respectively. For medical microbiology and immunology, a single or dual course presentation under direction of the respective faculty remains preferable to provide an appropriate foundation in the period required for basic science. To the contrary, several medical schools have been able to integrate “some” or “a large portion” of medical microbiology and immunology into other coursework. The purpose of developing core knowledge objectives was to provide, not regarding either stand-alone or integrated coursework, guidelines for those minimal concepts and principles that are essential for the integration of medical microbiology and immunology into the practice of medicine. These guidelines provide a yardstick with which all institutions can measure the mastery of basic principles as well as to evaluate understanding and competency of their students, regardless
of the curriculum used. The continuum of core knowledge objectives will be facilitated by future meetings and underscored by faculty participation at the wiki website.

ACKNOWLEDGMENTS

The authors wish to acknowledge and recognize the resources, collaborative efforts and support of AMSMIC [http://www.amsmic.org/index.htm], members of the Education Committee (Drs. Jerry W. Simecka, Agnes A. Day, Richard V. Goering, John M. Quarles, C. Jeffery Smith, Keith E. Weaver), program support (Paul Montgomery, Paul V. Phibbs) and meeting conferees for valuable assistance and guidance.

REFERENCES

### Table 1. Fundamental/Basic Microbiology Core Knowledge Objectives

**DIVISION I: BASIC BACTERIOLOGY**

<table>
<thead>
<tr>
<th>A. STRUCTURE AND FUNCTION OF BACTERIA</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>1. Compare and contrast prokaryotic and eukaryotic cells, particularly with respect to nuclear membranes, DNA structure, ribosomes, and cell walls</td>
<td>3.0</td>
</tr>
<tr>
<td>2. Describe the morphology and arrangement of bacterial cells using acceptable scientific terms (cocci, bacilli, etc)</td>
<td>3.0</td>
</tr>
<tr>
<td>3. Explain the use of the Gram and acid-fast stains</td>
<td>3.0</td>
</tr>
<tr>
<td>4. List some important gram-positive, gram-negative, and acid-fast bacteria and their morphology and arrangement</td>
<td>3.0</td>
</tr>
<tr>
<td>5. Explain how the Gram stain works (why are gram + bacteria blue and gram - bacteria red?)</td>
<td>2.5</td>
</tr>
<tr>
<td>6. Describe the structure and arrangements of flagella</td>
<td>1.0</td>
</tr>
<tr>
<td>7. Describe the functions of flagella</td>
<td>3.0</td>
</tr>
<tr>
<td>8. State another name for flagella (H-antigen)</td>
<td>1.8</td>
</tr>
<tr>
<td>9. Describe the structure of pili/fimbriae</td>
<td>1.0</td>
</tr>
<tr>
<td>10. Describe the functions of pili/fimbriae</td>
<td>3.0</td>
</tr>
<tr>
<td>11. Explain antigenic (phase) variation of pili or other cell surface proteins and describe its clinical significance</td>
<td>2.8</td>
</tr>
<tr>
<td>12. Describe the structure bacterial capsules</td>
<td>3.0</td>
</tr>
<tr>
<td>13. Describe the role of bacterial capsules in pathogenicity</td>
<td>3.0</td>
</tr>
<tr>
<td>14. List other terms used to describe capsules (e.g., K antigen, slime layer)</td>
<td>2.4</td>
</tr>
<tr>
<td>15. Describe the quellung reaction</td>
<td>1.3</td>
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<tr>
<td>16. Describe the formation and importance of a bacterial biofilms</td>
<td>3.0</td>
</tr>
<tr>
<td>17. Compare and contrast the structure of Gram-positive and Gram-negative cell walls</td>
<td>3.0</td>
</tr>
<tr>
<td>18. Describe the importance of peptidoglycan to bacteria</td>
<td>3.0</td>
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<tr>
<td>19. Explain the importance of peptidoglycan as a target for some antibiotics</td>
<td>3.0</td>
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<tr>
<td>20. Describe the biological activities in humans of Peptidoglycan</td>
<td>2.2</td>
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<tr>
<td>21. Explain what lysozyme is, where it is found, and its biological activity</td>
<td>2.0</td>
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<tr>
<td>22. Describe where teichoic acids are found and their importance</td>
<td>2.7</td>
</tr>
<tr>
<td>23. Describe the components and functions of the outer membrane of Gram-negative bacteria</td>
<td>3.0</td>
</tr>
<tr>
<td>24. Describe pores of Gram-negative bacteria and their importance</td>
<td>2.9</td>
</tr>
<tr>
<td>25. Discuss the structure and biological activities of endotoxin</td>
<td>3.0</td>
</tr>
<tr>
<td>26. Describe the Type III secretion system, including where it is found and its importance to pathogenicity</td>
<td>1.5</td>
</tr>
<tr>
<td>27. Describe the type IV secretion system and its importance to pathogenicity</td>
<td>2.9</td>
</tr>
<tr>
<td>28. Describe how to prepare protoplasts and spheroplasts and their importance</td>
<td>1.5</td>
</tr>
<tr>
<td>29. Explain the term “L-form”</td>
<td>1.1</td>
</tr>
<tr>
<td>30. Describe why mycoplasmas are unique among the bacteria</td>
<td>1.1</td>
</tr>
<tr>
<td>31. Describe the structure and functions of cytoplasmic membranes in bacteria</td>
<td>3.0</td>
</tr>
<tr>
<td>32. Describe a mesosome and where they are most commonly found</td>
<td>2.0</td>
</tr>
<tr>
<td>33. Explain the term “penicillin-binding protein”</td>
<td>1.0</td>
</tr>
<tr>
<td>34. Give another name for this protein (transpeptidase)</td>
<td>2.9</td>
</tr>
<tr>
<td>35. Explain its function in bacteria</td>
<td>2.6</td>
</tr>
<tr>
<td>36. Describe the major contents of bacterial cytoplasm</td>
<td>2.8</td>
</tr>
<tr>
<td>37. Describe the structure and functions of endospores</td>
<td>1.2</td>
</tr>
<tr>
<td>38. Name the two major genera of bacteria which produce endospores (Clostridium, Bacillus)</td>
<td>3.0</td>
</tr>
<tr>
<td>39. Describe the primary similarities and differences between the Clostridium and Bacillus (e.g., Gram reaction, oxygen requirements)</td>
<td>2.8</td>
</tr>
<tr>
<td>40. Describe the methods used to classify bacteria</td>
<td>2.8</td>
</tr>
<tr>
<td>41. Describe the methods used to identify bacteria in the clinical laboratory</td>
<td>1.4</td>
</tr>
<tr>
<td>42. List some important gram-positive, gram-negative, and acid-fast bacteria and their morphology and arrangement</td>
<td>3.0</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>B. NUTRITION AND GROWTH</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Explain the function of siderophores</td>
<td>3.0</td>
</tr>
</tbody>
</table>
2. Explain the term “fastidious” with respect to bacterial nutrition 2.2
3. Describe the classification of bacteria based upon oxygen requirements 2.9
4. List examples of each 3.0
5. Describe the classification of bacteria based upon temperature requirements 1.8
6. Describe the importance of proper pH for microbial growth 1.8
7. Describe the importance of proper osmotic pressure for microbial growth 1.2
8. Explain the term “halophile” 1.7
9. Name two halophilic pathogens (*V. vulnificus*, *V. parahaemolyticus*) 2.2
10. Explain the importance of growing in a high salt concentration with respect to *S. aureus* and *Enterococcus* 1.9
11. Explain the term “generation time” and the various factors that can affect it 3.0
12. Describe the four growth phases of bacteria and explain the importance of each 3.0
13. Describe the concept of quorum sensing and its importance 2.7

### C. MICROBIOLOGICAL TECHNIQUES

1. Describe how to perform a Gram stain, explaining the purpose of each manipulation or reagent 2.3
2. Explain how the Gram stain works (i.e., why are gram-positive bacteria blue and gram-negative bacteria red?) 2.7
3. Explain how to obtain a pure culture of bacteria 2.0
4. Demonstrate or describe the use and care of a bright-field microscope 1.1
5. Describe microscopic methods used to observe microbial pathogens 1.6
6. Differentiate between nonselective, selective, and differential media 2.9
   a. list common examples 2.6

### D. PHYSIOLOGY AND METABOLISM OF BACTERIA

1. Explain the following terms:
   a. glycolysis 2.9
   b. fermentation 2.9
   c. aerobic respiration 3.0
   d. anaerobic respiration 3.0
2. Using the terminology used to classify bacteria based upon oxygen requirements, list examples of bacterial species which typically perform the above metabolic processes 2.6
3. In general terms (not exact numbers) describe the amount of energy (ATP) generated by each of the above metabolic processes 2.3
4. Explain how the metabolic capabilities of bacteria can relate to pathogenicity 2.5
5. Explain how the metabolic capabilities of bacteria relate to identification of genera/species 2.2
6. Discuss the transport processes: active, passive, etc. 1.8

### E. MICROBIAL GENETICS

1. Describe the use of the terms “transcription” and “translation” 2.9
2. Define:
   a. mutation 3.0
   b. base substitution 2.7
   c. frame-shift mutation 2.7
   d. genotype 2.6
   e. phenotype 2.7
3. Describe an operon and its regulation mechanisms 2.6
4. Describe DNA repair mechanisms in bacteria 1.6
5. Describe transformation as it occurs in bacteria 3.0
6. Define:
   a. transfection 2.7
   b. homologous recombination 2.6
   c. nonhomologous recombination 2.5
   d. donor 3.0
1. Define:  
   a. male and female bacteria  
   b. F factor  
   c. plasmid  
   d. sex pilis  
   e. Hfr cell  
   f. episome  

8. Define:  
   a. F+  
   b. Hfr cell  
   c. F'  

7. Describe conjugation as it typically occurs in Gram-negative bacteria when the donor is:  
   a. F+  
   b. Hfr cell  
   c. F'  

9. Describe resistance transfer factors and discuss their significance to human medicine  

10. Describe the environmental pressures which favor the development of multiply antibiotic resistant bacteria  

11. Describe pathogenicity islands  

12. Define “insertion sequence” and “transposon” and discuss their importance  

13. Discuss selective pressures that can lead to antibiotic resistance  

14. Describe the essential features of bacterial viruses  

15. Define:  
   a. bacteriophage  
   b. capsid  
   c. capsomere  

16. Describe, in words or by a sketch, the lytic cycle as it occurs in bacteriophage infected bacteria  

17. Define “lytic”, “virulent”, and “temperate” phages  

18. Describe the lysogenic cycle (lysogeny)  

19. Define “ prophage”  

20. Define “lysogenic conversion” and discuss the clinical significance  

21. Describe transduction as it occurs in bacteria  

22. Define generalized transduction  

23. Define specialized transduction  

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**F. ANTIBIOTIC SUSCEPTIBILITY TESTING**  
1. Discuss the basis on which antibiotics are selected  
2. List side effects of antimicrobial agents and describe what is meant by each  
3. Discuss the use of antibiotic susceptibility testing  
4. Describe the basic procedures used to perform antimicrobial susceptibility testing and to interpret the test results:  
   a. broth dilution  
   b. agar plate dilution  
   c. agar disk diffusion (Kirby-Bauer)  
   d. gradient diffusion (E-test)  
   e. colorimetric (chromogenic)  
5. Discuss the pros and cons of each of the above methods of susceptibility testing  
6. Define MIC and MBC  

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**G. ANTIBIOTICS**  
1. Define the following terms as they apply to antimicrobial agents:  
   a. broad-spectrum  
   b. narrow-spectrum  
   c. expanded-spectrum  
2. For the following antimicrobial agents, discuss the primary mode of action, mechanisms of bacterial resistance, spectrum of activity, and any unique characteristics:
a. sulfonamides 2.9
b. trimethoprim 2.9
c. dapsone 2.1
d. daptomycin 2.1
e. isoniazid 2.6
f. ethambutol 1.7
g. pyrazinamide 1.6
h. beta-lactams (list examples) 3.0
i. cephalosporins (list examples; indicate differences in generations) 3.0
j. cephamycins 1.3
k. carbapenems (imipenem) 2.4
l. monobactams (aztreonam) 2.3
m. vancomycin 3.0
n. cycloserine 1.1
o. bacitracin 2.2
p. polymyxin 2.2
q. quinolones and fluoroquinolones (list examples) 3.0
r. rifampin 2.7
s. aminoglycosides (list examples) 3.0
t. tetracyclines 3.0
u. chloramphenicol 2.7
v. macrolides 3.0
w. lincosamides (clindamycin) 2.7
x. streptogramins and oxazolidinones 2.1
y. nitrofurans 1.3
z. metronidazole 3.0

3. Define bactericidal and bacteriostatic drugs 3.0
4. Explain why cell wall and membrane active agents are usually bactericidal 3.0
5. Draw the essential features of a beta-lactam antibiotic 1.2
6. Explain how a beta-lactamase works 1.3
7. Explain what clavulanic acid, tazobactam, and sulbactam have in common and what they are used for in clinical medicine 2.6
8. Explain why some antimicrobial agents (e.g., cell wall active) are most effective against rapidly growing cells while other agents (e.g., membrane active) are active against both rapidly growing and resting cells 2.6
9. Explain the mechanisms of the following inherent resistances to antimicrobial agents:
   a. mycoplasma resistance to cell wall active antibiotics 3.0
   b. anaerobe resistance to aminoglycosides 1.1
   c. aerobic resistance to metronidazole 2.6
   d. gram-negative resistance to vancomycin 2.6
10. Explain tolerance to beta-lactam antibiotics 2.2

H. PHYSICAL AND CHEMICAL AGENTS FOR CONTROL OF MICROBIAL GROWTH

1. Define:
   a. antiseptic 3.0
   b. aseptic 3.0
   c. bactericidal 3.0
   d. bacteriostatic 3.0
   e. disinfectant 3.0
   f. germicide 2.2
   g. sepsis 3.0
   h. sterilization 3.0
   i. pyrogen-free 2.9

