The Lymphatic System and Immunity
Study Guide for Immunity (C21B)

1. > What is a pathogen?

2. What "three lines of defenses" are used to protect us against pathogens?

3. > What does it mean in biolgoy to be an innate defense?

4. > What does it mean in biology to be an acquired defense?

5. Whic of the three line of defenses are innate and/or acquired? Significance?

16. How is skin a protective barrier against pathogens? Different types of protections?

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

18. Leukocytes (white blood cells) are produced by pluripotent stem cells in the red bone marrow.
    a) What are WBC's two cell types?
    b) How do WBC protect us from pathogens?
    c) What two WBC may produce a "respiratory burst" to kill pathogen?
    d) What common chemical found in many kitchens and made by two WBC types is used to kill pathogens?

19. Basophils are a formed elements of blood.
    a) What do we call basophiles after they immigrate the blood and attach themselves extracellular protein fibers in the interstitial space?
    b) What will cause these cells to "degranulate"?
    c) What are the two molecules released? Function of each?
    d) Which molecule dialate systemic arterioles but has opposite effect on bronchioles?

20. Where do mast cell receptors come from? Explain the significance between first exposure and second exposure.

21. Monocytes are a formed elements of blood.
    a) What do we call a monocyte after they leave the blood to "wanders" throughout the connective tissues of the interstitial spaces?
    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 they are located in different organs?

22. In order to understand the physiology of immunity, you need to know "the players" and then understand their function. This is a difficult subject because there are a lot of "players". Your challenge is to "put the puzzle together"!
    a. > What is interferon? What type of cell releases interferons? Does it "protect" the infected cell?
    b. What non-specific defensive cell type is activated by interferons?
    c. > What is complement? What organ makes complement's proteins? After complement is synthesized, where does it go? What happens to complement when it is activated?
    d. What four methods are used by complement to destroy phathogens?
    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 (the 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. Natural killer cells are formed elements, provide immune surveillance, and are part of the non-specific defense mechanism.
    a. > What type of pathogens are targeted by NKC?
    b. How do NK cells kill cancer cells and foreign cells? Hint: it's a kiss?
    c. How is perforins similar to complement?
    d. After perforins are released by NKC, what follows?
    e. Is the "death" caused by NKC described as necrosis or apoptosis? (Explain)
    f. What other cell uses this method to kill specific infected cells?

24. Inflammation is a response to tissue injury. The response is similar if it is caused by trama, chemicals, fire, or pathogens. (Note - Inflammation can be local or systemic.)
    a. > What type of injuries may 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 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?
    c. > Which cells release these chemicals?
    d. 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. Tissue secretions make endothelial cells "sticky" so neutrophils rolling along the inside of the blood vessels slow down. These same secretions make gaps between endothelial cells so neutrophils may enter the intertitial space.
    a. What is the cell adhesion protein secreted by endothelial cells that attracts leukocytes?
    b. > What is margination?
    c. > What is diapedesis?
    d. What is emigration?

26. Inflammation is a response to local tissue damage. To be successful, the response must keep the "cause" of the inflammatory response from spreading beyond the site of tissue damage.
    a. How is the site of the pathogen infection "walled-off "so it does not spread?
    b. How will the body "trap" the bacteria and other pathogens within a "contained fluid pocket"?

27. > Neutrophils are a type leukocyte known as a granulocyte. These cells are made 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. How may neutrophils kill bacteria when they are in the blood vessels?
    b. What is chemotaxis?
    c. How may neutrophils kill bacteria in the interstitial space?
    d. How long does it take for the first neutrophils to arrive at the site of tissue damage?
    e. Within hours after a bacterial infection, what will happen to the number of neutrophils in the blood? What is this called?

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" in this process?
    b. After the respiratory burst, how long before the cleanup crew arrives?
    c. How will edema contribute to the tissue cleanup process?
    d. What is pus? What is the color of pus?

30. Once the pathogen is defeated, the tisue will need to replace the dead cells. 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, eg. Replacement vs Regeneration / Scar tissue). // What is the function of platelet derived growth factor?

