

PCB 3233  
Exam 2 Study Guide  
Chpt. 3 and 4

Chapter 3

- 1) When the following receptors are engaged what mechanism is initiated in a phagocyte? Mannose receptor, CR3 and CR4, or Scavenger receptor.
- 2) When the following receptors are engaged what mechanism is initiated in a macrophage? TLR's.
- 3) What do TLR 3, 4, 5 and 7 bind to?
- 4) Know the TLR4 signaling mechanism. All of the extracellular including LBP and intracellular, MyD88-TIR domain and IKK-NFkB-→signaling.
- 5) In the previous question, what 5 genes (that you learned about) are transcribed because of the TLR-4 recognition?
- 6) What is the general term given to these 5 gene products? (Hint; 2 part name including what they result in along with what type of molecule they are)
- 7) Know what neutrophils do and what lineage they come from during hematopoiesis.
- 8) What is another name for neutrophils.
- 9) Know the 4 classes of adhesion molecules and which adhesion molecule they bind to. Ex; LFA-1 binds to ICAM-1 and vascular addressin (CD-34 binds to L-selectin)
- 10) Know the 4 steps of extravasation.
- 11) How can you tell the difference between a general cytokine vs. a chemokine?
- 12) Neutrophil granules and the names of them.
- 13) Respiratory burst associated with neutrophils.
- 14) What are the 2 acute phase proteins you learned about and what can they do? I am excluding serum amyloid A from this. Hint: We talked about each being able to do two things.
- 15) Know all 3 complement pathways. What has to be present for them to start, the initiating enzyme and all the following components cut in order.
- 16) Name of the complement convertases and their components.
- 17) Type I interferons vs. Type II interferons.
- 18) What cells can produce Type I interferons?
- 19) What is the main innate immune cell activated by Type I interferons?
- 20) Know the 2 effector functions of NK cells. Hint; one is cytotoxic
- 21) What type of infection do NK cells typically fight against?
- 22) What type of response is created in a cell that an NK cell has targeted (virally infected)?
- 23) What cytokine does an NK cell secrete to activate macrophages?
- 24) What happens to virally infected dendritic cells when NK cells outnumber them? What happens to virally infected dendritic cells when they outnumber NK cells?

Chapter 4

- 1) What does the germline configuration of the Ig variable region genes look like in all cells except mature B-cells? What does the germline configuration of the TCR variable region genes look like in all cells except mature T-cells?
- 2) What gene segments need to come together to make a functional gene (for the variable region) in B-cells for heavy and light chains?
- 3) What are the different isotypes of heavy chains and light chains?
- 4) Which chain (heavy or light) confers the antibody's isotype, heavy or light?
- 5) Know the major points for the different antibodies.
- 6) What do the constant domains do for the Ig's? Do they bind to the antigen, confer effector function or have no function at all?
- 7) What does CDR stand for and why are these important? What does HV stand for? Where specifically are these in the BCR/Ab?

- 8) Which gene segments provide diversity to CDR1 and 2? Where does the diversity for CDR 3 come from (which gene segment)?
- 9) Know the major characteristics of the different antibodies (Ab's). Ex. bivalent, highest concentration Ab in blood, allows for phagocytosis, can cross the placenta, small (good in extravascular areas)
- 10) What are the ways we can get diversity in the antigen binding sites. (combinatorial, multiple V, J and D segments can be used and N and P nucleotide additions ((junctional diversity))
- 11) Somatic recombination in greater detail. Rag genes and gene products.
  - a. RAG1 and RAG2 - which two cell types are these active in and what do they do?
  - b. RSS – what are these and what two cell types would these be useful?
  - c. 12/23 or 1turn-2turn rule
  - d. TdT
  - e. P and N nucleotide additions
- 12) What is somatic hypermutation? Where does it take place? Which cells does it affect? When does it happen, before or after activation)? Is there a specific area that is mutated more than other areas?
- 13) Switch regions. Where are they located? What do they switch? How does it switch? Which cells does this happen in and when does it happen? What benefit does it provide? What can be the Ab isotype be switched to and which isotypes can't be switched to?
- 14) Affinity maturation. What does this result in?
- 15) Look at figure 4.37 (the changes in the immunoglobulin genes that occur over a B cell's lifetime) Understand that chart. Know which changes are permanent (changes to DNA) vs. those that are not permanent (alternative splicing of RNA). You will most likely have to go back into the text to get a good understanding of what is going on in this chart.
- 16) An Ab is made up of 4 chains (2 heavy and 2 light) what holds them together?
- 17) In a given Ab, are the 2 heavy chains exactly the same and are the 2 light chains exactly the same?
- 18) What is allelic exclusion and how does it affect your adaptive immune cell receptors?
- 19) From IgG to IgG where do you expect to find the greatest amino acid differences? Constant regions or variable regions and if the answer is the variable regions, which specific regions of the variable regions?
- 20) Do light chains have isotypes? If so, how many different isotypes are there and what are they? Can one Ab have multiple light chain isotypes?
- 21) What happens during somatic recombination and what happens during somatic hypermutation? Where and when do each happen?
- 22) What enzyme is involved in both somatic hypermutation and isotype switching?
- 23) Look at the different epitopes. (ex. linear vs. discontinuous)
- 24) What does a B-cell do to change a membrane BCR into a secreted antibody?
- 25) IgD and IgM membrane immunoglobulins. Are the antigen binding sites the same? What is different between the two immunoglobulins? How are they both on the surface at the same time? If they cut the DNA to change isotype, could they both be expressed at the same time?
- 26) Look at fig. 4.21 and 4.28. Have an idea of the linearity of constant region genes and know which constant region genes have switch regions in front of them and which constant region genes don't have a switch region. Why doesn't C delta need a switch region in front of it? If you switched from IgM to IgG1 could you ever go back to IgG3 or IgD?
- 27) How many subclasses of IgG and IgA are there? Fig. 4.33