2. Describe the general effects chemical and physical agents have on membranes, proteins, and nucleic
Acids which are lethal to cells
3. Describe the major effect moist heat has on cells
4. Discuss the pros and cons of using boiling, pasteurization, and autoclaving to control microbial growth
5. Discuss the use of filtration for sterilization
6. Describe the effects of ionizing and nonionizing radiation on microbes
7. Give examples of each type of radiation and its use
8. Define a thymine dimer
9. Explain the difference between cationic and anionic detergents (list examples)
10. Explain the effects surfactants have on bacteria
11. Explain the mechanism of action and uses of the following in controlling microbial growth:
   a. quaternary ammonium compounds
   b. phenol and derivatives of phenol
   c. alcohols
   d. heavy metals
   e. halogens
   f. iodine
   g. tincture of iodine
   h. iodophor
   i. chlorine and its various forms
   j. hydrogen peroxide
   k. alkylating agents
   l. formaldehyde
   m. glutaraldehyde
   n. ethylene oxide
   o. beta-propiolactone
   p. mineral acids
   q. organic acids
   r. alkalis

I. HOST-PARASITE/PATHOGEN RELATIONSHIPS
1. Explain Koch’s Postulates
2. Explain how pathogenic microbes can evade the non-specific first-line defenses of the body
3. Describe the components of the non-specific second-line defenses of the body and their function as a barrier to disease
4. Compare and contrast true pathogens and opportunistic pathogens
5. Explain the difference between a toxigenic and an invasive pathogen
6. Compare and contrast exotoxins and endotoxins
7. Define superantigen
8. List several infectious diseases mediated by superantigens
9. Explain the biological activities of endotoxin and superantigens
10. Describe AB toxin structure
11. Explain the attributes of a microbe which can contribute to invasiveness
12. Explain the roles played in health and disease by the body’s normal flora
13. List the major normal flora microbes and where they are found
14. List the major normal flora microbes that are important opportunistic pathogens
15. Describe where they are normally found and the disease associations
16. Describe the major mechanisms of transmission of infectious diseases
17. Define the following:
   a. bacteremia
   b. carrier
   c. communicable disease
   d. endemic
   e. endotoxin
   f. enterotoxin
g. epidemic
h. exotoxin
i. fomite
j. infectious dose
k. latent infection
l. nosocomial infection
m. opportunistic pathogen
n. pandemic
o. pathogenicity
p. pyemia
q. pyogenic
r. pyrogenic
s. septicemia
t. subclinical infection
u. superinfection
v. systemic
w. toxoid
x. virulence
y. zoonosis

18. Discuss proper specimen collection from various anatomical sites.

DIVISION II: BASIC MYCOLOGY

A. PRINCIPLES

1. Compare the structure of fungal cells to other eukaryotic cells and to bacteria
2. Compare and contrast yeasts, molds, and dimorphic fungi
3. Describe the basis for fungal taxonomy
4. List the major attributes of the following:
   a. Deuteromycetes (fungi imperfecti)
   b. Zygomycetes
   c. Ascomycetes
   d. Archiascomycetes (Pneumocystis)
   e. Basidiomycetes
5. Define:
   a. hyphae
   b. septate
   c. nonseptate
   d. pseudohyphae
   e. mycelium
   f. rhizoids
   g. zygospores
   h. ascospores
   i. ascus
   j. basidiospores
   k. conidia
   l. arthroconidia
   m. chlamydoconidia
   n. blastospores
   o. sporangiospores
   p. macroconidia
   q. microconidia
   r. dimorphism

B. FUNGAL CLASSIFICATION

1. Describe the classification of human mycoses:
a. superficial (e.g., Malassezia furfur, etc.)
b. cutaneous (e.g., microsporum, trichophyton, etc.)
c. subcutaneous (e.g., Sporothrix schenckii, etc.)
d. systemic/endemic (e.g., Histoplasma, Coccidioides, etc.)
e. opportunistic (e.g., Aspergillus, Candida, etc.)

2. Describe the laboratory identification of fungi

3. Define:
   a. KOH preparation
   b. Sabouraud’s agar

C. ANTIFUNGAL AGENTS

1. Describe the mechanism of action and clinical use of:
   a. nystatin
   b. amphotericin b
   c. itraconazole
   d. voriconazole
   e. fluconazole
   f. ketoconazole
   g. butoconazole
   h. clotrimazole
   i. miconazole
   j. sertaconazole
   k. econazole
   l. tioconazole
   m. terconazole
   n. caspofungin
   o. anidulafungin
   p. micafungin
   q. terbinafine
   r. naftifine
   s. flucytosine
   t. griseofulvin
   u. tolnaftate
   v. potassium iodide

DIVISION III: BASIC PARASITOLOGY

A. PRINCIPLES

1. Define:
   a. cyst
   b. trophozoite
   c. oocyst
   d. schizogony
   e. vector
   f. intermediate host
   g. definitive host

B. CLASSIFICATION

1. Describe parasite classification
   a. protozoa
      (1) rhizopods (entamoeba, etc.)
      (2) ciliates
      (3) flagellates (trypanosoma, leishmania, etc.)
      (4) sporozoa (plasmodium, toxoplasma, etc.)
   b. helminths
(1) nematodes (ascaris, enterobius, etc.)
(2) cestodes (taenia, echinococcus, etc.)
(3) trematodes (schistosoma, clonorchis, etc.)

<table>
<thead>
<tr>
<th>DIVISION IV: BASIC VIROLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Compare a virus to a cell</td>
</tr>
<tr>
<td>2. Discuss/define the following features of viruses:</td>
</tr>
<tr>
<td>a. size</td>
</tr>
<tr>
<td>b. shape</td>
</tr>
<tr>
<td>c. nucleic acid</td>
</tr>
<tr>
<td>d. capsid</td>
</tr>
<tr>
<td>e. capsomere</td>
</tr>
<tr>
<td>f. nucleocapsid</td>
</tr>
<tr>
<td>g. capsid symmetry</td>
</tr>
<tr>
<td>h. icosahedral</td>
</tr>
<tr>
<td>i. helical</td>
</tr>
<tr>
<td>j. envelope</td>
</tr>
<tr>
<td>k. peplomer</td>
</tr>
<tr>
<td>3. Describe virus classification</td>
</tr>
<tr>
<td>a. list the DNA virus families, including the following features</td>
</tr>
<tr>
<td>(1) enveloped or naked</td>
</tr>
<tr>
<td>(2) DNA structure (DS, SS, linear, circular)</td>
</tr>
<tr>
<td>(3) replication site (cytoplasm, nucleus)</td>
</tr>
<tr>
<td>(4) list medically important examples from each family</td>
</tr>
<tr>
<td>b. list the RNA virus families, including the following features</td>
</tr>
<tr>
<td>(1) enveloped or naked</td>
</tr>
<tr>
<td>(2) RNA structure (DS, SS, linear, circular)</td>
</tr>
<tr>
<td>(3) positive-, negative-sense</td>
</tr>
<tr>
<td>(4) capsid symmetry</td>
</tr>
<tr>
<td>(5) replication site (cytoplasm, nucleus)</td>
</tr>
<tr>
<td>(6) list medically important examples from each family</td>
</tr>
<tr>
<td>4. Describe the following agents, including their replication cycle</td>
</tr>
<tr>
<td>a. defective virus</td>
</tr>
<tr>
<td>b. pseudovirion</td>
</tr>
<tr>
<td>c. viroid</td>
</tr>
<tr>
<td>d. prion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B. VIRUS MULTIPLICATION AND INFECTIVITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Describe virus multiplication, including</td>
</tr>
<tr>
<td>a. adsorption</td>
</tr>
<tr>
<td>b. hemagglutinin</td>
</tr>
<tr>
<td>c. entry</td>
</tr>
<tr>
<td>d. naked viruses</td>
</tr>
<tr>
<td>e. enveloped viruses</td>
</tr>
<tr>
<td>f. uncoating</td>
</tr>
<tr>
<td>g. site</td>
</tr>
<tr>
<td>(1) cytoplasm</td>
</tr>
<tr>
<td>(a) RNA viruses</td>
</tr>
<tr>
<td>= (+) ssRNA</td>
</tr>
<tr>
<td>= (-) ssRNA</td>
</tr>
<tr>
<td>= dsRNA</td>
</tr>
<tr>
<td>(2) nucleus</td>
</tr>
<tr>
<td>(a) RNA viruses</td>
</tr>
</tbody>
</table>
1. Retroviruses
   - Influenza virus
   (b) DNA viruses
   h. role of reverse transcriptase
   i. viral protein synthesis
   j. assembly
   (1) lysis
   (2) budding
   k. release
   l. virus load

2. Discuss the infectivity of naked virus:
   a. DNA
   b. RNA

3. Define
   a. conditional mutants
   b. recombination
   c. reassortment
   d. complementation
   e. phenotypic mixing

4. Describe
   a. antigenic drift
   b. antigenic shift

5. Describe how viruses are cultivated in the laboratory
   a. Describe and/or define the following
      (1) cell culture
      (2) one-step growth experiment
      (3) cytopathic effect
      (4) syncytia
      (5) plaque
      (6) hemagglutination assay

6. Describe the use of viruses in gene therapy

7. Explain and give examples of the following viral vaccines
   a. live
   b. inactivated
   c. recombinant

8. Discuss and/or define
   a. malignant transformation
   b. oncogene

C. ANTIVIRAL AGENTS
1. Describe the mechanism of action and clinical use of
   a. acyclovir 3.0
   b. famciclovir 1.8
   c. valacyclovir 1.8
   d. penciclovir 1.8
   e. docosanol 1.2
   f. trifluridine 1.2
   g. ganciclovir 3.0
   h. valganciclovir 1.0
   i. foscarnet 2.0
   j. cidofovir 1.5
   k. fomivirsen 1.0
   l. amantadine 2.7
   m. rimantadine 2.6
   n. oseltamivir 3.0
   o. zanamivir 2.2
   p. ribavirin 3.0
   q. adeovir dipivoxil 1.8
   r. entecavir 1.3
   s. imiquimod 1.5
   t. interferon alpha 3.0
   u. HAART 3.0
   v. nucleoside reverse transcriptase inhibitors 3.0
      (1) abacavir 3.0
      (2) didanosine 3.0
      (3) emtricitabine 3.0
      (4) lamivudine 3.0
      (5) stavudine 3.0
      (6) zalcitabine 3.0
      (7) zidovudine 3.0
   w. nucleotide reverse transcriptase inhibitors 3.0
      (1) tenofovir 3.0
   x. nonnucleoside reverse transcriptase inhibitors 3.0
      (1) delavirdine 3.0
      (2) efavirenz 3.0
      (3) nevirapine 3.0
      (4) etraverine 3.0
   y. protease inhibitors 3.0
      (1) amprenavir 3.0
      (2) atazanavir 3.0
      (3) fosamprenavir 3.0
      (4) indinavir 3.0
      (5) lopinavir 3.0
      (6) nelfinavir 3.0
      (7) ritonavir 3.0
      (8) saquinavir 3.0
      (9) tipranavir 3.0
   z. fusion inhibitors 2.3
      (1) enfuvirtide 2.3
   aa. integrase inhibitors 3.0

*Numbers represent a scale of ‘3’ for essential knowledge that was essential for inclusion in the curriculum, ‘2’ for important knowledge that should be included in the curriculum if time is available, and ‘1’ for information that was deemed trivial and therefore not required in the curriculum.
## DIVISION I: BASIC CONCEPTS IN IMMUNOLOGY

