Study guide for final exam

The final exam consists of the following types of questions:

- 20 T/F @ 3 pts each
- 10 Multiple Choice @ 4 pts each
- 10 Fill-in-Blank @ 4 pts each
- 10 Short Answer @ 6 pts each

The general format of these questions will be the same as for the midterm. There will be 200 points total, compared to 100 for the midterm. The exam will be cumulative, but will only cover topics from the first part of the course that are directly relevant to the second half. To help with choosing what to review from the first half, here is a guide of topics that will be covered on the final.

Components of the Immune System
- Organs of the immune system: primary and secondary organs, their function.
- innate vs. adaptive immunity
- clonal selection

Innate Immunity
- Phagocytosis: types of cells involved/ opsonization/ mode of microbe recognition
- inflammation
- Complement: classical and alternative C3 and C5 convertases; function of C3a and C5a

Antigen Recognition
- Types of epitopes recognized by T vs. B and how this influences T cell help to B cells
- Ab isotypes

MHC and antigen presentation
- polymorphism of MHC and gene families
- MHC restriction
- alloreactivity
- general distinctions between class I vs. class II antigen presentation (e.g. endogenous vs. exogenous; types of T cells responding)

Ig and TCR diversity generation
- Know where and when CSR and SHM occur during humoral immune response (as discussed in chapter 9).
Lymphocyte Development
- mechanisms to prevent maturation of autoreactive T and B cells
- importance of T cell positive selection on host MHC

Here are some examples of topics from the first half that you DO NOT need to review:
  - Mechanism of VDJ recombination and CSR and SHM
  - Details of antigen processing and presentation
  - Details of antibody and TCR and MHC structure other than the signaling chains
  - Stages of T and B cell development
  - Don’t need to know details of antibody structure for final exam

Here are important topics from the post-midterm portion of the course

B cell activation
- TD vs. TI-1 vs. TI-II antigens
- where B cell activation occurs in lymphoid organs
- T cell help
- germinal center architecture
- function of FDC
- plasma cells and memory B cells

Antibody isotypes and effector functions
- Distribution in tissues and circulation
- which isotypes form dimers and pentamers
- mechanism of IgA and IgG transcytosis
- neutralization
- opsonization
- initiation of antibody-dependent complement activation
- which isotypes are well-suited for these functions and how that determines the major roles of different isotypes in host defense
- Fc receptors

Memory T cells
- Be aware of central and effector memory cells and anatomic location.
- Know that antigen is NOT required for maintenance of memory T cells

Diseases involving the immune system
- Understand hypersensitivity responses
- Study underlying potential causes involved in initiation of autoimmune diseases following microbial infection
Manipulation of the immune response
- vaccination
- organ transplant and mechanisms associated with allograft rejection

In addition:

Be aware of how armed and activated T cells exert antimicrobial effects

Understand the different pathways involved in either Th1 or Th2 generation following microbial infection and/or exposure to allergen.

Study leukocyte trafficking and the receptors/ligands involved

Understand conceptual basis for immunological processes i.e. evolution of an adaptive immune response and how this is controlled, etc.

Study how microbes can evade immune defense mechanisms

The 2 co-receptors used by HIV (CXCR4 and CCR5) that allow for entry into CD4-positive cells

YOU DO NOT NEED TO KNOW:
- basics of HIV replication, HIV proteins encoded by virus, how HIV enters cells e.g. function of gp120 and gp41 in mediating fusion with host cell envelope
- specific interactions between adhesion molecules

Sample Questions

T/F
A key feature of perforin and granzyme (secreted by CTL’s) is the fact that these molecules are antigen-specific

Antigenic shift is associated with dramatic changes in either H and/or N antigens of influenza virus resulting in pandemic outbreaks

T cells are required for antibody responses to polysaccharide antigens.

IgG is transferred from the blood into tissues by the receptor FcRB.

Antibodies specific for MHC class I are important in contributing to chronic tissue rejection following allogeneic transplant

TLR’s are only expressed upon the surface of cells?
Multiple Choice
A mouse that does not express TLR4 (TLR4 knockout) would not be able to respond to:

a. LPS
b. peptidoglycan
c. HIV
d. a and c
e. b and c

NK cells can contribute to host defense following infection through various mechanisms including:

a) cytolytic activity
b) secretion of IFN-g
c) recognizing foreign peptide via high-affinity antigen receptors
d) production of high-affinity antibody
e) A and B

Mast cells with bound IgE to their surface can be activated via the following mechanisms:

a) allergen-induced cross-linking
b) environmental exposure to high levels of Th2-associated cytokines such as IL-4
c) CD40L:CD40 interaction
d) A, B, and C
e) A and C

Potential mechanisms by which infectious agents may initiate autoimmune diseases include:

a) increasing mast cell degranulation
b) release of circulating autoreactive T cells from a resting state
c) expression of antigens that similar to host tissue antigens
d) A and C
e) B and C

Complement is efficiently activated by the following Ig isotype(s):

A. IgM
B. IgD
C. IgE
D. IgG2
E. All of the above

Fill-in-Blank
Antigen-specific CD4+ T cells can activate macrophages by what 2 signals:

_____________ and ______________
Phagocytic cells utilize the complement receptor ______ to bind to _______ coating the surface of the bacterial pathogen resulting in endocytosis and subsequent destruction of engulfed pathogen.

The immunological synapse is divided into two regions defined as ______ and ____________.

______________________________ are cells in the germinal center that trap antigens in the form of immune complexes.

Natural killer cells can kill virally infected cells coated with antibodies. This process is known as ____________________________.

**Short Answer**

You work in an immunology lab that has just created a strain of mice lacking CD40L. What effects would you predict this mutation to have on humoral immunity? Explain your reasoning.

Explain why *free* immunoglobulin molecules do not activate the C1q component of complement, and why they don’t cause Fc receptor signaling.

Provide three examples of antigenic variation utilized by *specific* microbial pathogens.

What are the four types of hypersensitivity responses and provide a brief definitive description of each response.