

Spring 2006 Immunology Exam #1 - Chapters 1 - 4

There is no time limit on this test, though I have tried to design one that you should be able to complete within 3 hours. You are not allowed to use your notes, any books, any electronic sources, nor are you allowed to discuss the test with anyone until all exams are turned in at 9:30 am on Friday February 3. **EXAMS ARE DUE AT CLASS TIME ON FRIDAY FEBRUARY 3.** Turning in an exam late will cost you a letter grade for each 24 hours. The **answers to the questions must be typed** unless the question specifically says to write/draw the answer in the space provided. If you do not type your answers on the appropriate pages, I may not find them unless you have indicated where the answers are. You will need black, blue, and red ink pens, as well as a regular pencil to answer at least one question on this exam.

There are 3 pages to this exam, including the cover sheet.

-3 pts if you do not follow this direction.

Please do not write or type your name on any page other than this cover page. Staple all your pages (INCLUDING THE TEST PAGES) together when finished with the exam.

Name (please print here):

Be sure to type your name here

Write out the full pledge and sign:

On my honor I have neither given nor received unauthorized information regarding this work, I have followed and will continue to observe all regulations regarding it, and I am unaware of any violation of the Honor Code by others.

avg. = 80%

range = 100 – 55%

How long did this exam take you to complete this exam?

~ 3.5 hours avg.

30 pts.

I. Define these terms: 3 pts each. First define the terms and then provide a specific example to further demonstrate your knowledge. Do not draw pictures as part of your definitions. These terms can be defined succinctly, so using a lot of words is not the best way to demonstrate your fluency with these terms. If you do not know a real example, make up one that is very similar to a real one, but be sure to indicate when you are making up a hypothetical example.

I am providing some key points. Variations are possible. Many examples were acceptable.

Langerhan's cell – immature dendritic cells living in the tissue that mature after moving to secondary lymphoid organs.

self antigen – any peptide or protein that can be recognized by a TCR or BCR. These can be normal proteins that lead to clonal deletion, or part of an autoimmune response.

γ/δ T cells – Antigen presenting cells that has γ/δ TCR with little polymorphism. Binds to free antigen like a BCR.

zymogen – pre-enzyme produced in an inactive form that must be cleaved in order to become activated.

Complement proteins C2, C4, C5, etc are good examples with C2b etc being the activated enzyme.

C5a – smaller portion of C5 after cleavage. Acts as a cytokine that induces inflammation.

adjuvant – typically part of bacterial cells and added to vaccines to induce a vigorous innate immune response so a vigorous adaptive immune response will be stimulated. Lipopolysaccharide (LPS) is an example.

hapten – a small molecule that cannot be detected by the immune system unless it is attached to a carrier protein. It can be recognized by antibodies; cAMP was a common example.

β_2 microglobulin – a non-variable portion of MHC I proteins that helps stabilize the α subunit. Does not bind to peptide.

allotype – allelic variation within the population. For any given isotype (e.g. IgG₁), there are many possible allotypes.

12/23 – rule applied to somatic recombination of variable regions of BCR and TCR. These are 12 or 23 base pair spacers separating a 7mer from a 9mer within an RSS. An RSS with a 12 spacer can “only” be recombined with an RSS that has a 23 bp spacer.

Part II

10 pts.

1) a. In outline format, explain the clonal selection theory.

Diverse BCRs are produced with different specificities.

Strongly self-reactive B cells will be deleted (clonal deletion)

B cells with high affinity for a particular shape will be activated and begin to divide (clonal selection).

B cells with low affinity will die due to loss of survival signal.

A similar outline could have been made using TCRs.

b. If all the mature B cells in a small population (≤ 50) came from one immature B cell, describe in detail the BCRs on these mature B cells.

An immature B cell has a rearranged Heavy chain, thus all its 50 daughter cells would have the same H chain protein, including the CDRs.

The 50 cells would have 50 different versions of light chains, λ or κ .

They would have IgD > IgM on the surface of the mature cells.

10 pts.