<table>
<thead>
<tr>
<th>Table 2. Immunology/Host Defense Core Knowledge Objectives</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. GENERAL PRINCIPLES</strong></td>
</tr>
<tr>
<td>1. Describe in overall terms what the host defense system is, why we need it, what it does and how it does it</td>
</tr>
<tr>
<td>2. Explain the difference between self and non-self</td>
</tr>
<tr>
<td>3. Describe characteristics of active versus passive immunity</td>
</tr>
<tr>
<td>4. Compare and contrast innate and adaptive immunity</td>
</tr>
<tr>
<td>5. Be familiar with the cells of the innate immune response – neutrophils, macrophages and NK cells – know their general function in terms of recognition of microbes, production of cytokines and destruction of microbes</td>
</tr>
<tr>
<td>6. List several examples of physical barriers to infection (e.g., skin, mucous, etc.)</td>
</tr>
<tr>
<td>7. List examples of physiological barriers to infection (temperature, pH, etc.)</td>
</tr>
<tr>
<td>8. Describe the role that complement plays in innate immunity</td>
</tr>
<tr>
<td>9. Describe the phagocytic barrier to infection</td>
</tr>
<tr>
<td>10. Understand the concept of innate pattern recognition of microbes by phagocytic cells</td>
</tr>
<tr>
<td>11. Describe in overall terms the major components of the inflammatory response</td>
</tr>
<tr>
<td>12. Know the local and systemic effects of the innate immune response as they relate to TNF-alpha, IL-1 and IL-6</td>
</tr>
<tr>
<td>13. Understand the transition from innate to adaptive immunity</td>
</tr>
<tr>
<td>14. Describe the essential characteristics of humoral and cell-mediated immunity</td>
</tr>
<tr>
<td>15. List the features of the adaptive immune response – specificity, diversity, specialization, self limitation, memory</td>
</tr>
<tr>
<td>16. Describe ‘generation of diversity’ in the immune system</td>
</tr>
<tr>
<td>17. Explain the essential role of gene families in the evolution of antigen recognition in the immune system</td>
</tr>
<tr>
<td>18. Describe the theory of clonal selection</td>
</tr>
<tr>
<td>19. Describe the cells involved in the adaptive immune response – T cells, B cells and antigen presenting cells</td>
</tr>
<tr>
<td>20. List the phases of the adaptive immune response – recognition, activation, effector, decline and memory</td>
</tr>
<tr>
<td>21. Describe the basic aspects of T and B cell activation and the role of antigen presenting cells in this process</td>
</tr>
<tr>
<td>22. Describe the basic effector function of T and B cells in an immune response</td>
</tr>
<tr>
<td>23. Describe the significance of immunology in medicine by:</td>
</tr>
<tr>
<td>(A) Listing several examples of disorders affecting the immune response</td>
</tr>
<tr>
<td>(B) Listing several benefits of immunology to medicine</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>B. DEVELOPMENT OF CELLS AND FUNCTION OF ORGANS</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Describe in general terms the development of white blood cells from stem cells to progenitor cells to mature cells</td>
</tr>
<tr>
<td>2. Describe the different maturational stages of B and T cells (small lymphocyte, blast cell, plasma cell, etc.)</td>
</tr>
<tr>
<td>3. List the markers used to distinguish different lineages, subsets and maturational stages of lymphocytes. Be familiar with the functional significance and/or cellular distribution of the following: CD2, 3, 4, 5, 8, 11b, 14, 16, 19, 21, 23, 25, 40, 45, 56, TCR, BCR, B220</td>
</tr>
<tr>
<td>4. Describe the characteristics and functions of monocytes and macrophages (phagocytosis, antigen processing, etc.)</td>
</tr>
<tr>
<td>5. Describe the characteristics and functions of the granulocytic cells (neutrophils, eosinophils, basophils), mast cells, dendritic cells and natural killer cells</td>
</tr>
<tr>
<td>6. Describe the role of the bone marrow in lymphocyte origin</td>
</tr>
<tr>
<td>7. Describe the role of the thymus in maturation and selection of T lymphocytes</td>
</tr>
<tr>
<td>8. Recall the developmental pathway of T cells in the thymus</td>
</tr>
</tbody>
</table>
9. Recall that the thymus is the major site of selection and maturation of both helper T cells and CTLs  
10. Explain the processes of positive and negative selection in the thymus. Describe the process of 
programmed cell death (apoptosis) and its role in the thymus  
11. Describe the overall structure of the TCR (alpha/beta and gamma/delta) and associated polypeptides  
12. Describe the development of TCR and CD4/CD8 expression in maturing T cells  
13. Describe the sequence of events in the B cell developmental pathway from stem cell to immature B 
cell in the bone marrow  
14. Diagram the order of rearrangement and expression of Ig heavy chain and light chain genes during 
development of the B cell  
15. Describe the structure of the BCR and associated polypeptides  
16. Recall the antigen-independent and antigen-dependent phases in B cell ontogeny  
17. Recognize that tolerance is the antigen-induced, immunologically specific inactivation of 
lymphocytes  
18. Recall that two basic mechanisms exist to induce tolerance: (a) clonal deletion and (b) clonal anergy 
(or functional inactivation)  
19. Describe the major T cell effector populations in the periphery (helper, cytotoxic, regulatory)  
20. Describe the different T helper subpopulations and their role in controlling the immune response  
21. Describe the different peripheral B cell subpopulations (e.g. follicular, marginal, B1 vs. B2)  
22. Recall the role of the germinal center in B cell responses to antigen  
23. Describe the role of the lymphatic system in the transport of antigen and immune cells in the body  
24. Recall the distribution of lymph nodes in the body  
25. Describe the function of the secondary lymphoid organs in trapping and processing of antigens  
26. Recall the functions of different regions of the spleen and lymph nodes in the adaptive immune 
Response  
27. Recall the location and function of specialized lymphoid tissues such as the mucosal-associated-
lymphoid tissues (GALT, BALT, etc.)  
28. Recall the recirculation of lymphocytes and the role of adhesion molecules in lymphocyte trafficking

<table>
<thead>
<tr>
<th>C. INNATE IMMUNITY</th>
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</tr>
</thead>
<tbody>
<tr>
<td>1. Recall the role of physical barriers in innate immunity</td>
<td>3.0</td>
</tr>
<tr>
<td>2. Recall the role of physiological barriers in innate immunity</td>
<td>3.0</td>
</tr>
</tbody>
</table>
| 3. Discuss the role of the acute phase response and associated soluble effector proteins in the innate 
immune response | 2.0 |
| 4. Discuss the inflammatory process and its role in innate immunity | 2.0 |
| 5. Recall the key inflammatory cytokines and their local as well as systemic role in innate immunity | 3.0 |
| 6. Recall definition of complement | 3.0 |
| 7. Recall principles of complement nomenclature | 1.0 |
| 8. Describe basic components of complement system | 3.0 |
| 9. Describe the complement receptors, their expression pattern and their function | 2.0 |
| 10. Discuss the three main complement pathways – classical, lectin and alternative | 3.0 |
| 11. List the effector molecules of complement activation and their biologic function | 3.0 |
| 12. Describe the biological function of complement - chemotaxis, anaphylatoxins, clearance of immune 
complexes, B cell activation, opsonization, enhancement of phagocytosis, respiratory burst, and 
cytokine production | 3.0 |
| 13. Recall the role of complement in bacterial clearance and lysis | 3.0 |
| 14. Recall the use of plasma CH50 levels in the assessment of disease processes | 2.0 |
| 15. Recall the regulation of C3 convertase and MAC | 2.0 |
| 16. Describe various phagocytic cells in the body | 3.0 |
| 17. Define the functional role of cell adhesion molecules including selectins, integrins 
immunoglobulin superfamily members and accessory molecules | 3.0 |
| 18. Recall the role of endothelial cell activation in leukocyte recruitment | 2.0 |
| 19. List the chemotactic factors involved in the recruitment of various inflammatory cells | 3.0 |
| 20. Discuss the steps involved in phagocytic cell recruitment and migration into sites of inflammation – 
rolling, activation, tight adhesion and transendothelial migration | 3.0 |
### D. ANTIGENS AND ANTIBODIES

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<table>
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<tbody>
<tr>
<td>21.</td>
<td>Define the role of Toll Like Receptors in recognition of pathogen associated molecular patterns (PAMPS) and the activation of innate immune cells.</td>
</tr>
<tr>
<td>22.</td>
<td>Define the role of Fc receptors and complement receptors in opsonization, phagocytosis and activation of phagocytic cells.</td>
</tr>
<tr>
<td>23.</td>
<td>Describe the stages of phagocytosis – ligand binding, activation, engulfment, fusion of endosome with lysosome and bacterial destruction.</td>
</tr>
<tr>
<td>24.</td>
<td>Discuss the role of macrophages in antigen processing and presentation.</td>
</tr>
<tr>
<td>26.</td>
<td>Recall the pathways involved in reactive oxygen burst and the formation of reactive oxygen metabolites following tissue injury.</td>
</tr>
<tr>
<td>27.</td>
<td>Recall the role of antimicrobial peptides such as defensins or cathelicidins in innate immunity.</td>
</tr>
<tr>
<td>28.</td>
<td>Discuss the role of natural killer (NK) cells in mediating antiviral immunity and the role of activating and inhibitory receptors in the control of their function.</td>
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</tbody>
</table>

### D. ANTIGENS AND ANTIBODIES

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<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>1.</td>
<td>Compare and contrast antigenicity and immunogenicity.</td>
</tr>
<tr>
<td>2.</td>
<td>List the chemical classes of antigens.</td>
</tr>
<tr>
<td>3.</td>
<td>Define antigen, antigenic determinant, epitope and hapten and give examples of each.</td>
</tr>
<tr>
<td>4.</td>
<td>Describe the fundamental difference between B cell and T cell epitopes.</td>
</tr>
<tr>
<td>5.</td>
<td>Describe antigen-antibody interaction as a subset of receptor-ligand type interactions.</td>
</tr>
<tr>
<td>6.</td>
<td>Define affinity, avidity, and describe their role in immune processes.</td>
</tr>
<tr>
<td>7.</td>
<td>Describe the difference between soluble and insoluble immune complexes.</td>
</tr>
<tr>
<td>8.</td>
<td>Describe the basic structure of the immunoglobulin molecule.</td>
</tr>
<tr>
<td>9.</td>
<td>Recall the overall chain structure of the major classes and subclasses of immunoglobulins.</td>
</tr>
<tr>
<td>10.</td>
<td>Recall the different types and subtypes of light-chains.</td>
</tr>
<tr>
<td>11.</td>
<td>Explain the differences between isotype, allotype and idioype.</td>
</tr>
<tr>
<td>12.</td>
<td>Recall the overall structure of the major Ig fragments (e.g., Fab, Fc) and describe the enzymatic digestion used to obtain these fragments.</td>
</tr>
<tr>
<td>13.</td>
<td>List the major regions of the Ig molecule (e.g., hinge, variable hypervariable, etc.) and describe their overall structure and function.</td>
</tr>
<tr>
<td>14.</td>
<td>Diagram the basic domain structure of the Ig molecule and the essential features of the tertiary structure. Describe constant, variable and hypervariable regions with respect to antibody structure.</td>
</tr>
<tr>
<td>15.</td>
<td>Explain the differences between isotype, allotype and idioype.</td>
</tr>
<tr>
<td>16.</td>
<td>Recall the specialized functions of the human Ig isotypes. List examples of the specific role of different Ig isotypes in host defense, e.g. IgA may neutralize toxins in the gut.</td>
</tr>
<tr>
<td>17.</td>
<td>Describe the composition and function of secretory immunoglobulins.</td>
</tr>
<tr>
<td>18.</td>
<td>Diagram the process by which IgA crosses the epithelium and recall the role of the poly-Ig receptor in IgA secretion.</td>
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</tbody>
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### E. ANTIGEN RECEPTOR DIVERSITY

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<table>
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<tbody>
<tr>
<td>1.</td>
<td>Describe and explain the molecular genetic mechanisms involved in the generation of antibody diversity (e.g. multiple V region gene elements, variable recombination, junctional diversity etc.).</td>
</tr>
<tr>
<td>2.</td>
<td>Diagram the organization of the BCR heavy chain gene locus.</td>
</tr>
<tr>
<td>3.</td>
<td>Diagram the organization of the BCR light chain loci.</td>
</tr>
<tr>
<td>4.</td>
<td>Discuss variable gene recombination at BCR heavy and light chain loci.</td>
</tr>
<tr>
<td>5.</td>
<td>Describe reasons for junctional diversity during V gene recombination.</td>
</tr>
<tr>
<td>6.</td>
<td>Explain allelic exclusion with respect to immunoglobulin gene expression.</td>
</tr>
<tr>
<td>7.</td>
<td>Describe the genetic mechanism used to produce membrane-bound and secreted forms of Ig.</td>
</tr>
<tr>
<td>9.</td>
<td>Describe the mechanism used to regulate expression of IgD.</td>
</tr>
<tr>
<td>11.</td>
<td>Describe the overall structure of the TCR molecule.</td>
</tr>
<tr>
<td>12.</td>
<td>Differentiate between the two types of TCR (alpha/beta, gamma/delta) and distinguish their subsets.</td>
</tr>
<tr>
<td>13.</td>
<td>Recall the molecular genetic mechanisms used to generate diversity in the TCR.</td>
</tr>
</tbody>
</table>
14. Compare the gene organization of the TCR loci with that of the BCR loci.

**F. MHC, ANTGEN PROCESSING AND PRESENTATION**

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<table>
<thead>
<tr>
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<tbody>
<tr>
<td>1.</td>
<td>Describe the function of MHC molecules in antigen presentation and in cell-cell interactions in the immune system.</td>
<td>3.0</td>
</tr>
<tr>
<td>2.</td>
<td>Diagram the genetic organization of the HLA complex.</td>
<td>2.0</td>
</tr>
<tr>
<td>3.</td>
<td>Describe the three major classes of MHC gene products.</td>
<td>1.0</td>
</tr>
<tr>
<td>4.</td>
<td>List the major structural features of the MHC gene products (e.g., Class I molecules are two chains, a heavy chain and a beta-2 microglobulin chain.</td>
<td>3.0</td>
</tr>
<tr>
<td>5.</td>
<td>Identify the tissue distribution of class I and class II MHC.</td>
<td>3.0</td>
</tr>
<tr>
<td>6.</td>
<td>Recall several examples of MHC/disease correlations and provide a hypothesis to account for this correlation.</td>
<td>3.0</td>
</tr>
<tr>
<td>7.</td>
<td>Explain MHC polymorphism and the likely selective advantage of such a system.</td>
<td>2.0</td>
</tr>
<tr>
<td>8.</td>
<td>Describe the concept of MHC restriction and provide examples of the functional consequences of the “restriction” of T cell recognition.</td>
<td>3.0</td>
</tr>
<tr>
<td>9.</td>
<td>Define: haplotypes, genotypes, phenotypes, alleles, linkage disequilibrium.</td>
<td>2.0</td>
</tr>
<tr>
<td>10.</td>
<td>Diagram a proposed model of MHC-Ag-TCR interaction.</td>
<td>3.0</td>
</tr>
<tr>
<td>11.</td>
<td>Recall that T lymphocytes recognize antigen bound to MHC molecules and define aggretope versus epitope.</td>
<td>2.0</td>
</tr>
<tr>
<td>12.</td>
<td>Recall that T lymphocytes primarily recognize protein antigens and in fact recognize linear determinants as opposed to the conformational determinants recognized by B cells.</td>
<td>3.0</td>
</tr>
<tr>
<td>13.</td>
<td>Recall that T lymphocytes recognize antigen on the surface of other cells (antigen-presenting-cells (APC’s) or target cells).</td>
<td>3.0</td>
</tr>
<tr>
<td>14.</td>
<td>List several examples of APC’s (e.g. dendritic cells, macrophages, B cells, etc.).</td>
<td>3.0</td>
</tr>
<tr>
<td>15.</td>
<td>Describe the phenomenon of MHC-restricted antigen recognition and diagram this recognition process for both helper and cytotoxic T lymphocytes.</td>
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<tr>
<td>16.</td>
<td>Describe in overall terms the conversion of polypeptides to peptide antigens via antigen processing (e.g. the role of acidic intracellular compartments, proteases, etc.).</td>
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<tr>
<td>17.</td>
<td>Diagram the pathway of processing of an exogenous protein antigen.</td>
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<tr>
<td>18.</td>
<td>Diagram the pathway of processing of an endogenous protein antigen.</td>
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<tr>
<td>19.</td>
<td>Describe the selectivity of binding of peptide antigens to Class I and Class II MHC.</td>
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<tr>
<td>20.</td>
<td>Compare and contrast the presentation of exogenous and endogenous antigens to T lymphocytes.</td>
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</table>

