The Lymphatic System and Immunity
Study Guide for Immunity - C22B
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1. >What is a pathogen?

2. >How does your body defend itself against pathogens? What are the "three lines of defense"? Which are innate? Which are acquired?

3. What is the difference between nonspecific and specific defenses?

16. How does skin form a protective barrier against pathogens? List different mechanisms.

17. How does the mucosa form a protective barrier against pathogens? Explain different mechanisms.

18. Leukocytes are produced by pluripotent stem cells in the red bone marrow.
    a) List the individual cells from each category.
    b) Explain what their role is in fighting pathogens.
    c) >Which two cell lines use their lysosomes to create a "respiratory burst"?
    d) >What two chemicals, one which is common in many homes, are responsible for creating a killing zone by neutrophils and eosinophils?

19. Basophils are a formed elements of blood.
    a) >What do we call a basophile when they leave the blood and takes up residence in the connective tissue?
    b) What do these cells release?
    c) >How is the molecule released by mast cells that dialates arterioles in the systemic tissue and constricts airways in the respiratory tissue?
20. d) >What are the receptors on mast cells?

21. Monocytes are a formed elements of blood.
    a) >What do we call a monocyte that leaves the blood and "wanders" throughout the connective tissue?
    b) >What are the two primary functions of this cell in the connective tissue?
    c) How do these cells help the immune system?
    d) What other "names" are associated with this cell type when located in different organs?

22. In order to understand the physiology of immunity, you need to the function of the individual components:
    a. >What is interferons? What type of cell releases interferons? Does it "protect" the infected cell?
    b. What type of non-specific defensive cells are activated by interferons?

c. >What is complement? What organ makes complement? After complement is formed, where does it go? What happens to complement when it is activated?
d. >What are the four methods by which complement can destroy a phathogen?
e. What is the difference between antibody-dependent and anti-body independent complement activation?
f. Which complement mechanism requires RBC to transport pathogens to the liver?
g. Which complement mechanism requires the pathogen (foreign cell) to be "buttered up"? Why? What is this process called?
h. Which complement mechanism creates a "MAC"? What does a MAC molecule look like and where is it located? What happens to a pathogen when a MAC is attached to a plasma membrane?
i. >What is the significance of C3 in complement activation?

23. Immune surveillance is part of the non-specific defense mechanism. Natural Killer Cells (NK Cells) play a key role.
    a. >What type of infections are targeted by NKC?
    b. >How is the action of NKC characterized?
    c. How do NK cells kill cancer cells and foreign cells?
    d. How is perforins similar to complement?
    e. What does perforins require to complete the job?
    f. Is the "death" caused by NKC described as necrosis or apoptosis? (Explain)
    g. >What other cell uses this method to kill specific infected cells?

24. Inflammation is a local defensive response to tissue injury.
    a. >What type of injury can initiate inflammation?
    b. >What are the four cardinal signs of inflammation?
    c. >When you see "-itis", what should you assume?
    d. How are cytokines used in inflammation?
    e. Two cytokines, paracrine and autocrine, also influence the metabolism of target tissues. Explain how their action is both similar but different than endocrine secretions.

25. In many non-specific defensive mechanisms, the response is not diffused throughout the body. On the contrary, the mechanism are focused in the "local area" of tissue damage.
    a. >How does the body bring all the components of inflammation to the area of damage?
    b. What chemicals cause blood vessels to dilate? Which cells release these chemicals?
    c. What is an intercellular cleft? As this structure "widens", what moves from the blood into the interstitial space? Why?

27. Endothelial cells play an important role in inflammation. These simple squamous epithelial cells allow both cells and proteins in the blood to move into the interstitial space.
    a. What is the cell adhesion protein secreted by endothelial cells that attracts leukocytes?
    b. >What is the difference between margination, emigration, and diapedesis?

26. Inflammation is a response to local tissue damage. To minimize tissue damage, the response must keep the "cause" of the inflammatory response from spreading beyond the site of tissue damage.
    a. >How does the body "wall-off "the site of tissue damage?
    b. How does the body "trap" the bacteria and other pathogens within a "contained fluid pocket"?

27. Neutrophils (i.e. granulocytes) play a key role in immunity. These cells are "born" in the bone marrow, circulate in the blood, and have the ability to leave the blood and wander throughout the connective tissues of the body. They are a chief enemy of bacteria?
    a. What is chemotaxis? (Give two examples)
    b. >What are the two mechanisms used by neutrophils to destroy bacteria?
    c. >How long does it take for the first neutrophils to arrive at the site of tissue damage? Nickname?
    d. >Within a few hours, what happens to the number of neutrophils at the site of inflammation?

