

**BIO 401. IMMUNOBIOLOGY. FALL 20111.**  
**REVIEW SHEET FOR SECOND** (This is only a study aid to help you prepare for the exam!!!)

**CHAPTER 7. Antigen- Antibody interactions.**

1. Be able to describe the nature of the bonds made between the antigen and antibody. Given the nature of these bonds, be able to describe the reasons that the antigen and antibody must interact in a way so that the antibody reaction site is a mirror image of the topology of the binding surface of the antigen. Why can cross reactivity occur between two antigens with a single antibody?
2. Be able to interpret the concepts of affinity and avidity as they relate to monoclonal and polyclonal antibodies. Do not forget the concept of cross-reactivity!!! (last exam question!)
3. Be able to outline the basic steps of techniques describing the usefulness of antibodies: double diffusion, radial immunodiffusion, SDS-PAGE, ELISA (seen in the lab; for Ag or sandwich), Western blotting, immunofluorescence, immune magnetic isolation, and FACS. Which ones are most sensitive?
4. Be familiar with the different versions of the ELISA technique (direct, indirect, sandwich).
5. How would you purify T cells from a mouse lymph node or spleen? Check magnetic isolation and FACS. What are the common fluorochromes used in immunofluorescence/FACS? Remember, in FACS isolation you start with 10,000 cells (100%) and you have to provide % of cells in each of your quadrants.

**CHAPTER 7. Complement.**

1. What is complement? What activates complement? What are the biological consequences of complement activation? What antibody isotypes can activate complement (human is OK)?
2. Be able to know the different pathways of complement activation. How each of these pathways is activated? Outline the classical pathway. Keep in mind that I am interested in knowing: 1) how it gets started?, 2) how the C3 convertase is formed?, 3) how the C5 convertase is formed?, 4) how is the MAC formed? (This is mostly the same for the Lectin pathway of complement activation).
4. Outline the alternative pathway. Keep in mind that I am interested in knowing: 1) how it gets started?, 2) how the C3 convertase is formed?, 3) how the C5 convertase is formed?, 4) how is the MAC formed?
5. What are the proteolytic (small degradation products) components in the classical, alternative and lectin pathways? Why are they important?
6. Complement system is not specific and must be regulated to avoid attacking the host. Describe at least 3 proteins involved in complement regulation and indicate at which step they are involved.... Early, middle, and late activation, pathway?
7. Be able to associate biological function (ex. Cell lysis) with particular complement products.
8. Be able to identify complement receptors with major ligands.
9. Be able to describe how Gram (+) and Gram (-) bacteria can avoid complement-mediated damage.
10. Be familiar with at least ONE clinical consequence of complement deficiency.

**CHAPTER 8. MHC complex:**

1. Know what a haplotype is in term of the MHC complex. Be familiar with MHC molecules in both the mouse and human.
2. Why are the MHC proteins referred to as "histocompatibility" proteins?
3. Why are the two MHC genes (alleles) that you inherited very important to you?
4. What is meant by the statement that the MHC genes are very polymorphic. How can you explain this phenomenon?
5. What functions are represented by the three types of MHC genes?
6. What is meant by the term, "congenic mouse", syngenic mouse?

7. What is the structure of the type I and type II MHC complexes? What is the role of the  $\beta_2$ -microglobulin in these structures?
8. Describe the structure of the type I and II MHC complexes. Is the antigens bound the same for both? Can these complexes bind antigen so long as the polypeptide antigen is of appropriate length? What are the differences in the way that these two structures bind antigen? (Table 8.2).
9. Be familiar with the distribution of MHC molecules in the F1 progeny when you cross mice with different haplotypes.
10. Considering all of the polymorphisms of the MHC I structure that have been studied, what is the significance of the differences in structure between one polymorphism and another?
11. Be able to present an argument as to the relative advantages of both the up regulation of MHC complexes as a result of viral infection as well as the down regulation as a result of infection with other viruses.
11. What is the selective advantage of the up regulation of MHC I complexes by cytokines such as IFN- $\gamma$  as well as IFN- $\alpha$  and IFN- $\beta$  and by tumor necrosis factor- $\alpha$ ?
12. Be able to make a general hypothesis relative to the polymorphisms of MHC complexes present and the relative susceptibility (risk) of the population to some diseases.

### **MHC Class I and MHC Class II Antigen Processing and Presentation**

1. Know what the term "restriction" means as it applies to MHC molecules. Be familiar with the experimental evidence (Fig 8.15) that lead to this finding.
2. What is the role of antigen presenting cells? Know the experiment that demonstrated the need for antigen processing by APC for activation of T cells (Fig. 8.3).
3. Know the difference between a professional APC and a non-professional APC. Why are DC better APC than macrophages? (see Table 8.3).
4. You must know the pathway for endogenous (cytosolic) antigen processing and presentation by MHC-I. Be able to draw and describe the role(s) played by the different chaperones/molecules. It is easier if you use different section/compartments to illustrate this pathway: from the initial antigen until it is displayed on the surface.
5. You must know the pathway for exogenous (endocytic) antigen processing and presentation by MHC II. Be able to draw and describe the role(s) played by the different molecules.
6. Understand the alternative presentation of non-peptide antigens by CD1 molecules. Why presentation by CD1 molecules is important?