**G. B AND T LYMPHOCYTE ACTIVATION**

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<tbody>
<tr>
<td>1.</td>
<td>Discuss the steps involved in lymphocyte activation – protein synthesis, proliferation, differentiation, homeostasis and memory cell formation.</td>
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<tr>
<td>2.</td>
<td>Describe the overall structure of the B cell antigen receptor.</td>
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<tr>
<td>3.</td>
<td>Discuss the fact that the B cell antigen receptor recognizes a wide range of antigens.</td>
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<td>4.</td>
<td>Describe the BCR complex including CD79a/b and the role of these associated polypeptides.</td>
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<tr>
<td>5.</td>
<td>Recall the functional significance of Immunoreceptor Tyrosine-based Activation Motifs (ITAMs).</td>
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<tr>
<td>6.</td>
<td>Describe the overall structure of the TCR molecule.</td>
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<td>7.</td>
<td>Describe the composition and function of the CD3 complex including the zeta:zeta homodimer.</td>
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<td>8.</td>
<td>Describe the activation of T cells, e.g. the interactions between APCs and T cells leading to T cell activation.</td>
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<tr>
<td>9.</td>
<td>List several examples of biochemical events triggered in T cells by antigen recognition.</td>
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<tr>
<td>10.</td>
<td>Discuss the functional role of the T cell accessory protein CD4 and CD8 in recognition of antigen and T cell activation.</td>
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<tr>
<td>11.</td>
<td>List examples of cell adhesion molecules, e.g. ICAM, LFA-1 and discuss their role in T cell activation.</td>
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<tr>
<td>12.</td>
<td>Diagram the mechanism of superantigen activation of T cells.</td>
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<tr>
<td>13.</td>
<td>Describe the mechanism of antigen induced B lymphocyte activation.</td>
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<tr>
<td>14.</td>
<td>List several examples of biochemical events triggered in B cells by antigen recognition.</td>
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</tr>
<tr>
<td>15.</td>
<td>Compare and contrast the effects of T-independent and T-dependent antigens on B cell activation.</td>
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</tbody>
</table>
16. List several examples of T independent antigens and describe the typical characteristics of such antigens (e.g., polyclonal activators, BcR cross-linkers).

17. Recall the role of B lymphocytes as antigen-presenting cells (APCs).

18. Describe the mechanism of TH-B cell collaboration and explain the observation known as the “hapten-carrier effect.”

19. Diagram the cell-cell interactions in a humoral immune response to a protein antigen (e.g., TH, cytokines, APCs interact with T cells, activated T cells interact with antigen-specific B cells, etc.).

20. Define the two-signal model of T cell activation and the role of costimulatory CD28 molecules.

21. Recall that most B cell responses require CD40-dependent signals in addition to that provided by antigen alone – discuss the functional role of CD40 signaling.

22. Recall that antigen-dependent signaling in the absence of co-stimulation leads to induction of anergy in T and B cells and what this means.

H. REGULATION OF THE IMMUNE RESPONSE

1. Lymphocyte Tolerance and Selection
   a. Describe the concept of central tolerance as it pertains to T cell development in the thymus.
   b. Define the roles played by mechanisms leading to apoptosis and anergy in the regulation of T cell development in the thymus.
   c. Discuss positive versus negative selection of thymocytes.
   d. Describe the concept of central tolerance as it pertains to B cell development in the bone marrow.
   e. Recall the role that receptor editing plays in B cell selection.
   f. Discuss the factors that control T and B cell selection and tolerance including avidity and affinity of interactions between the antigen receptor and antigen.
   g. Discuss the concept of peripheral tolerance induction for T and B cells.
   h. Recall the elimination of autoreactive B cells during the transitional phase in the spleen.
   i.Discuss the role of regulatory T cells in mediating peripheral tolerance.
   j. Discuss the function of tolerogenic dendritic cells in the periphery.

2. Regulation of Lymphocyte Activation Response
   a. Recall how the process of activation induced cell death relates to feedback control of T cell activation.
   b. Discuss the role of Fas and Fas ligand in mediating apoptosis of activated T and B cells.
   c. Understand the major pathways that lead to apoptosis in lymphocytes (i.e. the mitochondrial pathway and the death receptor pathway).
   d. Discuss the role of CTLA-4 in attenuation of T cell activation.
   e. Discuss the role of PD-1 and PD-1 ligand in regulating T cell activation.
   f. Discuss the role of Baff/BLyS and Apil and their receptors BAFF-R/BR3, TACI and BCMA in regulating B cell survival, activation and differentiation.
   g. Understand the process by which B cell co-receptors modulate antigen receptor signaling through the recruitment of effector proteins to ITIMs, and ITAMs.
   h. Understand the process of antibody-dependent feedback in negative regulation of B cells and the role played by the FcR-gamma-llb, ITIM.
   i. Discuss the role of CD19 in positively regulating activation of B cells.

3. Cytokines
   a. Be familiar with cytokine nomenclature and major classifications of cytokines.
   b. Recall basic functions of cytokines in cell-to-cell communication.
   c. Describe the basic properties of cytokines.
   d. Discuss the basic families of cytokine receptors.
   e. Describe the general functions of cytokines (IL-1, 2, 3, 4, 5, 6, 8, 10, 12, IFN, GM-CSF, G-CSF, M-CSF, TNF-alpha, TGF-beta).
   f. Compare and contrast the individual effect of several cytokines (including IL-1, TNF-alpha, IL-6 and interferons) on innate immunity.
   g. Recall the lipopolysaccharide (LPS)-induced cytokine cascade.
   h. List the cytokines involved in the acute phase response.
   i. Recall the role of several cytokines (including IFNs, Lymphotoxin, IL-5, IL-12) as regulators of
### I. CELL MEDIATED IMMUNITY

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<tbody>
<tr>
<td>1.</td>
<td>Recall the different populations of effector T cells and their activation requirements.</td>
</tr>
<tr>
<td>2.</td>
<td>Discuss the process whereby effector CTLs are generated from CTL precursors.</td>
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<tr>
<td>3.</td>
<td>Recall the process by which effector CTLs recognize target cells.</td>
</tr>
<tr>
<td>4.</td>
<td>Discuss the role of Fas and Fas ligand in CTL-mediated lysis of target cells.</td>
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<tr>
<td>5.</td>
<td>Diagram the process of CTL-mediated cell lysis, e.g. role of perforin.</td>
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<tr>
<td>6.</td>
<td>Discuss the role of NK cells in mediating lysis of virally infected target cells.</td>
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<tr>
<td>7.</td>
<td>Recall the mechanism by which NK cell activation is controlled (i.e. activating receptors versus inhibitory receptors).</td>
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<tr>
<td>8.</td>
<td>Describe the mechanisms used by NK cells to lyse target cells.</td>
</tr>
<tr>
<td>9.</td>
<td>Recall what antibody dependent cell-mediated cytotoxicity (ADCC) is.</td>
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<tr>
<td>10.</td>
<td>Recall cell-mediated immune responses induced by: NK responses, ADCC, LAK, DTH and give some clinical examples (e.g. contact sensitivity, intracellular infections, granulomas).</td>
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### DIVISION II: THE IMMUNE SYSTEM AND DISEASE

#### A. HYPERSENSITIVITIES, ALLERGY AND ASTHMA

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<tbody>
<tr>
<td>1.</td>
<td>List the Gell and Coomb's classification of hypersensitivity.</td>
</tr>
<tr>
<td>2.</td>
<td>Describe the pathophysiologic mechanisms associated with Type I (IgE)-mediated injury.</td>
</tr>
<tr>
<td>3.</td>
<td>Diagram the process of mast cell degranulation.</td>
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<tr>
<td>4.</td>
<td>List the primary effector mediators released by mast cells.</td>
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<tr>
<td>5.</td>
<td>Describe the pathologic changes in tissues during anaphylactic reactions – compare and contrast the acute phase reaction with the late phase reaction.</td>
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<td>6.</td>
<td>Explain the modulator role of eosinophils in allergic and anaphylactic reactions.</td>
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<tr>
<td>7.</td>
<td>Correlate the effect of mediators on target organs with clinical expression of allergic reactions.</td>
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<tr>
<td>8.</td>
<td>Discuss therapeutic modulation of type I hypersensitivity.</td>
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<tr>
<td>9.</td>
<td>Describe the clinical expression of anaphylactic reactions and diagnosis via skin tests, RAST, immunoassays, etc.</td>
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<tr>
<td>10.</td>
<td>Describe allergic asthma.</td>
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<tr>
<td>11.</td>
<td>List the bronchial wall changes that occur in asthma.</td>
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<tr>
<td>12.</td>
<td>Recall the treatment considerations of various forms of asthma.</td>
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<tr>
<td>13.</td>
<td>Define type II and type III reactions.</td>
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<tr>
<td>14.</td>
<td>Compare complement mediated cell lysis and antibody dependent cell cytotoxicity.</td>
</tr>
<tr>
<td>16.</td>
<td>Diagram the effects of antibodies on cell surface receptors.</td>
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<tr>
<td>17.</td>
<td>Recall drug-induced type I and II hypersensitivity.</td>
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<tr>
<td>18.</td>
<td>Recall erythroblastosis fetalis.</td>
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<tr>
<td>19.</td>
<td>Recall mechanism and histopathology of Arthus reaction.</td>
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<tr>
<td>20.</td>
<td>Describe type IV cell mediated hypersensitivities.</td>
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<tr>
<td>21.</td>
<td>Recall the basis for and examples of contact hypersensitivity.</td>
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<tr>
<td>22.</td>
<td>Discuss the tuberculin reaction.</td>
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<tr>
<td>23.</td>
<td>Describe the granulomatous reaction.</td>
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#### B. AUTOIMMUNITY

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<tbody>
<tr>
<td>1.</td>
<td>List autoimmune diseases associated with specific organs.</td>
</tr>
<tr>
<td>2.</td>
<td>List autoimmune diseases that are systemic in nature.</td>
</tr>
</tbody>
</table>
3. List several examples of autoimmune diseases mediated by autoantibodies, e.g. myasthenia gravis, Graves’ disease, Lupus, etc.
4. List several examples of autoimmune diseases mediated by T cells, e.g. EAE.
5. Discuss the role that gender, genetics, environment and infectious disease play in the development of autoimmunity.
6. Describe several mechanisms that help to explain anti-self responses (e.g. immunological cross-reaction or molecular mimicry).
7. Describe the role of MHC genes in autoimmunity.
8. Provide several hypotheses to account for the association of autoimmune diseases with MHC genes, e.g. “molecular mimicry”, etc.
9. Describe the basic types of therapeutic intervention used to treat autoimmune disease.

### C. TRANSPLANTATION IMMUNOLOGY

| 1. Discuss the immunologic basis of graft rejection. | 3.0 |
| 2. Recall the principle of first set and second set rejection. | 3.0 |
| 3. Understand the terms autograft, isograft, allograft and xenograft. | 3.0 |
| 4. Discuss the role of CD4 and CD8 T cells in graft rejection. | 3.0 |
| 5. Recall that the major molecular targets in graft rejection are the non-self MHC molecules. | 3.0 |
| 6. Describe the difference between major and minor MHC molecules. | 1.0 |
| 7. Recall the overall molecular structure of the MHC Class I and II molecules. | 2.0 |
| 8. Recall the overall genetic organization of the HLA complex. | 2.0 |
| 9. Recall the laws of transplantation. | 1.0 |
| 10. Describe hyperacute, acute and chronic rejection. | 3.0 |
| 11. Compare and contrast the immunological reactions occurring in the above types of host response to a foreign graft. | 3.0 |
| 12. Give examples of tests used to measure tissue histocompatibility. | 3.0 |
| 13. Recall areas of clinical organ transplantation. | 3.0 |
| 14. List several approaches to prolonging graft survival (e.g. immunosuppressive drugs, mAbs, immune modulators). | 3.0 |
| 15. Recall the mechanism of inhibition of T cell activation used by several drugs, e.g. cyclosporin A. | 3.0 |
| 16. Describe the special immunological complexities that can be associated with bone marrow transplantation (GVHD, etc.). | 3.0 |

### D. IMMUNODEFICIENCIES – CONGENITAL AND ACQUIRED

| 1. Define congenital versus acquired immunodeficiency. | 3.0 |
| 2. Recall the basic classification of congenital immunodeficiencies. | 1.0 |
| 3. Discuss the presentation and pathophysiology associated with severe combined immunodeficiencies; list specific examples of SCID. | 3.0 |
| 4. Describe the condition associated with DiGeorge Syndrome. | 3.0 |
| 5. Be familiar with B cell defects, including X-linked agammaglobulinemia, Hyper-IgM Syndrome, Common variable immunodeficiency and selective IgA deficiency. | 3.0 |
| 6. Be familiar with phagocytic defects, including chronic granulomatous disease, leukocyte adhesion deficiencies and Chediak-Higashi syndrome. | 2.0 |
| 7. Recall miscellaneous immunodeficiencies, including Wiscott-Aldrich syndrome, Ataxia-telangiectasia and IFN-gamma/IL-12 receptor deficiencies. | 2.0 |
| 8. Explain the effects of specific complement deficiencies on patients. | 2.0 |
| 9. Recall basic therapeutic approaches for treatment of SCID, B cell deficiencies and phagocytic cell deficiencies. | 2.0 |
| 10. List examples of acquired immunodeficiencies and their causes (e.g. AIDS, drug induced, radiation induced). | 3.0 |
| 11. Recall the immunological abnormalities associated with HIV infection. | 3.0 |
**E. TUMOR IMMUNOLOGY**

1. Describe the concept of immunosurveillance.  
2. Recall several examples of tumor antigens, e.g. TSTAs, oncogenic viral antigens, etc.  
3. Describe the roles of antibody, T cells, NK cells, macrophages, etc. in tumor immunity.  
4. Describe the involvement of MHC molecules in tumor immunity, e.g. the effect of virally induced low MHC expression.  
5. List several ways in which tumors evade immune recognition, e.g. antigen modulation.  
6. Describe several approaches to tumor immunotherapy, e.g. antibody-toxin conjugates, IL-2, etc.  
7. Recall the causes of lymphoproliferative disorders.  
8. Recall the different tumors of the immune system.