28. Neutrophils recruit macrophage to the site of tissue damage. Macrophage are prodigous phagocytes and help to "clean up the mess".
    a. >What type of chemical is secreted by neutrophils to signal macrophage to come into the area of tissue damage?
    b. How do macrophage help neutrophils in their struggle to win the war against bacteria?

29. After every "war" there is a mess to clean up. The aftermath of inflammation is no different.
    a. >Which agranulocyte is the "cleanup crew" and how long after an injury does it take for this cell to arrive?
    b. >How does edema contribute to tissue cleanup?
    c. >What is pus? What is the color of pus?
    d. When the puss becomes encapsulated, what do we call this structure?

30. Once the war is won you need to rebuild. Endothelial cells and platelets play a key role in the process of restoring the tissue to its original state (or simply replacing the original tissue with a connective tissue (i.e. Replacement vs Regeneration / Scar tissue). // What is the function of platelet derived growth factor?

31. Normal body temperature is 37 degrees Celsius. When your temperature rises above 37C, then we say that you have a fever.
    a. What is another term for fever?
    b. >What is a pyrogen?
    c. >Where do exogenous and endogenous pyrogens come from?
    d. What do we call medications that reduce fever?
    e. How do neutrophils and macrophage play a role in fever?
    f. >Where is the body's "thermostat" located?
    g. What are the six events associated with fever? (Be able to define onset, stadium, and defervescence).
    h. Why must you never give children younger than 15 aspirin to try to control a fever resulting from flue-like symptoms?

32. >How does the liver respond to fever? What is the benefit?

33. >What two characteristics distinguish immunity from nonspecific resistance?

34. Immunity was first described as early as 500 BC. In the 1893, Dr. William Coely infected patients with bacteria to shrink their tumors. In the mid-1900's it was discovered that there are two types of immunity: cellular immunity and humoral immunity.
    a. >What is humoral immunity effective against?
    b. Why do we say that humoral immunity only works against the "extracellular" form of the infection?
    c. >What is cellular immunity effective against?
    d. Can pathogens "hide" from cellular immunity?
    e. >Why do we say that cellular and humal immunity both target the same pathogen but in different ways?

35. >How are these terms used to classify immunity? (natural vs artificial vs active vs passive) List and explain how these four varibles create four catagories of immunity.

36. An antigen is anything that triggers an immune response.
    a. Are antigens large or small?
    b. >What do we mean by "self and non-self antigen"?
    c. >What is a epitope?
    d. >What is a hapten?

Lymphocytes and macrophage are the major cells of the immune system. We have already studied macrophage and natural killer cells (a type of lymphocytes). Now we need to study two lymphocytes called T-cells and B-cells.

37. The life cycle of T-Cells can be described as birth, education and deployment.
    a. >Where are T-cells born?
    b. >Where do T-cells go to be educated?
    c. >What do T-cells receive if they become educated?
    d. What does immunocompetent means
    e. >What does it mean for a T cell to be called "naive immunocompetent?
    f. What is the function of thymic reticular cells? ("test" T-cells to see if they are capable of becoming functional immunocompetent T-cells.)
    g. What is negative selection? What are the two forms?
    h. >What percent of the T-cells become functional immunocompetent T-cells?
    i. What is positive selection?
    j. Where do these cells go after they become immunocompetent T-cells?

38. B-cells are "born" in the red bone marrow but stay in the bone marrow where they become immunocompetent. Like T-cells, B cells need to be educated before they can be deployed.
    a. >What occurs when a B cell is "educated"?
    b. Where does naive immunocompetent mean?
    c. >Where do B cells go after they are deployed?

39. Cytotoxic-T-cells (C-Tc) are the only immune cell that can "specifically" kill an infected host cell. But C-Tc can not self-activate against an infected cell displaying a foreign antigen. They need helper-T cells activated against the same pathogen to secrete a cytokine to the C-Tc in order to complete the T-cell activation.
    a. >Can naive immunocompetent cytotoxic T-cells bind to infected host cells?
    b. >After the T cell binds to the host cell, can it immediately kill the host cell? l?
    c. >What must happen to complete the activaton of the cT-cell?
    d. >What is secreted by helperT-cells to initiate clonal selection? Outcome?

