Problem Set #4

1) What is responsible for the initial lag in the production of antigen-specific antibody observed after the initial injection of antigen? What happens to this lag period upon subsequent exposure to the same antigen and why?

2) How can an antigen activate B cell directly, without T cell help?

3) Do T and B cells have to recognize the same epitope on a complex antigen in order to cooperate with one another? Why or why not?

4) Which of the following require alterations at the (a) DNA level, (b) RNA level, (c) protein level:

- a) antigen receptor gene assembly
- b) class switching
- c) membrane vs. secreted form of Ig
- d) biosynthesis of IgA dimer
- e) somatic hypermutation

5) Many resting B cells express both IgM and IgD. How is this possible? Some splenic B cells in immunized mice have both IgG and very low levels of IgM on the cell surface. How is this possible?

6) Which of the following factors are required for both V(D)J recombination and class switching: RAG1, Ku70, RNA polymerase II, AID (activation-induced cytidine deaminase)?

7) Is somatic hypermutation limited to the expressed Ig alleles in a B cell? Design an experiment to find out (extra credit).

8) What cellular and molecular interactions are required to activate switch recombination? How is switch recombination regulated?

9)Explain how somatic mutation results in increases in antibody affinity during an immune response?

10) List the genetic events which must occur properly in order to produce a high-affinity IgG specific for a novel antigen. Name three genetic diseases which interfere with one or several of these events.