**F. IMMUNITY TO MICROBES AND VACCINES**

1. Recall the functional differences between the innate versus adaptive immune response.  
2. Discuss the regulation and function of Natural Killer cells in innate immunity.  
3. Describe the role of CD4+ and CD8+ T cells in the adaptive immune response to viral infection.  
4. Recall the role of CD4+ T cells in activation of macrophages.  
5. Discuss the activation and differentiation of CD8+ T cells into cytolytic T cells.  
6. Diagram the process of CTL-mediated cell lysis, e.g. role of perforin.  
7. Describe the immune response to extracellular bacterial infections.  
8. Discuss the immune response to intracellular bacterial infections.  
9. Describe delayed type hypersensitivity as it relates to host responses against intracellular bacteria.  
10. Recall the host immune response to parasitic infection.  
11. Discuss mechanisms of immune evasion.  
12. Recall different types of vaccines (inactivated, attenuated, recombinant vaccines, DNA vaccines).  
13. Recall active vs. passive immunity to microbes.  
14. Recall primary versus secondary immune responses to vaccines and microbes.  
15. Explain the mode of action of adjuvants and recall some examples of adjuvant materials and give examples.

**DISTRIBUTION III: APPLIED IMMUNOLOGY**

**A. IMMUNOTHERAPEUTICS**

1. Recall the use of monoclonal antibodies to modulate immune cell function or to remove specific immune cells from the body (e.g. anti-CD20 to delete B cells in lymphoma/leukemia or in certain autoimmune diseases).  
2. Discuss the use of immunosuppressive drugs for the treatment of autoimmune disease or to prevent transplant rejection.  
3. Discuss the use of bone marrow transplantation in the treatment of congenital immunodeficiencies, or cancer.  
4. Recall the use of IVIG in the treatment of autoimmune disease and congenital immunodeficiencies.  
5. Discuss the potential therapeutic roles of cytokines or antibodies specific for cytokines and/or their receptors in: Sepsis, Inflammatory Bowel Disease, Rheumatoid Arthritis and Graft-versus-Host Disease.

**B. IMMUNODIAGNOSTICS**

1. Describe a range of tests used in studying human disease that are based on the specificity of antibodies, e.g. ELISA, Western blotting, flow cytometry, immunofluorescence staining, RIA, etc.  
2. Describe in overall terms the general principles of each test, e.g. ELISA is based on bound antibody being detected by an enzyme-dependent color change reaction, etc.  
3. Give examples of how different tests are utilized, e.g. immunofluorescence microscopy to detect antigen in tissue sections.

*Numbers represent a value of ‘3’ for essential knowledge, ‘2’ for important knowledge and ‘1’ for information that was found to be trivial and not an absolute requirement for the curriculum; in this table numbers were rounded to the nearest whole integer.**

**Data not available**
### Table 3. Pathogenesis/Infectious Diseases Core Knowledge Objectives

<table>
<thead>
<tr>
<th>DIVISION I: ESSENTIAL CONCEPTS IN INFECTIOUS PATHOGENESIS</th>
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<tbody>
<tr>
<td><strong>A. ENCOUNTHER WITH PATHOGEN</strong></td>
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<tr>
<td><strong>1. Basic Principles</strong></td>
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<tr>
<td>a. Define, in detail, endogenous (i.e. normal flora) versus exogenous sources of infection.</td>
<td>2.8</td>
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<tr>
<td>b. Explain how normal flora on skin or mucosal membranes can cause disease when introduced into deeper tissues.</td>
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<tr>
<td>c. Explain how exogenous infections are a result of encounters with organisms in the environment. (e.g. food, water, air, inanimate objects, insect bites, other humans, animals, etc.).</td>
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<tr>
<td>d. List and discuss the following common mechanisms of microbial transmission.</td>
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<td>(1) Direct skin or mucosal contact</td>
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<td>(2) Inhalation</td>
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<tr>
<td>(3) Ingestion</td>
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<td>(4) Vertical transmission (congenital; mother to baby)</td>
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<tr>
<td>(5) Vector –born transmission</td>
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<td>e. Discuss how anatomical sites exposed to the environment serve as portals of microbial entry. (i.e. nose, mouth, respiratory tract, alimentary tract, female genital tract, urinary tract and anus).</td>
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<td>f. Discuss how entry may or may not involve the crossing of epithelial barrier (e.g. inhalation vs. the carrying of microorganisms into deeper tissues by macrophages, or insect bites).</td>
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<tr>
<td><strong>B. INVASION AND DISSEMINATION</strong></td>
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<tr>
<td><strong>1. Virulence Factors: Adhesins/Colonization Factors</strong></td>
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<tr>
<td>a. Explain the significance of microbial adhesion as a component of the establishment of an infection.</td>
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<tr>
<td>b. Explain which microbial surface structures can function as adhesins</td>
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<tr>
<td>c. Differentiate between bacterial fimbrial and afimbrial adhesins</td>
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<tr>
<td>d. Describe what structures act as adhesins for enveloped versus nonenveloped viruses.</td>
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<tr>
<td>e. Identify the host cell surface components that can act as receptors.</td>
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<tr>
<td>f. Discuss the function of neutralising antibodies in preventing microbial attachment.</td>
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<tr>
<td>g. Clarify how attachment helps microorganisms to remain at a particular location/evade innate defense mechanisms.</td>
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<tr>
<td>h. List some antimicrobial compounds and the targets that are used to interfere with attachment.</td>
<td>1.2</td>
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<tr>
<td><strong>2. Invasins</strong></td>
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<tr>
<td>a. Define the action of invasins.</td>
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<tr>
<td>b. Describe the factors responsible for invasiveness of Shigella.</td>
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<tr>
<td>c. Describe the role of secreted enzymes in invasiveness of bacteria.</td>
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<td><strong>3. Anti-phagocytic Mechanisms</strong></td>
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<tr>
<td>a. Describe the advantage of encapsulation for bacteria.</td>
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<tr>
<td>b. Name several organisms with anti-phagocytic capsules.</td>
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<tr>
<td><strong>4. Hemolysins, Cytolysins</strong></td>
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<tr>
<td>a. Define hemolysin and cytolysin and give an example of a toxin and its producing microorganism for each.</td>
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<tr>
<td>b. Explain the mechanisms of action for the pore-forming and phospholipase cytolysins.</td>
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<tr>
<td>c. Discuss the streptococcal hemolysins in terms of their mechanisms of action.</td>
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<tr>
<td>d. Explain how hemolysis patterns on blood agar can help with species differentiation and disease diagnosis.</td>
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<tr>
<td>e. List some hemolysins/cytolysins and their functions in terms of the damage seen with a particular infection.</td>
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<tr>
<td><strong>5. Intracellular vs. Extracellular Multiplication</strong></td>
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<tr>
<td>a. Discuss the advantages of intracellular growth from a microbial perspective.</td>
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<tr>
<td>b. Contrast mechanisms of bacterial entry into a phagocytic versus a non-phagocytic cell.</td>
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<tr>
<td>c. Identify bacteria that rearrange actin to enable their entry and identify the basic steps in the process.</td>
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<tr>
<td>d. Make a list of (a) Obligate intracellular bacteria and (b) Facultative intracellular bacteria</td>
<td>2.3</td>
</tr>
<tr>
<td>e. Fully characterize each of the following intracellular survival mechanisms, giving specific microbial</td>
<td>3.0</td>
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</tbody>
</table>
examples:
(1) Escape from phagolysosome
(2) Prevention of phagosome-lysosome fusion
(3) Evasion/neutralization of lysosomal contents
(4) Alteration of phagolysosomal environment
f. Describe the challenges facing an extracellular bacterium
2.2

g. List the adaptations/virulence factors utilized by extracellular bacteria to evade the host’s antimicrobial defenses.
2.0

h. Assess the significance of intracellular growth when selecting an appropriate antimicrobial agent.
2.0

i. Describe the significance of intracellular growth when selecting an appropriate antimicrobial agent.
2.0

6. Tissue Tropism
a. Explain the significance of tissue tropism in helping to understand microbial pathogenesis
3.0

b. List some bacterial, viral and fungal examples of microorganisms that are tropic for a particular tissue/cell type.
2.2

c. Identify the other factors, both host and microbial, that influence the colonization of a particular site by a microorganism.
2.2

d. Describe how the adhesin-receptor interaction determines the tissue tropism of a microorganism.
2.0

e. Hypothesize as to the significance of a microorganism being able to use more than one receptor-ligand combination.
1.0

C. OUTCOMES OF INFECTION
1. Colonization vs. Disease
a. Define symbiosis, commensalism and parasitism.
3.0

b. Describe the benefits to the host of colonization by microorganisms.
2.0

c. Describe several sources of exogenous infection.
3.0

d. Name several factors that predispose to the development of disease when host encounters a microorganism.
3.0

e. Mechanisms of host cell damage
3.0

f. Direct damage from the organism
3.0

2. Toxins
a. Explain the genetic control of bacterial toxin production.
2.3

b. Explain the difference between exotoxins and endotoxins.
3.0

c. What determines the cell to which an exotoxin binds?
3.0

d. Name several enterotoxins and describe their mechanism of action.
3.0

e. Name two clostridial neurotoxins and describe their mechanism of action.
3.0

f. Name two cytotoxins that exert their effects via inhibition of protein synthesis.
2.8

g. Describe the effects of 3 types of toxins produced by Staphylococcus aureus.
2.5

h. Describe the effect of streptococcal pyrogenic exotoxin.
2.0

i. Explain the mechanism of action of pertussis toxin.
2.0

j. Explain the mechanism of action of tracheal cytotoxin in whooping cough
2.0

k. Name the organisms that produce Shiga toxin and explain its damaging effects.
3.0

l. Describe the differences between apoptosis and necrosis.
2.0

m. Define the source of endotoxins.
2.8

n. Explain the pathogenesis of septic shock produced by endotoxins.
3.0

o. Explain the infectious pathogenesis of disseminated intravascular coagulation.
2.8

3. Invasion
a. What is the major mechanism of tissue damage of fungi?
2.4

b. What are the two morphologic growth patterns of fungi and which of them is advantageous for the organisms' invasion of host tissue?
3.0

c. What host cell surface molecule is a receptor for several bacteria and viruses?
2.2

d. Explain the process that occurs with bacterial invasion into host cells with the example of Shigella
2.3

e. What process of cell death may be triggered on bacterial invasion of host cells?
2.2

f. Describe the mechanism of amebic enteric disease
2.0

4. Viral Cytopathic Effect
a. Describe the process by which a virus enters the host cell and brings out cell death in a lytic infection.

b. Describe the changes in the host cell seen as a result of viral infection.

c. Explain occurrences in a virally infected cell that result in persistent or latent infection.

d. Describe the changes in a cell that is transformed by viral infection.

5. Damage from the Inflammatory Response

a. Which bacterial components are active in eliciting a host immune response?

b. Describe the elicitation of the cytokine response to microbial infection of the host.

c. Which type of immune response is involved in the development of lesions characteristic of *Mycobacterium tuberculosis*?

d. Differentiate between the immune responses in tuberculoid and lepromatous leprosy.

e. Describe the mechanism of damage to the host that may occur from virus-antibody immune complexes.

f. Describe the mechanism of damage to the host that may occur from the cell-mediated response to a virus.

g. Describe the relationship of the cellular immune response and leishmaniasis.

h. Explain the damage that may occur with autoimmune sequelae of an infection.

i. Name the four types of hypersensitivity reactions and give examples of their involvement in host damage in infections.