40. >What cells may function as antigen presenting cells (APC)?
    a. >What type of MHC proteins do they display?
    b. >What two APC may be used to activate other cells?
    c. >What APC secretes interleukin-1 to create a positive feedback loop?

41. Antigen presenting cells and host cells use major histocompatability proteins to display their cytoplasmic proteins on their plasma membranes. The displayed proteins maybe normal or indicate cancer or a viral infection.
    a. >How are MHCP produced and what do they do?
    b. >Explain the hot dog bun and hot dog metaphor?
    c. >Who eats the hot dog"?

There are three classes of T-cells (cytotoxic T-cells, helper T-cells, and memory T-cells). These cell lines work together to provide acquired cellular immunity. You need to understand the process in terms of three functional stages: ***recognize, react and remember****.*

42. You already know much about MHC proteins. Now you need to learn how the two types of MHC proteins (type I and type II) regulate immune function.
    a. >What type of cells have type I MHC proteins?
    b. >If the hot dog bun picks up a self antigen in the cytoplasm and presents it on the cell's plasma membrane, what happens?
    c. >If the hot dog bun picks up a virus protein or cancer protein and places it on the cell's plasma membrane, what immune cell will recognize this complex and what will happens?
    d. >What type of cells have type II MHC proteins?
    e. >What do MHC II proteins display on their cell's membrane?
    f. >What type of cell recognizes MHC II proteins?

43. >What is clonal selection? When does this occur?

44. Helper T-cells play a central role in the overall immune response. After a helper T-cell is activated by the antigen-MHC-II complex, it releases interleukin-2, a signal molecule.
    a. >What three events are triggered by the release of interleukin-2 by the helper T-cell?
    b. >If you lack helper T-cells, what happens to your immune system? What remains?

45. >What is the primary role of cytotoxic T-cells? What is a lethal hit?

46. >What are memory cells?
    a. >What type of cells make memory cells?
    b. >When are memory cells made?
    c. >How long do memory cells live?
    d. >Where are memory cells stored?
    e. >What role do memory cells play in a second exposure to a pathogen?

B-cells provide a more indirect method of defense than the T-cells. B-cells are going to "tag" the toxin, host cell, or foreign cell in such a way so that other defensive mechanisms can eliminate the problem. However, humoral immunity like cellular immunity takes place in three stages: recognition, attack and memory.

47. >What molecule on the B-cell's plasma membranes will allow B-cells to complete the "first step" in the identification of the foreign antigen?
    a. What is the role of receptor-mediated endocytosis?
    b. What is the role of MHC II protein?
    c. >What is the role of helper T-cells?

d. >After B cells are stimulated, what do B cells change into?
e. >What happens in clonal selection?
f. >What do we call the molecule made by plasma cells that attacks to the antigen?
g. >Do antibodies kill pathogens? Explain
h. How many of these molecules does one cell make in one second over what period of time?
i. >What happens if step one in B-cell activation occurs but no helper-T-cell secrete interlukin-2 to complete step two, then what happens?

48. >What are the five different classes of antibodies?
    a. >Where do antibodies carry out their function?
    b. >What antibody rapidly increases after a first exposure bacterial infection? As days pass, what antibody becomes the dominant circulating antibody?
    c. >After a second exposure to a bacteria, what antibody will spike on day one? Significance?
    d. >Which antibodies are used as the B-cell surface receptors?
    e. >Which antibody is the surface receptor on mast cells?
    f. >Which antibody can cross the placenta and provide natural passive immunity to a newborn?
    g. >Which antibody can be acquired in breast milk to provide the newborn natural passive immunity for the first 2 or 3 days of breast feeding?
    h. >What antibody is the surface receptor for mast cells?
    i. >What antibody is the surface receptor for RBC?
    j. What are the four mechanisms used by antibodies to render the antigen harmless?
    k. >What phrase describes the function of antibodies?

49. When your B-cells are first exposed to an antigen, it is called the primary response.
    a. >How long does it take before you see the first antibodies in your body fluids after the first exposure to a pathogen?
    b. >How long does it take to see antibodies in your body fluids after a second exposure?
    c. >What is the different types of antibodies seen during first and second exposure?

50. >What is a hypersensitivity?
    a. >What are the four different types of hypersensitivity?
    b. >What are the characteristics of the four different types of hypersensitivities?
    c. >What term is associated with a Type-1 hypersensitivity?
    d. >What is the difference between a Type-1 reaction if it is local vs systemic?