PCB 3233
Chapters 9 + 10 Study Guide

Chapter 9

1. Plasma cells can differentiate directly from activated B cells, somatically hypermutated and isotype switched centrocytes, or memory cells.
2. The B cell co receptor is made up of CR2 (complement receptor, can bind C3d), CD19 (the receptor signaling chain), and CD81.
3. CR1 will bind C3b, which makes it susceptible to cleavage into C3d and iC3b.
4. TD antigens are thymus dependent antigens and B cells must receive T cell help in order to become activated to these types of antigens. TI antigens are thymus independent antigens, and B cells will not be required to get a signal from a T cell to be activated. A TI-1 antigen will activate other receptors on the B cell like TLR-4 (LPS). A TI-2 antigen will bind to so many BCRs because of a repetitive epitope that it will activate a B cell by itself. TI-1 uses LBP (soluble LPS binding protein), which binds to LPS. Bacterial cell wall polysaccharides are an example of a repetitive epitope. Since a B-1 cell will be activated by a TI-2 antigen, only IgM will be secreted.
5. 1. Receptor clustering 2. Blk, Fyn, or Lyn will phosphorylate the ITAMs 3. Syk will bind to the phosphorylated ITAMs of the Ig-beta tail 4. When 2 Syks are close together, they will phosphorylate each other, and lead to intracellular signaling.
6. First a BCR signal is needed. Then a BCR co-receptor signal is needed. CR2 binding C3d will cluster the BCR and co-receptor. CD19 will get phosphorylated. CD19 now binds Lyn to send signal. This is still not enough to activate a B cell usually. When a T cell binds the antigen on the B cell, a CD40-CD40L interaction takes place. A signal is sent to activate NFKB, which then up-regulates ICAM1 which allows the B cell to bind to the T cell better. The T cell will now secrete cytokines like IL-4 to drive B cell proliferation.
7. Follicular Dendritic cells are dendritic cells presenting antigen in the follicles or germinal centers of a lymph node. The dark zone consists of dividing centroblasts, and the light zone consists of centrocytes and FCDs. Iccosomes are bundles of membrane coated with immune complexes that also bud off from the surface of FDCs.
8. Antibodies do not kill a pathogen directly, they either opsonize it or neutralize it.
9. The 2 roles in B cell activation by the BCR is 1. Binding antigen to change gene expression, and 2. Internalizing antigen to put it up on MHC II and present to T cells. Cognate interactions are interactions between a B cell and a T cell that recognize the same antigen. A primary focus is formed between T cells that are activating B cells in the medullary cords. IgM is secreted here. The medullary cord is found near the efferent lymphatic vessel. IL-4 drives B cell proliferation. IL-5 and IL-6 influence the B cells found in the primary focus.

10. IL-4 will tell a B cell to become a memory cell, whereas IL-10 will tell it to become a plasma cell.
11. Patients who lack the CD40L on T cells have hyper IgM syndrome. Since their B cells cannot be activated by T cells, they have abnormally high IgM levels since they cannot isotype switch. Males are mainly affected because the gene is located on the X chromosome.
12. Fc regions will deliver antibodies to anatomical sites inaccessible otherwise, and also link antibodies to cells via Fc receptors. Phagocytic cells do not express a Fc mu receptor, so IgE is not an opsonizing antibody.
13. Brambell receptors (FcRB, FcRn) are used to transport IgG from blood to extracellular places. They are present on the endothelial cells. Two molecules of FcRb bind to the Fc region of one antibody. The antibodies bind to the alpha 1 and 2 regions of the FcRB.
14. Poly-Ig receptors are used to transport IgA across mucosal surfaces into the lumen. Only dimeric IgA can use these.
15. Transcytosis – receptor mediated transport from one side of a cell to the other of a macromolecule
16. IgG is transported through the placenta to the baby using the Brambell receptor.
17. IgA is found in the breast milk, and in the baby it will protect its mucosal surfaces.
18. For venoms, passive immunization is used. (antibodies created using a large animal inoculated with the venom)
19. These antibodies are gathered from the animal and given to a snake bite victim to neutralize the toxins (no long term immunity). A horse can be given snake venom, make antibodies, and then the antibodies can be given to the snake bite victim.
20. An immune complex consists of antibodies, antigens, and sometimes complement. Erythrocytes are a major part of their uptake and destruction.
21. Dimeric IgA is made in the lamina propria underneath mucosal surfaces and is used to stop pathogen from entering.
22. Fc receptors allow antibodies to be carried to places where they can't be carried by blood, and allow antibodies to attach to effector cells.
23. ADCC is a mechanism used to lyse specific cells that have been coated with a specific antibody (tumor cells i.e.). NK cells (mainly) , macrophages, neutrophils, and eosinophils can mediate ADCC. Antibody Dependent Cell-Mediated Cytotoxicity. CD16 receptors on NK cells recognize complement.
24. One IgM and two IgG can activate the classical complement cascade. C3b is used for opsonizing. Classical C3 convertase is C4b2a .
25. The alternative pathway can amplify the cascade started by the classical system. Once C3b has been deposited onto pathogen surface, the alternative C3 convertase and be made, which is C3bBb.
26. CR1 can bind to C3b. CR2 can bind to C3d and iC3b.
27. NK cells are part of the innate immune system. They are lymphocytes. They enter the site of infection immediately. They circulate throughout the blood. Macrophages activate NK cells by secreting cytokines. NK cells have 2

effector functions, secreting cytotoxins and cytokines. NK cells are partially activated before entering infection site. NK cells use NKG2D to detect MIC A and B (activating), and KIRs to detect MHC I (inhibiting).

28. You would have much high antibody affinity and much higher levels of antibody in a 2nd immunization.
29. Protective immunity are antibodies that are residual from the primary infection, not due to memory cells. Secondary immunity is due to the memory cells.
30. MALT has the highest number of lymphocytes. Most of the antibody found in the body is found on mucosal surfaces. IgA is found in highest concentration in the body.
31. M cells are involved in the transcytosis of pathogens. They don't secrete anything. They take pathogen from the gut into a Peyer's Patch.
32. Peyer's Patches are dome shaped structures with B cell follicles or germinal centers, and also T cell areas with dendritic cells.
33. The macrophages and dendritic cells in the gut are much less likely to cause an inflammatory response. Macrophages are still phagocytic and destructive, just not able to cause inflammation.
34. If a lymphocyte is activated in the gut it can only serve the MALT now because they express intergrin alpha 4: beta 7 which binds to vascular addressin MAdCAM-1 on endothelial cells of the gut. CCL25 will guide their migration in the lamina propria.
35. Lymphocytes vs. Leukocytes
36. Memory t cells can be activated at the site of infection because a B7-CD28 interaction isn't needed. Naïve T cells can be activated in Peyer's patches.
37. Peyer's patches are characteristic secondary lymphoid tissue of the small intestine
38. Mesenteric lymph nodes are the largest lymph nodes of the body. The tonsils guard the airways. The Peyer's patches are in the gut.
39. A naïve B cell cannot be activated in a secondary immune response.
40. Immunological response will remain for a few months after infection.
41. Naïve B cells will not help in a secondary immune response because they are inhibited.
42. Yes, B cells will still undergo somatic hypermutation and isotype switching during a secondary immune response. The same process happens.