6. Mechanisms of Evasion of the Host Defenses

a. For each of the bacterial virulence factors listed, describe how the factor facilitates evasion of the host immune response (innate and/or adaptive):

<table>
<thead>
<tr>
<th>Factor Type</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polysaccharide capsule</td>
<td><em>Streptococcus pyogenes</em></td>
</tr>
<tr>
<td>Pili/fimbriae</td>
<td><em>Neisseria gonorrhoeae</em></td>
</tr>
<tr>
<td>IgA protease</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td>Leucocidin</td>
<td><em>Neisseria meningitidis</em></td>
</tr>
<tr>
<td>Coagulase</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td>Protein A</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td>M protein</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td>Lipoteichoic acid</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
</tbody>
</table>

b. For each of the bacterial virulence factors listed, give specific examples of medically-important bacteria that possess the factor:

<table>
<thead>
<tr>
<th>Factor Type</th>
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</thead>
<tbody>
<tr>
<td>Polysaccharide capsule</td>
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<tr>
<td>Lipoteichoic acid</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
</tbody>
</table>

c. List several medically-important bacteria that are able to survive intracellularly and extracellularly, and explain how their ability to invade and survive inside cells helps them evade the host immune response.

d. Describe 3 different mechanisms used by some bacteria to evade the degradative enzymes inside phagocytic cells (polymorphonuclear cells, macrophages, or monocytes) and survive intracellularly.

e. Explain how bacteria in a biofilm are often more resistant to host immune responses.

f. Explain how antigenic variation facilitates evasion of the host immune response by microbial pathogens, and how this affects host and therapeutic/prophylactic mechanisms to prevent re-infection.

g. Describe how antigenic variation in the microbial structures listed below contribute to the pathogenesis of the organism:

<table>
<thead>
<tr>
<th>Structure</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus pyogenes</em> M protein</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td><em>Neisseria gonorrhoeae</em> pilin protein</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td><em>Streptococcus pneumoniae</em> capsule</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td><em>Neisseria meningitidis</em> capsule</td>
<td><em>Streptococcus pneumoniae</em></td>
</tr>
</tbody>
</table>
(5) Salmonella O and H antigens
(6) Influenza virus hemagglutinin and neuraminidase
(7) Rhinovirus capsid protein
(8) HIV envelope proteins
(9) HCV envelope proteins

h. Explain how cytokine “decoy” receptors (or cytokine decoys) produced by some viruses enhance their virulence.
i. List several viruses that produce cytokine decoys and the host cytokines that are targeted.
j. Explain what “virokines” are and how they enhance the ability of some viruses to evade the host immune response.
k. Describe several mechanisms used by viruses to evade the anti-viral interferon response.
l. Explain how HIV- and CMV-mediated downregulation of MHC class I expression enhances their ability to evade the host immune response.
m. List several viruses that produce syncytia and how this mechanism of cell-to-cell spread enhances their ability to evade the host immune response.
n. Explain how HIV infection of T cells affects the host immune response to this virus and other infectious agents.
o. Explain what is meant by “immune privileged” sites in the body and list several viruses that exhibit a tropism for these sites.
p. Describe several mechanisms used by viruses to produce persistent infections.
q. Describe the mechanism by which herpesviruses produce a latent infection in their host and how this contributes to their ability to evade the host immune response.
r. Describe how “immune tolerance” is developed in neonates infected with hepatitis B virus, rubella virus, or CMV and the effects on the infant.
s. Compare and contrast the mechanisms of persistence for HBV and HIV.
t. Compare and contrast the mechanisms of persistence for HBV and HCV.
u. Explain why prion diseases do not induce a host immune response.
v. Explain how antigenic shift and antigenic drift contribute to the ability of influenza virus to evade the host immune response.
w. Explain how viral “quasispecies” are generated and how this contributes to the ability of some viruses to evade the host immune response.

7. Transmissability
a. Define/Describe and give examples of the following modes of transmission of infectious agents:
   (1) Person-to-person
   (2) Nosocomial/Hospital-acquired
   (3) Endogenous infection
   (4) Percutaneous/blood-associated
   (5) Fomites
   (6) Soil
   (7) Vertical transmission
   (8) Horizontal transmission
   (9) Aerosols
   (10) Food, water
   (11) Zoonotic
   (12) Sexual contact
   (13) Fecal-oral
b. Describe structural features of viruses that often affect their stability in the environment and mode of transmission.
c. List the major sites of entry for infectious agents into the body and the barriers they must overcome at these sites to survive.
d. Describe conditions that enhance the transmission of infectious agents from person-to-person via non-sexual modes.
e. Define “reservoir” and “vector” in the context of zoonoses.
f. Define self-limited vs. resolution of infection vs. chronic infection.
g. Describe the steps that occur in an acute, self-limiting infection with respect to the pathogen.
pathogenesis, and host immune response.
h. List several infectious agents that cause acute, self-limiting infections in healthy, immunocompetent hosts.
i. List several infectious agents that cause acute, self-limiting infections in healthy, immunocompetent hosts, but can cause persistent infections in immunocompromised/immuno-immature hosts.
j. Compare and contrast the major characteristics of a chronic viral infection vs. a latent viral infection.
k. List several infectious agents that can produce chronic infections.
l. Describe the roles of humoral vs. cell-mediated immune responses in mediating clearance of different types of viruses.
m. Explain what is meant by the term “chronic carrier” and list examples of infectious agents that can induce this state in human hosts.
n. List an example of a slow virus and explain how slow virus infections are defined.

D. TREATMENT AND PREVENTION

1. Pharmacotherapy – Antibacterial Agents
   a. Differentiate bacteriostatic and bactericidal anti-bacterial agents and give examples of each.
   b. Describe major classes of antibacterial agents based on their mechanisms of action.
   c. What is the most important structural feature of penicillins and cephalosporins?
   d. What is their mechanism of action?
   e. Describe the spectrum of activity of penicillins.
   f. What is the source of bacterial resistance to penicillin?
   g. Name several penicillins that have been developed to overcome this resistance and describe how that has been done.
   h. Name several penicillins (beta-lactam antibiotics) that have been developed to overcome this resistance
   i. Describe how that has been done.
   j. What has been done to develop extended spectrum penicillins?
   k. What is the major side effect of penicillins and why is their toxicity generally limited?
   l. Describe the spectrum of activity of the various generations of cephalosporins.
   m. What is the mechanism of action of vancomycin?
      (1) Against what organisms is it the antibiotic of choice?
      (2) Describe the toxicity of vancomycin.
   n. Describe the mechanism of action and spectrum of activity of daptomycin.
   o. Name antibacterial agents whose activity depends on inhibition of nucleotide synthesis.
   p. Describe the toxicities seen with sulfonamides.
   q. Explain the usage of the sulfamethoxazole-trimethoprim combination.
   r. Name the group of antibacterial agents active through inhibition of DNA synthesis.
   s. What is the spectrum of activity and toxicities of the fluoroquinolones?
   t. Describe the mechanism of action, spectrum of activity, and toxicity of metronidazole.
   u. Name an antibacterial agent that acts through inhibition of RNA synthesis.
   v. In which infections is rifampin used?
   w. Name an antibacterial agent that acts in the early translation steps of bacterial protein synthesis.
   x. Describe the mechanism of action, spectrum of activity, and toxicities of aminoglycosides.
   y. Which antibacterial agent is especially useful against intracellular organisms?
   z. Describe the mechanism of action, spectrum of activity, and toxicity of the tetracyclines.
   aa. What are the adverse effects that have led to decreased usage of chloramphenicol?
   bb. Name the macrolide antibiotics and their specific usages.
   cc. At what step in bacterial protein synthesis are they active?
   dd. What is the reason for their numerous drug interactions?
   ee. What is the major adverse effect of clindamycin?

2. Pharmacotherapy – Antiviral Agents
   a. What steps in the process of viral pathogenesis are targets of antiviral agents?
   b. Name a group of natural antiviral compounds.
   c. Describe the clinical uses and antiviral effects of the Type I and Type II interferons.
d. Describe the mechanism of action and clinical usage and amantidone and rimantidone.

2.0

e. How does Pleconaril exert its antiviral effect on enteroviruses?

3.0

f. Name antiviral agents that act through inhibition of DNA polymerase.

3.0

g. Why are acyclovir and its related drugs relatively nontoxic?

3.0

h. Describe the mechanism of action of the reverse transcriptase inhibitors, their usages and side effects.

3.0

i. What is the role of protease inhibitors in antiretroviral therapy?

2.0

j. Name two neuraminidase blockers in current usage and show how they are useful.

3.0

k. Describe the mechanism of action of ribavirin and the areas of its usage.

3.0

l. How does foscarnet exert its antiviral effects?

2.0

m. Describe the mechanisms and usage of adefovir, tenofovir, and cidofivir.

1.0

3. Pharmacotherapy – Antifungal Agents

a. Name the targets of attack for current antifungal agents.

3.0

b. What antifungal drug has been used in most severe life-threatening fungal infections? Why is its usage currently decreasing?

3.0

c. What related drug is clinically useful only in Candida albicans infections of skin, mucous membranes, and GI infections?

3.0

d. Describe the mechanism of action, clinical uses, and side effects of the azoles and allylamines.

2.0

e. Name the class of antifungal agents that attacks fungi at the cell wall. How are they used.

2.0

f. Name the antifungal agent that is now used almost exclusively for dermatophyte infections of the hair and explain its mechanism of action.

3.0

4. Pharmacotherapy – Antiparasitic Agents

a. Explain why the choice of antiparasitic drugs is limited.

1.0

b. Describe the mechanism of action and usage of metronidazole.

3.0

c. What drug is used for asymptomatic amoebiasis?

2.0

d. Explain the usage of nitrofurantoin and allopurinol for trypanosomiasis.

1.0

e. How are the pentavalent antimonials effective in leishmaniasis?

1.0

f. Name the current recommended drugs for malaria and explain their mechanisms.

2.0

g. Explain the mechanism of sulfamethoxazole/trimethoprim in parasitic diseases.

2.0

h. Explain the mode of action of antibacterial agents in parasitic diseases.

2.0

i. Explain the usage of the benzimidazoles and ivermectin in helminth infections.

2.0

5. Vaccines

a. Explain the origin of the term “vaccination”.

1.5

b. Describe the types of vaccines and explain their differences in effectiveness.

3.0

c. Name 2 inactivated viral vaccines currently in use.

3.0

d. What attenuated bacterial vaccine is recommended throughout the world with the exception of the US and the Netherlands?

2.5

e. Name several live attenuated viral vaccines in current use.

3.0

f. Describe the advantages and disadvantages of the oral polio vaccine.

3.0

g. Name two bacterial polysaccharide vaccines and explain their disadvantages.

3.0

h. How has the immunogenicity of Hemophilus influenzae vaccine been enhanced?

2.5

i. Name two bacterial toxoid vaccines.

3.0

j. What type of vaccine is the current pertussis vaccine?

2.5

k. Give an example of a viral component vaccine in current use.

3.0

l. Explain the concept of recombinant vaccines.

2.0

DIVISION II: SYSTEMS-BASED DISEASES

A. UPPER RESPIRATORY TRACT INFECTIONS

1. Rhinitis

a. Define rhinitis

3.0

b. Name the two types of viruses that cause most cases of rhinitis

2.0

3.0

c. Identify the characteristics of each virus

2.0

d. Describe the attachment mechanisms of each virus

3.0

e. Describe the means by which the viruses are spread

1.0
f. Identify the major host defenses preventing infection by these viruses

g. Identify treatment recommended for rhinitis

h. What are important causes of rhinitis (rhinoviruses, coronaviruses)?

<table>
<thead>
<tr>
<th>2. Pharyngitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Define pharyngitis</td>
</tr>
<tr>
<td>b. Name the viruses that cause pharyngitis</td>
</tr>
<tr>
<td>c. Identify the characteristics of each of these viruses</td>
</tr>
<tr>
<td>d. Describe the means by which the viruses are spread</td>
</tr>
<tr>
<td>e. Identify sites other than the pharynx that may be associated with pharyngitis caused by some of these viruses</td>
</tr>
<tr>
<td>f. Describe treatment for viral pharyngitis</td>
</tr>
<tr>
<td>g. Name the most common cause of bacterial pharyngitis</td>
</tr>
<tr>
<td>h. Identify the virulence factors of this species</td>
</tr>
<tr>
<td>i. Describe the method of diagnosing bacterial pharyngitis</td>
</tr>
<tr>
<td>j. Describe the normal reservoir of this species</td>
</tr>
<tr>
<td>k. Identify the complications of infection by this species</td>
</tr>
<tr>
<td>l. Describe the events that lead to the complications</td>
</tr>
<tr>
<td>m. Identify the antibiotic(s) used to treat bacterial pharyngitis</td>
</tr>
<tr>
<td>n. List important causes of pharyngitis</td>
</tr>
</tbody>
</table>

1) Viral
   a) Rhinoviruses
   b) Adenoviruses
   c) Coronaviruses
   d) Epstein Barr virus

2) Bacterial
   a) *Streptococcus pyogenes*
   b) *Corynebacterium diphtheria*
   c) *Neisseria gonorrhoeae*

<table>
<thead>
<tr>
<th>3. Sinusitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Define sinusitis</td>
</tr>
<tr>
<td>b. Name the three major bacterial causes of sinusitis</td>
</tr>
<tr>
<td>c. Identify the characteristics of each of these bacteria</td>
</tr>
<tr>
<td>d. Identify the virulence factors of these bacteria</td>
</tr>
<tr>
<td>e. Describe the normal reservoir of the bacteria</td>
</tr>
<tr>
<td>f. Identify the major host defenses that protect against infection by these bacteria</td>
</tr>
<tr>
<td>g. Identify factors that predispose a patient to sinusitis</td>
</tr>
<tr>
<td>h. Identify the major complication of sinusitis</td>
</tr>
<tr>
<td>i. Identify the treatment recommended for sinusitis</td>
</tr>
<tr>
<td>j. Important causes of sinusitis</td>
</tr>
</tbody>
</table>

1) *Streptococcus pneumonia*

2) *Haemophilus influenzae*

3) *Moraxella catarrhalis*

<table>
<thead>
<tr>
<th>4. Otitis media</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Define otitis media</td>
</tr>
<tr>
<td>b. Name the three major bacterial causes of otitis media</td>
</tr>
<tr>
<td>c. Identify the characteristics of each of these bacteria</td>
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<td>f. Identify the major host defenses that protect against infection by these bacteria</td>
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<td>g. Identify factors that predispose a patient to otitis media</td>
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<td>i. Identify the treatment recommended for otitis media</td>
</tr>
<tr>
<td>j. Important causes of otitis media</td>
</tr>
</tbody>
</table>

1) *Streptococcus pneumonia*

2) *Haemophilus influenzae*
### B. LOWER RESPIRATORY TRACT INFECTIONS

#### 1. Bronchitis

<table>
<thead>
<tr>
<th>a. Define bronchitis</th>
<th>3.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>b. List the types of infectious agents that are involved in most cases of bronchitis</td>
<td>3.0</td>
</tr>
<tr>
<td>c. Identify the clinical presentation associated with each infectious agent</td>
<td>1.0</td>
</tr>
<tr>
<td>d. Identify the characteristics of each etiologic agent</td>
<td>2.0</td>
</tr>
<tr>
<td>e. Describe the attachment mechanisms of each etiologic agent</td>
<td>1.0</td>
</tr>
<tr>
<td>f. Describe the major virulence factors and mechanism of pathogenesis of each infectious agent</td>
<td>1.0</td>
</tr>
<tr>
<td>g. Describe the means by which the etiologic agents are spread</td>
<td>3.0</td>
</tr>
<tr>
<td>h. Identify the major host defenses preventing infection by these agents</td>
<td>2.0</td>
</tr>
<tr>
<td>i. Identify treatment recommended for bronchitis</td>
<td>1.0</td>
</tr>
</tbody>
</table>

