Quiz 3 practice questions

1. Why could a CCR5 deletion mutation protect a patient from AIDS?
   a. It prevents the replication of virus by compromising its ability to mature newly synthesized viral particles
   b. It makes the patient more responsive to the adoptive transfer of B cells
   c. It enhances complement activation
   d. CCR5 is a MAMP and increases anti-bacterial responses
   e. **It could prevent HIV attachment by eliminating an important co-receptor**

2. What is the consequence of defective RAG1 and RAG2 genes?
   a. There is a decrease in complement synthesis.
   b. There is an increase in complement synthesis.
   c. There is a change in phospholipase protein activation
   d. **These genes are important in mediating recombination of the antigen receptors on T and B cells, resulting in an ineffective adaptive immune response.**
   e. These genes are important in the production of hair.

3. What might the effect of an abnormally high level of MHC class II expression on antigen presenting cells be?
   a. This would increase endogenous antigen presentation.
   b. There would be a decrease in pigmentation in the host animal.
   c. **This could result in inappropriate presentation of exogenous self-antigens to helper T cells and lead to a propensity for the development of autoimmune disease.**
   d. There would be a change in $\beta_2$ microglobulin stability.
   e. This would have no effect on host immunity.

4. Cancer cells have to overcome genetic instability that occurs when the chromosomal ends shorten too much. How can they do this?
   a. **By expressing telomerase, an enzyme that can maintain the telomeres.**
   b. By expressing angiogenic factors.
   c. By replicating endogenous virus.
   d. By converting oncogenes to protooncogenes.
   e. All of the above (a-d) are correct.

5. Ricin is a toxic molecule of the Castor Bean plant. How could this molecule work as an immune conjugate?
   a. **An antibody conjugated to Ricin A could cause the accumulation of this subunit at a cancer cell, while Ricin B could be targeted with a second cancer-specific antibody. The B subunit would enable A to cross the membrane where A could inactivate ribosomal function.**
b. Ricin is intrinsically radioactive and can induce mutations that result in programmed death cascades to start.
c. Ricin is a lectin that glues cells together so that they starve to death.
d. Ricin activates the complement cascade leading to cell death from the formation of the membrane attack complex.
e. Ricin causes a resetting of the epigenetic regulation of cancer cells (such as teratocarcinoma Embryonic Carcinoma cells) to return them to normal differentiation programs.

6. Some chemicals are both mutagens and carcinogens. How might the mutagenic potential of a chemical in the environment be measured?
   a. Using the Ames assay to measure the reversion of chemically-exposed Salmonella typhimurium mutants that cannot make histidine to the wild type phenotype
   b. Measuring the number of cell cycles that a cell exposed to the chemical will go through in one day
   c. Measuring the kinase activity of the exposed cell
   d. Measuring the levels of transcription factor in an exposed cell
   e. All of the answers (a-d) are incorrect

7. What kinds of gene mutations can contribute to the progressively malignant character of a cancer cell?
   a. Genes encoding Kinases
   b. Genes encoding Phosphatases
   c. Genes encoding angiogenic factors
   d. Genes encoding Transcription factors
   e. All of the above (a-d) are correct

8. What is the principle use of phage display technology?
   a. To identify short gene sequences that encode binding partners for a specific target molecule from a large library of alternative sequences
   b. This measures the phagocytic potential of macrophages
   c. This measures the proliferative response of leukocytes
   d. This technology can be used to determine the affinity (Kd) of antibodies
   e. This technology can evaluate llama immunity to fungal challenge

9. What relevance do cannelid (e.g. camel and llama) antibodies have to immunotherapy?
   a. These antibodies are inexpensive to synthesize
   b. These antibodies are able to activate complement at a higher rate than other mammalian antibodies
c. These antibodies are very thermostable and can also function in chemically challenging environments.
d. These antibodies are able to crosslink all forms of fungal antigen
e. All of the above (a-d) are correct

10. Why does the NSG mouse include the gamma common chain knockout defect?
   a. The gamma common chain is a component of all immunoglobulins so the defect prevents B cell activation.
   b. The gamma common chain in this mouse model is a transmembrane component of several cytokine receptors, and thus compromises the response to those cytokines in this broadly immunodeficient mouse strain.
   c. The gamma common chain is part of CD3 and thus prevents T cell activation.
   d. The gamma common chain is a part of the complement cascade and thus prevents the formation of the membrane attack complex (MSC).
   e. None of the above (a-d) are true.