31. Normal body temperature is 37 degrees Celsius. If your temperature rises above 37C, then we call this condition a fever.
    a. What is another term for fever?
    b. > What is a pyrogen?
    c. > Where do exogenous pyrogens and endogenous pyrogens come from?
    d. What medication is used to reduce fever? Is this good or bad? Explain.
    e. What role do neutrophils and macrophage play 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. > When you have a fever, what happens in the liver? What is the benefit?

33. What characteristics distinguish immunity from nonspecific resistance?

34. Immunity was first "recognized" as early as 500 BC. In the 1890s William Coely saw cancer tumors shrink in patients with a baterial infection. In the mid-1900's, researchers discovered two types of immunity: cellular immunity and humoral immunity.
    a. Why would a bacterial infection cause a cancer tumur to shrink?
    b. > What is humoral immunity effective against?
    c. Why do we say that humoral immunity only works against the "extracellular" form of the infection?
    d. > What is cellular immunity effective against?
    e. Can pathogens "hide" from cellular immunity?
    f. > Why must we have both cellular and humal immunity to target the same pathogen using different mechanisms that target pathogens in different locations? Extremely important!

35. > How are these terms used to classify immunity? (natural vs artificial and 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 -antigen and non-self antigen"?
    c. > What is a epitope?
    d. > What is a hapten?

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

37. > Cytoxic T-cell (cT-cell) are responsible for cellular immunity. Before cT-cell are able to kill pathogen they must be "born, education and then deployed".
    a. Where are T-cells born?
    b. Where do T-cells go to become immunocompetent?
    c. What does it mean to be immunocompetent?
    d. What does it mean for a cT cell to be called "naive immunocompetent?
    e. What is the function of thymic reticular cells? (Ans: "test" T-cells to see if they are not going to kill good cells but can recognize foreign antigen)
    f. What percent of cT-cells become functional immunocopetent T-cells?
    g. What happens to cT-cells if they do not pass the "test"?
    h. What does it mean for a cT-cell to be deployed?
    i. After a cT-cell is deployed, where do they go? For how long?

38. > B-cell life cycle are responsible for humoral immunity Before B cells are able to render pathogens harmless and tag them for destruction they must be born, education, and then deployed.
    a. Where are B-cells born?
    b. Where are B cell is "educated"?
    c. What must happen to the B-cell to make them immunocompetent?
    d. What does it mean for a B-cell to be called naive immunocompetent?
    e. Do all the born B-cells become immunocompetent?
    f. What does it mean for a B-cell to be deployed? Where do B cells go after they are deployed? For how long?

39. Cytotoxic T-cells are the only immune cell that can specifically identify the pathogen inside a host cell and kill the infected cell along with the the pathogen inside the host cell. But cT-cells can not self-activate against an infected cell that displays the foreign antigen. If a naive immunocompetent cT-cell docks to a dendritic APC and a helper Tcell which was also activated by similar pathogen now secretes interleukin then the cT-cell becomes "activaed". Now the cT-cell functions as an assassin and may attach to infected host cells and give them the kiss of death.
    a. Can naive immunocompetent cytotoxic T-cells bind to infected host cells?

b. > What must a naive immuocompetent cT-cell bind to in order to start the process in order to become an assassin?
c. Will this by itself activate the cT-cell so it can kill other cells infected with same bacteria?
d. > What must happen to complete the activaton of this cT-cell?
e. What is secreted by helperT-cells to initiate clonal selection? Outcome?

40. Antigen presenting cells and host cells use major histocompatability proteins to display on their plasma membranes the different types of proteins present in their cytoplasm.
    a. What is a major histocompatability protein comlex?
    b. > Where are MHCP made?
    c. What do they pick up from the cytoplasm?
    d. > Where do MHCP display the protein they pick up from the cytoplasm?
    e. > If the MHC protein is the hot dog bun, then what is the hot dog?
    f. The epitope is what part of the bun-hot dog complex?
    g. What is going to eat (i.e. attach to) the "bun and hot dog complex"?