#### 1.0 Important causes of bronchitis

1) **Bacterial**
   - a) *Bordetella pertussis*
   - b) *Mycoplasma pneumoniae*
   - c) *Chlamydia pneumoniae*

2) **Viral**
   - a) Influenza virus
   - b) Adenovirus
   - c) Respiratory syncytial virus (RSV)

#### 2. Bronchiolitis

<table>
<thead>
<tr>
<th>a. Define bronchiolitis</th>
<th>3.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>b. Name the viruses that cause bronchiolitis</td>
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</tr>
<tr>
<td>c. Identify the characteristics of each of these viruses</td>
<td>2.0</td>
</tr>
<tr>
<td>d. Describe the major virulence factor(s) and mechanism(s) of pathogenesis of each virus</td>
<td>1.0</td>
</tr>
<tr>
<td>e. Describe the means by which the viruses are spread</td>
<td>3.0</td>
</tr>
<tr>
<td>f. Describe treatment for viral pharyngitis</td>
<td>1.0</td>
</tr>
<tr>
<td>g. Name the most common cause of bacterial bronchiolitis</td>
<td>2.0</td>
</tr>
<tr>
<td>h. Identify the clinical presentation associated with each bacterium</td>
<td>1.0</td>
</tr>
<tr>
<td>i. Describe the method of diagnosing bacterial bronchiolitis</td>
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</tr>
<tr>
<td>j. Describe the major virulence factors and mechanism of pathogenesis of each infectious agent</td>
<td>1.0</td>
</tr>
<tr>
<td>k. Describe the normal reservoir of this species</td>
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</tr>
<tr>
<td>l. Identify the complications of infection by this species</td>
<td>3.0</td>
</tr>
<tr>
<td>m. Describe the events that lead to the complications</td>
<td>2.0</td>
</tr>
<tr>
<td>n. Identify the antibiotic(s) used to treat bacterial bronchiolitis</td>
<td>1.0</td>
</tr>
</tbody>
</table>

#### 2.0 Important causes of bronchiolitis

1) **Bacterial**
   - a) *Mycoplasma pneumoniae*
   - b) *Bordetella pertussis*

2) **Viral**
   - a) Respiratory syncytial virus

#### 3. Pneumonia

<table>
<thead>
<tr>
<th>a. Define pneumonia</th>
<th>3.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>b. Differentiate between chronic and acute pneumonia</td>
<td>1.5</td>
</tr>
<tr>
<td>c. Name the major etiologic agents of pneumonia</td>
<td>3.0</td>
</tr>
<tr>
<td>d. Describe the normal reservoir of these etiologic agents</td>
<td>3.0</td>
</tr>
<tr>
<td>e. Identify the clinical presentation associated with each infectious agent</td>
<td>2.5</td>
</tr>
<tr>
<td>f. List pneumonia agents suggested by environmental history</td>
<td>3.0</td>
</tr>
<tr>
<td>g. Discuss the differential diagnosis of cavitary lesion on chest radiograph</td>
<td>2.0</td>
</tr>
<tr>
<td>h. Identify the characteristics of each etiologic agent</td>
<td>2.5</td>
</tr>
<tr>
<td>i. Describe the attachment mechanisms of each etiologic agent</td>
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</tr>
<tr>
<td>j. Describe the major virulence factors and mechanism of pathogenesis of each infectious agent</td>
<td>2.5</td>
</tr>
</tbody>
</table>
k. Describe the means by which the etiologic agents are spread
l. Identify the major host defenses preventing infection by these agents
m. Identify factors that predispose a patient to pneumonia
n. Identify treatment recommended for pneumonia
o. Important causes of pneumonia

1) Bacterial
   (a) Streptococcus pneumoniae
   (b) Legionella pneumoniae
   (c) Mycoplasma pneumoniae
   (d) Mycobacterium tuberculosis
   (e) Bacillus anthracis
   (f) Chlamydia psittaci
   (g) Rickettsia
   (h) Coxiella burnetti
   (i) Klebsiella
   (j) Pseudomonas (COPD, cystic fibrosis)

2) Fungal
   (a) Histoplasma capsulatum
   (b) Coccidioides immitis
   (c) Pneumocystis jiroveci
   (d) Blastomyces dermatitidis

3) Viral
   (a) Respiratory syncytial virus
   (b) Influenza virus
   (c) Severe Acute Respiratory Syndrome Coronavirus
   (d) Human metapneumovirus
   (e) Bunyaviridae (Hantavirus pulmonary syndrome)
   a. Adenovirus

C. CARDIAC INFECTIONS

1. Endocarditis
   a. Name the organisms that commonly cause endocarditis.
   b. Explain the epidemiologic factors (exposure, portal of entry) underlying specific etiologies in
      particular patients (i.e., Strep or Staph are common causes due to repeated transient exposure from
      the normal flora of the patient, for instance transient viridans Strep viremia associated with
      brushing teeth or dental work; Candida and other infectious agents associated with prosthetic
      valves or injection drug users; etc.)
   c. Describe the "vegetative" lesions associated with endocarditis and explain how such lesions
      contribute to the diagnosis (persistently positive blood cultures, mass on valves by echocardiogram)
      and affect therapeutic options (choice of bacteriostatic versus bactericidal antibiotic therapy, etc.)
   d. Explain how laboratory procedures could distinguish between these various organisms.
   e. What clinical sample would be used, what lab procedures, which selective & differential media, and
      which biochemical assays would be necessary to distinguish between these pathogens?
   f. What are important virulence factors for these pathogens? How do these factors contribute to the
      virulence of the organisms?
   g. Important causes of endocarditis
      1) Streptococci
      2) Pneumococci
      3) Enterococci
      4) Staphylococci
      5) Gram (-) bacilli
      6) Candida
      7) Other microbes
a. Name the most common infectious cause of myocarditis.  
2.5
b. Describe the epidemiology and pathogenesis of coxsackievirus infections and explain why most coxsackievirus infections are subclinical.  
2.0
c. What is the protective acquired immune response that prevents disease in most people infected with this virus and how does the timing of this immune response correlate with symptomatic versus nonsymptomatic infection?  
1.0
d. Important causes of myocarditis  
1) Coxsackieviruses  
2) Many other infectious agents

<table>
<thead>
<tr>
<th>D. GASTROINTESTINAL INFECTIONS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Define diarrhea.</td>
<td>3.0</td>
</tr>
<tr>
<td>b. Differentiate gastroenteritis and enterocolitis.</td>
<td>3.0</td>
</tr>
<tr>
<td>c. Name the most common cause of diarrhea in infants.</td>
<td>3.0</td>
</tr>
<tr>
<td>d. Describe the clinical findings in acute gastroenteritis.</td>
<td>3.0</td>
</tr>
<tr>
<td>e. Differentiate an invasive infection vs. a toxin-mediated illness based on clinical findings.</td>
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<tr>
<td>f. Describe the two main modes for transmitting infectious agents that cause gastroenteritis and diarrhea.</td>
<td>3.0</td>
</tr>
<tr>
<td>g. Describe the pathogenesis of bacterial diarrhea.</td>
<td>2.5</td>
</tr>
<tr>
<td>h. Explain the mechanisms of damage from enterotoxins, cytotoxins, and invasive organisms.</td>
<td>2.5</td>
</tr>
<tr>
<td>i. Differentiate bacterial and viral causes of gastroenteritis based on clinical findings.</td>
<td>3.0</td>
</tr>
<tr>
<td>j. Describe the diagnostic techniques used to identify organisms causing gastroenteritis.</td>
<td>3.0</td>
</tr>
<tr>
<td>k. Describe the recommended treatment for gastroenteritis.</td>
<td></td>
</tr>
</tbody>
</table>
| l. Important causes of gastroenteritis  
1) Bacteria  
a) *E. coli*  
b) *Shigella sp.*  
c) *V. cholerae*  
d) *V. parahaemolyticus*  
e) *C. difficile*  
f) *Salmonella sp.*  
g) *Yersinia sp.*  
h) *C. perfringens*  
2) Viruses  
a) Norovirus  
b) Rotavirus  
3) Parasites  
a) *Entamoeba histolytica*  
b) *Giardia lamblia*  
c) *Cryptosporidium* |
|  |
| 2. Hepatitis |  |
| a. Define hepatitis. | 3.0 |
| b. Define jaundice. | 3.0 |
| c. Describe the symptoms and laboratory findings in hepatitis. | 3.0 |
| d. Describe the mechanism of liver damage in hepatitis. | 3.0 |
| e. Name the potential long-term sequelae of hepatitis. | 3.0 |
| f. Name several external factors that greatly accelerate microbe-induced liver damage. | 2.0 |
| g. What is the fatality rate of fulminant hepatitis? | 1.0 |
| h. For the following hepatotropic viruses describe the basic viral properties, principal routes of infection, global prevalence, potential to establish chronic infections, clinical symptoms, means of diagnosis including serologic markers, treatment options, and availability of vaccines: |  |
| 1) Hepatitis A Virus (HAV; Picornavirus) | 3.0 |
| 2) Hepatitis B Virus (HBV; Hepadnavirus, Pararetrovirus) | 3.0 |
3) Hepatitis C Virus (HCV; Flavivirus) 3.0
4) Hepatitis D Virus (HDV; Unclassified defective virus, needs HBV helper) 3.0
5) Hepatitis E Virus (HEV; Unclassified – Calicivirus-like) 3.0
6) Yellow Fever Virus (YFV; Flavivirus) 2.0
   i. Name several additional viruses that may target the liver. 2.0
   j. Name 2 spirochetes that may target the liver. 2.0
   k. Name 2 parasites that may target the liver

3. Other food/water-borne diseases 3.0
   a. Typhoid fever 3.0
   b. Campylobacter jejuni infection 2.5
   c. Botulism 2.5
   d. Infant botulism 3.0
   e. Staphylococcus aureus infection

4. Oral/oral diseases 3.0
   a. Helicobacter

E. GENITOURINARY INFECTIONS

1. Urinary Tract: Cystitis; Pyelonephritis 3.0
   a. Define cystitis and pyelonephritis 3.0
   b. Distinguish acute from chronic pyelonephritis 1.5
   c. List the most common causes of community-acquired v. nosocomial urinary tract infections (UTIs) 3.0
   d. Explain the routes of transmission of agents of UTIs 3.0
   e. Describe the primary virulence factors of bacterial agents of UTIs 2.0
   f. Identify the major host defenses that protect against infection by these bacteria 2.0
   g. Identify factors that predispose patients to UTIs 3.0
   h. Explain the prevalence of bacterial UTIs in females 3.0
   i. Describe diagnostic methods for bacterial UTIs 3.0
   j. Identify the treatment recommended for bacterial UTIs 3.0
   k. List viral and parasitic agents of UTIs 1.5

2. Common causes of urinary tract infections:
   a. Aerobic gram-negative rods, esp. 3.0
      (1) Uropathogenic Escherichia coli 3.0
      (2) Pseudomonas aeruginosa 3.0
      (3) Klebsiella 3.0
      (4) Proteus 3.0
      (5) Staphylococcus sp., esp. S. saprophyticus 3.0
      (6) Enterococcus sp. 2.0

3. Less common causes of urinary tract infections 2.0
   a. Adenovirus-hemorrhagic cystitis
   b. Schistosoma haematobium-schistosomiasis (blood in urine, associated with rural Africa) 1.0

F. GENITAL TRACT

1. Syphilis 3.0
   a. Describe structural and cultural characteristics of Treponema pallidum
   b. Describe the epidemiology and pathogenesis of syphilis, including primary, secondary and tertiary manifestations of the disease 3.0
   c. Define congenital syphilis and describe its manifestations and prevention 3.0
   d. Define neurosyphilis and describe its manifestations 3.0
   e. Describe the mode of transmission of the disease 2.5
   f. Describe methods for the diagnosis of syphilis 3.0
   g. Explain the difference between non-specific and specific serological tests for syphilis and the pattern of the immune response vis-à-vis these tests in treated and untreated cases 3.0
   h. Identify antibiotics of choice in treating syphilis 3.0

2. Gonorrhea
### Neisseria gonorrhoeae

- **a.** Describe structural and cultural characteristics of *Neisseria gonorrhoeae*  
- **b.** List the virulence factors associated with *Neisseria gonorrhoeae*  
- **c.** Describe modes of transmission of gonorrhea  
- **d.** Describe the diagnosis and treatment of gonorrhea  
- **e.** Distinguish between a diagnosis of gonococcal and non-gonococcal urethritis  
- **f.** Describe disseminated gonococcal infections and distinguish them from gonococcal infections of the eyes and throat.  
- **g.** Describe the mechanisms of acquired penicillin resistance and alternative drugs for treating resistant strains  
- **h.** Explain the importance of phase and antigenic variation in pathogenesis of *Neisseria gonorrhoeae*  
- **i.** Appreciate that *Neisseria gonorrhoeae* infections can lead to pelvic inflammatory disease in women

