

The Lymphatic System and Immunity - S2017
Study Guide C22 (Part B – Immunity)
Tortora and Derrickson

1. What is a pathogen?
2. How does your body defend itself against pathogens? What are the “three lines of defenses”? Which are innate and 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 and lymphocytes 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 does one of the chemicals released by mast cells effect the arterioles in the systemic tissue and the airways in the respiratory tissue?
20. Where do the mast cells’ 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 that leaves the blood and “wanders” throughout the connective tissue? b) What is the primary role 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 (hint: found in select organs)?
22. In order to understand the physiology of immunity, you need to know each individual component and then understand it’s function before you can understand how the immune system works.
 - a. What is interferons? What type of cell releases interferons? Does it “protect” the infected cell? What type of non-specific defensive cells are activated by interferons?

- b. What type of molecule is complement? What organ makes complement? Where is complement located after it is formed? What happens to complement when it is activated?
 - c. What are the four methods by which complement can destroy a pathogen?
 - d. What is the difference between antibody-dependent and antibody-independent complement activation?
 - e. Which complement mechanism requires RBC to transport pathogens to the liver?
 - f. Which complement mechanism requires the pathogen (foreign cell) to be “battered up”? Why? What is this process called?
 - g. 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 its membrane?
 - h. 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 NK cells?
 - b. How do NK cells kill cancer cells and foreign cells?
 - c. How is perforin similar to complement?
 - d. What does perforin require to complete the job?
 - e. Is the “death” caused by NK cells described as necrosis or apoptosis? (Explain)
 - f. Can you think of another non-specific mechanism that uses a similar mechanism?
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. What are cytokines?
 - 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 mechanisms are focused in the “local area” of tissue damage.
- a. How does the body bring all the components 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 type of objects move from the blood into the interstitial space?
27. Endothelial cells play an important role in inflammation. These simple squamous epithelial cells are able to mobilize both cellular and protein objects in the blood to come to the site of tissue damage.
 - 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 be successful, the response must keep the “cause” of the inflammatory response from spreading beyond the “local” 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, surrounded by a capsule of fibrin?
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 wander throughout the connective tissues of the body. They are the 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?
 - 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 prodigious 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. What is the contained puss called?
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, eg. 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, we say that you are “running a fever”.
 - a. What is another term for fever?
 - b. What are medications called that reduce fever? Do these drugs do more harm than good?
 - c. How do neutrophils and macrophage play a role in fever?
 - d. Where is the body’s “thermostat” located?
 - e. What are the six events associated with fever? (Be able to define onset, stadium, and defervescence).
 - f. 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 characteristics distinguish immunity from nonspecific resistance?
34. Immunity was first “recognized” as early as 500 BC. In the mid-1900’s it was recognized 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” stage 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 can work against the same infection but in different ways?
35. How are these terms used to classify immunity? (active vs passive and natural vs artificial) List and explain the four catagories.
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”?
 - 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. T-cells’ life cycle consists of three stages: birth, training (also known as education) and deployment.
 - a. Where are T-cells born?
 - b. Where do T-cells become immunocompetent?

- c. What happens to the T-cells to make it immunocompetent?
 - d. Thymic reticular cells “test” T-cells to see if they are capable of becoming functional immunocompetent T-cells.
 - e. What is negative selection? What are the two forms?
 - f. What percent of T-cells actually become functional immunocompetent T-cells?
 - g. What is positive selection? Where do these cells go?
38. B-cells are produced in the red bone marrow and stay there to become immunocompetent. Like T-cells, they need to pass a test to become functional B-cells. What do self-tolerant B-cells produce on their plasma membranes?
39. As a general rule, T-cells can not recognize antigen by themselves. They need the help of antigen-presenting cells.
- a. What are some of the cells that function as APCs?
 - b. What is a major histocompatibility complex?
 - c. What is a MHC protein? Where are they produced and where do they go?
 - d. If the MHC protein is the hot dog bun, what is the hot dog?
 - e. Who is going to eat the “bun and hot dog”?
 - f. What molecule is used to signal that the meal was eaten?

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

40. You already know about MHC proteins. Now we need to introduce two types of MHC proteins (type I and type II).
- 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 type protein in the cell’s cytoplasm and presents it on the cell’s plasma membrane, what happens?
 - d. Key Idea: What type of cell recognizes MHC I proteins?
 - e. What type of cells have type II MHC proteins?
 - f. What do MHC II proteins display on their cell’s membrane?
 - g. Key Idea: What type of cell recognizes MHC II proteins?
41. What is clonal selection?
42. Helper T-cells perform a “central” role in the overall immune response. After a helper T-cell recognizes an 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 healthy helper T-cells, can you respond to foreign pathogens?

43. What is the primary role of cytotoxic T-cells? What is a lethal hit?
44. What are memory T-cells? What “type” of T-cells become memory T-cells?

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.

45. What do B-cells have on their plasma membranes?
- What is the role of receptor-mediated endocytosis?
 - What is the role of MHC II protein?
 - What is the role of helper T-cells?
 - What happens in clonal selection?
 - What happens in differentiation?
 - What happens in the attack stage?
 - What do we call the molecule that attacks the antigen?
 - How many of these molecules does one cell make in one second over what period of time?
46. What are the five different classes of antibodies?
- Which antibodies are used as the B-cell surface receptors?
 - Which antibody can cross the placenta and provide natural passive immunity to a newborn?
 - 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?
 - What are the four mechanisms used by antibodies to render the antigen harmless?
 - Do antibodies directly “kill” pathogens? If not then what do they do?
47. When B-cells are first exposed to an antigen it is called the primary response.
- How long does it take before you see the first antibodies after being exposed to an antigen for the first time?
 - How is a secondary response different and what type of cell allows for this to happen?
48. What is hypersensitivity?
49. What are allergies? Give examples of common allergens:
50. One classification system recognizes four different types of hypersensitivity. What are the four different types of hypersensitivity? Explain how these conditions are characterized and give an example of each type?