We are interested in knowing the function of the 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 these three different stages: ***recognize, react and remember****. These stages occur after both T cells and B cells are deployed!*

41. You know the general function of MHC proteins. Now you need to learn the function of MHC-I and MHC-II. Also, which cell types have and can recognize MHC-I and MHC-II.
    a. > What type of cells display proteins using MHC-I?
    b. > What cells have receptors able to bind to the MHC-I complex?
    c. If the hot dog bun picks up a self antigen in the cytoplasm and presents it on the cell's plasma membrane, what may happen?
    d. If the hot dog bun picks up a virus protein or cancer protein from the cell's cytoplasm and presents it on the cell's plasma membrane, then what immune cell may be able to recognize this complex? What will happens?
    e. > What cells have receptors able to bind to MHC-I proteins?
    f. > What cells have receptors able to bind to MHC-II proteins?
    g. What do MHC II proteins display on their cell's membrane?

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

43. 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 interleukins, a signal molecule.
    a. What three events are triggered by the release of interleukin by the helper T-cell?
    b. > If you do not have helper T-cells, can you active cT-cells and B cells?
    c. > If you do not have helper T-cells, can you initiate inflammation?

44. > What is the primary role of cytotoxic T-cells? What is the kiss of death?

45. What are memory T-cells? When are they formed? When are they to used?

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 so other defensive mechanisms can eliminate the problem. However, humoral immunity like cellular immunity takes place in three stages: recognition, attack and memory.

46. Naive immunocompetent B-cells are resing in lymph nodes and other tissues throughout your body. When a bacteria is in the fluids of your body the B-cell can bind to the antigen on the pathogen.
    a. What type of molecule is the receptor on the B-cell matched to bacterial antigens?
    b. After a B-cell binds to the bacteria, what will happen?
    c. What is the role of receptor-mediated endocytosis?
    d. > What type of MHC protein will the B-cell use to present the foreign antigen onto the B-cell's plasma membrane?
    e. The naive immunocompetent helper T-cells has a receptor able to bind to what type of MHC protein?
    f. > What happens after a helper T cell binds to a B cell presenting foreign antigen?
    g. What happens in clonal selection?
    h. > What type of cell will activated B cells change into?
    i. > What happens in the humoral attack stage? Are pathogens killed immediately?
    j. What do we call the molecule made by plasma cells that attach to the antigen?
    k. > How many antibodies are made by plasma cells in one second over what period of time?
    l. > If the first step in B-cell activation occurs but a helper-T-cell doses not secrete interlukin-2 then what happens?

47. What are the five different classes of antibodies? (Hint: remember MADGE)
    a. > What antibody increases rapidly after a first exposure to a bacterial infection?
    b. As days pass, what antibody becomes the dominant circulating antibody?
    c. On 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 can cross the placenta and provide natural passive immunity to a newborn?
f. > 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?
g. What antibody is the surface receptor for mast cells?
h. What antibody forms a linkage between a toxin and RBCs?
i. What four mechanisms are used by antibodies to render the antigen harmless?
j. > Do antibodies directly "kill" pathogens? If not then what do they do?

48. When B-cells are first exposed to an antigen it is called the first (primary) response.
    a. How long does it take before you see the first antibodies after being exposed to an antigen for the first time?
    b. > What cell type initiates the second exposure response? How is this different from the first response?
    c. When do you see a spike in the antibodies after a secondary response?
    d. What type of antibody is present during a secondary response?
    e. > Will you have a fever during a second exposure event?
    f. Should it surprise you to know you have many different types of G antibodies curculating in your blood all the time? Why? Significance?

49. > What is a hypersensitivity?

50. > What are the four different types of hypersensitivity.
    a. What is significant about these terms: natural vs artificial and active vs passive?
    b. What are the four different types of hypersensitivity? Mechanisms?
    c. Explain how these conditions are characterized and give an example of each type?
    d. What is the difference between a Type-1 reaction if it is local vs systemic?
    e. What term is associated with a Type-1 hypersensitivity?