### Non-gonococcal Urethritis

- **a.** List the causative agents of non-gonococcal urethritis  
- **b.** Distinguish between a diagnosis of gonococcal and non-gonococcal urethritis  
- **c.** Describe the life cycle and unique properties of *Chlamydia trachomatis*  
- **d.** Describe structural and cultural characteristics of *Ureaplasma urealyticum*  
- **e.** Describe structural and cultural characteristics of *Mycoplasma genitalium*  
- **f.** Describe the diagnosis and treatment of non-gonococcal urethritis  
- **g.** Appreciate that these bacteria can also cause pelvic inflammatory disease in women  
- **h.** Describe the characteristics of *Lymphogranuloma venereum*  
- **i.** Describe the causative agent of *Lymphogranuloma venereum* (LGV) and *Chlamydia trachomatis*  
- **j.** Describe the clinical progress and symptoms of LGV  
- **k.** Explain the recent increase in LGV cases among travelers to Asia  
- **l.** Describe the diagnosis and treatment of LGV  
- **m.** Granuloma inguinale  
- **n.** Describe structural and cultural characteristics of *Klebsiella (Calymmatobacterium) granulomatis*  
- **o.** Describe the pathogenesis and symptoms of granuloma inguinale (GI)  
- **p.** Describe the diagnosis and treatment of GI  
- **q.** What is chancroid (soft chancre)  
- **r.** Describe structural and cultural characteristics of *Hemophilus ducreyi*  
- **s.** Describe the pathogenesis and symptoms of chancroid  
- **t.** Describe the diagnosis and treatment of chancroid  
- **u.** Appreciate how symptoms of chancroid can be confused with those of primary syphilis, LGV, GI, or genital herpes

### Trichomoniasis

- **a.** Describe characteristics of the protozoan *Trichomonas vaginalis*  
- **b.** Describe symptoms associated with trichomoniasis  
- **c.** Describe the diagnosis and treatment of trichomoniasis

### Bacterial Vaginosis

- **a.** List the four signs associated with non-specific vaginitis  
- **b.** Describe the organisms associated with bacterial vaginosis (BV)  
- **c.** Describe the diagnosis and treatment of BV

### Vulvovaginal Candidiasis

- **a.** Describe the structural and cultural characteristics of *Candida albicans*  
- **b.** Explain how candida can cause disease as a member of normal human flora  
- **c.** Describe the diagnosis and treatment of vulvovaginal candidiasis

### Genital Herpes

- **a.** Describe the virion and genome structure of herpes simplex type 2 (HSV-2)  
- **b.** Describe the transmission and pathogenesis of HSV-2 infections  
- **c.** Discuss the concept of viral latency/reactivity and its significance with respect to genital herpes infections  
- **d.** Describe the current strategies for preventing and treating HSV-2 infections

### Genital Warts

- **a.** Describe the virion and genome structure of human papillomavirus (HPV)  
- **b.** Describe the transmission and pathogenesis of HPV
c. Appreciate the association of cervical cancer with certain types of HPV infections 3.0
d. Describe methods for detection and treatment of HPV infections 3.0

9. Cytomegalic inclusion disease 2.0
   a. Describe the virion and structure of human cytomegalovirus (HCMV) 3.0
   b. Describe the epidemiology and pathogenesis of HCMV 3.0
   c. Appreciate that primary HCMV infection in a healthy individual is clinically inapparent but in adults can lead to a Mononucleosis syndrome 2.0
   d. Appreciate that HCMV causes the most common intrauterine viral infection and that cytomegalic inclusion disease in pregnant women can cause fetal death or damage to liver, spleen, blood-forming organs and nervous system 3.0

10. Other sexually transmitted diseases 3.0
   a. Appreciate that other organisms can be sexually transmitted without causing disease in the genital tract 2.0
   b. Describe the genome, pathogenesis and transmission of hepatitis b virus 3.0
   c. Describe the transmission and life cycle of the human immunodeficiency virus (HIV) 3.0

G. MUSCULOSKELETAL INFECTIONS 3.0

1. Myositis 1.0
   a. Name the most common infectious cause of myositis. 1.0
   b. How does coagulase help Staphylococcus aureus evade host immunity? 1.0
   c. What are other important virulence factors for this pathogen? 1.0
   d. How do these factors contribute to the virulence of the organism? 1.0
   e. Name an important cause of myositis is Staphylococcus aureus 1.0

2. Necrotizing Fasciitis 3.0
   a. Name the infectious causes of necrotizing fasciitis. 3.0
   b. Explain the pathogenesis of necrotizing fasciitis. 3.0
   c. How do the pathogens' virulence factors contribute to the pathogenesis? 3.0
   d. Why is surgery, even amputation, often necessary in the treatment of necrotizing fasciitis and antibiotic therapy alone inadequate? 3.0
   e. Name an important cause of necrotizing fasciitis is Streptococcus pyogenes (Group A Strep) 3.0

3. Osteomyelitis 2.5
   a. Name the most important infectious causes of osteomyelitis. 2.5
   b. What are important virulence factors for these pathogens? 2.5
   c. How do these factors contribute to the virulence of the organisms? 2.5
   d. Describe the routes by which various microbes gain access to bone (hematogenous spread, trauma-wound, prosthetic device, etc..) and be aware that lesions are often polymicrobial. 3.0
   e. Explain why surgical debridement and prolonged bactericidal antibiotic therapy are needed in chronic osteomyelitis. 2.5
   f. Explain how laboratory procedures could distinguish between these various bacteria. 2.5
   g. What procedures, media & biochemical assays would be necessary to distinguish between these pathogens? 2.5
   h. Important causes of osteomyelitis are:
      (1) Staphylococcus aureus (common) 3.0
      (2) Group B streptococcus (common) 2.5
      (3) Escherichia coli (common) 2.5
      (4) Pseudomonas aeruginosa 1.5
      (5) Enterococcus sp. 1.0
      (6) Salmonella sp. (in Sickle Cell disease patients) 3.0

4. Gas Gangrene 3.0
   a. Name the most important infectious cause of gas gangrene. 3.0
   b. How is Clostridium perfringens identified from gangrenous tissue and how is the disease diagnosed and treated? 3.0
   c. What are important virulence factors for c. Perfringens? How do these factors contribute to the virulence of the organism? 3.0
d. Reconcile why gas gangrene is so infrequent despite the presence of relatively large amounts of the organism in human intestines and pervasive presence in soil.

e. Explain why wounds are important in the pathogenesis of gas gangrene.

f. Name an important cause of gas gangrene (Clostridium perfringens)

5. Tetanus

a. Explain why wounds are typically necessary for tetanus.

b. How can tetanus be prevented?

c. Why is honey dangerous for infants?

d. What are important virulence factors for Clostridium tetani?

e. How do these factors contribute to the pathogenesis of tetanus?

6. Skin and Soft Tissue Infections

a. Define abscess, boil, carbuncle, furuncle, folliculitis, pyoderma (impetigo), erysipelas, cellulitis.

b. Define macule, papule, plaque, pustule, vesicle, bulla.

c. For the paired diseases and pathogens in the chart below:
   (1) Describe the clinical case setting in which the disease would be found.
   (2) Describe the microbial pathogens known to cause the disease.
   (3) Describe the pertinent microbial structures related to virulence (virulence factors, including toxins).
   (4) Describe the pertinent biochemical pathways related to pathogen virulence.
   (5) Describe the epidemiology of the disease.
   (6) Describe the etiology/pathogenesis of disease.
   (7) Describe clinical aspects of the disease.
   (8) Describe the immune response to pathogens causing the disease.
   (9) Describe methods of diagnosis of the disease.
   (10) Describe current therapy (and antibiotic resistance) for the disease.
   (11) Describe methods of prevention of the disease.

<table>
<thead>
<tr>
<th>PATHOGEN</th>
<th>DISEASE</th>
</tr>
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<tbody>
<tr>
<td>Staphylococcus aureus</td>
<td>Scalded skin syndrome, carbuncle, furuncle, folliculitis, impetigo, wound infection sol</td>
</tr>
<tr>
<td>Streptococcus pyogenes (Group a strep)</td>
<td>Impetigo, erysipelas, cellulitis, necrotizing fascitis, gas gangrene, scarlet fever, toxic shock syndrome</td>
</tr>
<tr>
<td>Clostridium perfringens</td>
<td>Gas gangrene</td>
</tr>
<tr>
<td>Clostridium tetani</td>
<td>Tetanus (local infection but systemic toxin)</td>
</tr>
<tr>
<td>Propionibacterium acnes</td>
<td>Acne</td>
</tr>
<tr>
<td>Mycobacterium leprae</td>
<td>Leprosy</td>
</tr>
<tr>
<td>Treponema pallidum</td>
<td>Syphilis</td>
</tr>
<tr>
<td>Treponema sp.</td>
<td>Yaws, pinta</td>
</tr>
<tr>
<td>Borrelia burgdorferi</td>
<td>Lyme disease rash</td>
</tr>
<tr>
<td>Rickettsia rickettsii</td>
<td>Rocky mountains spotted fever rash</td>
</tr>
<tr>
<td>Rickettsia prowazekii</td>
<td>Epidemic typhus</td>
</tr>
<tr>
<td>Rickettsia typhi</td>
<td>Endemic typhus</td>
</tr>
<tr>
<td>Erysipelothrix rhusiopathiae</td>
<td>Erysipeloid</td>
</tr>
<tr>
<td>Nocardia</td>
<td>Cutaneous nocardiosis</td>
</tr>
<tr>
<td>Superficial and cutaneous mycoses</td>
<td>Tinea versicolor</td>
</tr>
<tr>
<td>Malassezia furfur</td>
<td>Tinea versicolor</td>
</tr>
<tr>
<td>Microsporum, trichophyton, epidermophyton</td>
<td>Tinea corporis, tinea pedis, tinea cruris, tinea nigra, onychomycosis</td>
</tr>
</tbody>
</table>
Systemic mycoses with skin manifestations

- *Sporothrix schenckii* (3.0) - Sporotrichosis
- *Phialophora and Cladosporum* - Chromomycosis
- *Petriellidium and Madurella* - Mycetoma

Parasites

- *Leishmania tropica* - Cutaneous leishmaniasis (3.0)
- *Leishmania braziliensis* - Mucocutaneous leishmaniasis (1.0)
- Hookworms (Ancylostoma and Necator) - Cutaneous larva migrans (2.0)
- *Onchocerca volvulus* - Onchocerciasis (1.0)

Viruses/Disease presentation

- Papilloma viruses - Warts (3.0)
- Poxvirus – molluscum contagiosum - Fleshy papules (2.0)
- *Herpes simplex, coxsackievirus* - Vesicles (3.0)
- Measles, rubella, dengue, parvovirus B19 - Maculopapular rash (3.0)

Infections of the nervous system

**1. Principles**

a. Differentiate meningitis from encephalitis. (3.0)
b. Name the 2 organisms that cause 80% of cases of bacterial meningitis beyond the neonatal period. (3.0)
c. Name the common causes of bacterial meningitis in infants less than 1 month of age. (3.0)
d. Describe host factors that may increase the risk for bacterial meningitis. (2.0)
e. Describe the methods of acquisition of the organisms causing bacterial meningitis. (2.5)
f. Define aseptic meningitis. (2.5)
g. Describe the clinical signs and symptoms, pathogenesis, diagnostic techniques, and treatment options for bacterial, viral, and fungal meningitis and encephalitis. (3.0)

**2. Describe the Bacterial Causes of Meningitis/Encephalitis**

a. *Streptococcus pneumoniae* (3.0)
b. *Neisseria meningitidis* (3.0)
c. *Hemophilus influenzae* (2.0)
d. Group b strep (3.0)
e. *Escherichia coli* (2.0)
f. *Klebsiella sp.* (2.0)
g. *Listeria monocytogenes* (2.5)
h. *Mycobacterium tuberculosis* (2.0)
i. *Treponema pallidum* (2.0)

**3. Describe the Viral Causes of Meningitis/Encephalitis**

a. Enteroviruses (3.0)
b. Mumps (1.0)
c. Arboviruses (3.0)
d. Lymphocytic choriomeningitis (1.0)
e. Herpes viruses (3.0)
f. Influenza viruses  1.0
g. HIV  2.0
h. CMV  1.0
i. Rubella  1.0

4. Describe the Fungal Causes of Meningitis/Encephalitis
   a. *Candida albicans*  1.0
   b. *Cryptococcus neoformans*  2.0
   c. *Coccidioides immitis*  1.0
   d. *Histoplasma capsulatum*  2.0

5. Parasitic Causes of Meningitis/Encephalitis
   a. *Toxoplasma gondii*  2.0
   b. *Plasmodium falciparum*  1.0
   c. *Acanthamoeba* sp.  1.0
   d. *Naegleria fowleri*  1.0

I. DEGENERATIVE BRAIN DISEASES

1. Principles
   a. Describe the pathophysiology of subacute sclerosing panencephalitis and progressive multifocal leukoencephalopathy.  2.0
   b. Define prion disease.  2.5
      (1) Describe the course of Creutzfeldt-Jakob disease.  2.0
      (2) Describe the relationship of bovine spongiform encephalopathy to its associated human disease.  2.0

J. ZOONOTIC DISEASES

1. Describe the Etiologic Agents
   a. *Yersinia pestis* (PLAGUE)  2.5
   b. *Pasteurella multocida* cellulitis (cat/dog bite)  2.0
   c. *Francisella tularensis* (tularemia)  2.5
   d. *Bartonella* (cat scratch)  2.5
   e. *Brucella* sp.  1.5
   f. *Leptospira*  2.0
   g. *Ehrlichia*  2.0
   h. Anaplasmosis  1.5
   i. *Borrelia hermsii* (relapsing fever western U.S.)  2.0
   j. Rabies  3.0
   k. Viral hemorrhagic fever  1.0

K. OPPORTUNISTIC INFECTIONS

1. Describe the Etiologic Agents
   a. *Enterobacter*  1.5
   b. *Vibrio vulnificus*  2.0
   c. *Aeromonas* sp.  1.5
   d. *Hemophilus influenzae* (non-typeable)  2.0
   e. *Eikenella corrodens*  1.5
   f. *Pseudomonas aeruginosa*  3.0
   g. *Actinomyces*  2.0
   h. *Bacteroides*  2.5
   i. *Fusobacterium*  2.0
   j. *Prevotella*  2.0
   k. *Porphyromonas*  2.0
   l. *Peptostreptococcus*  2.0
*Represents values of ‘3’ for information that is essential knowledge to be included, ‘2’ for information that is important knowledge to be included if there is time in the curriculum and ‘1’ for information that is trivial knowledge not required in a curriculum on Pathogenesis/Infectious Diseases.