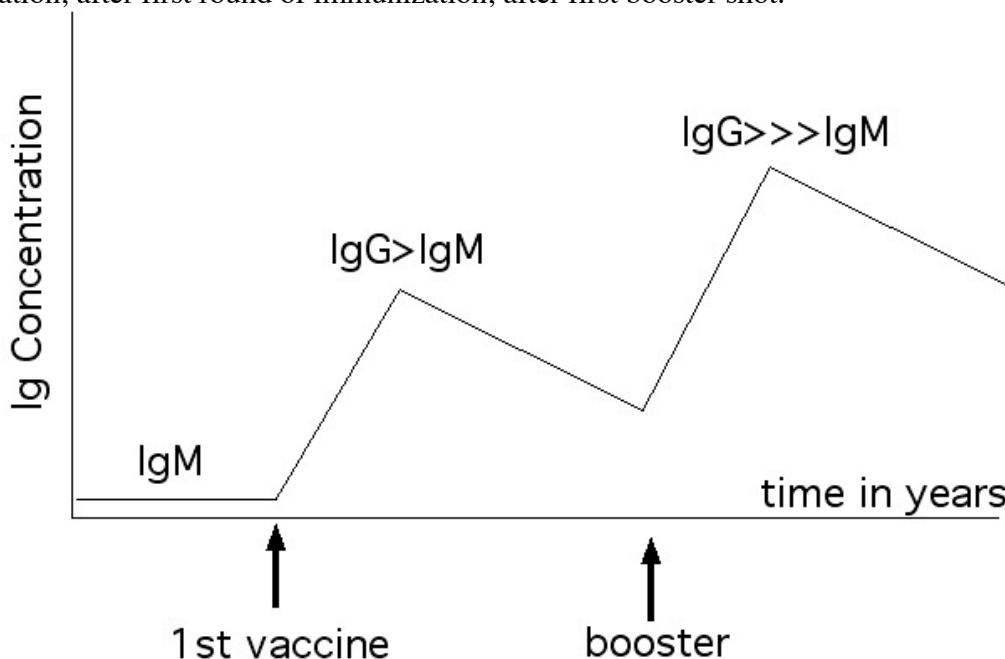
2) a. Give three reasons we get booster shots?

increase antibody cell titre

increase specificity of immune response

generate the right isotypes

b. Graph the anti-tetanus toxin antibody concentration and isotype in your blood over time (in years): prior to immunization; after first round of immunization; after first booster shot.



10 pts.

3) Warts are caused by viruses living in your skin cells. Explain why warts are able to survive in your skin and your immune system can do very little to get rid of them.

Based on chapters 1 – 4...

Viruses are not susceptible to the antimicrobial peptides on our skin cells.

They live IN the barrier, not beneath it.

They do not induce inflammation.

They do not stimulate INF-g and if they do, the skin cells cannot respond much at all.

Tc cannot reach the infected cells since living outside bodily fluid.

Not much MHC I on cells that are dying (normal skin cells).

No selection pressure since they are not harmful.

8 pts.

4) Based only on what we know so far (Chapters 1 – 4) about the immune system, explain a typical reaction to a mosquito bite.

Based on chapters 1 – 4...

inflammation is caused by breach of epithelium and leakage of serum.

Mosquitoes do not deposit many pathogens, typically.

Innate immunity only since the reaction is universal.

Must be reacting to either physical damage and/or repetitive antigens injected as anticoagulant.

Blood clots do form eventually.

Localized increased temperature and possible redness due to inflammation.

We have not discussed the adaptive immune response yet (IgE mediated).

10 pts.

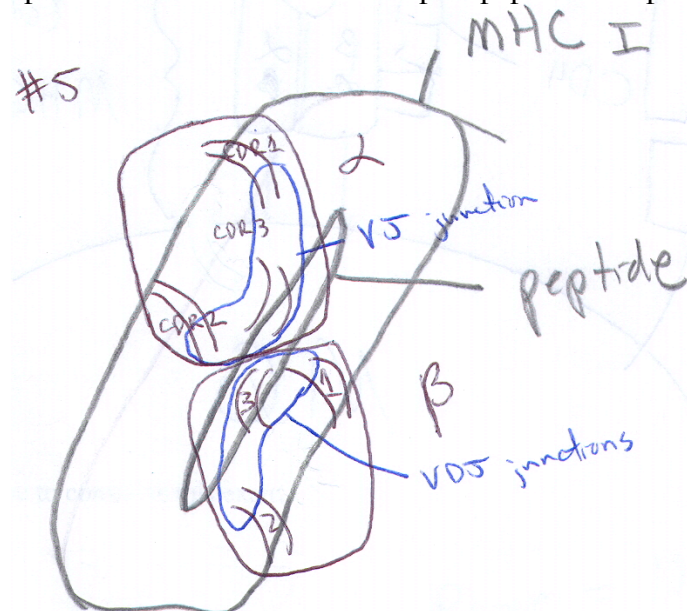
5) For this question, you will make one single drawing that has multiple layers. I suggest you practice at least once so you get it the way you want.

a. Use **black ink** to draw a picture of a TCR and label the CDRs.

b. Use **blue ink** to indicate the sites of TCR somatic recombination junctions.

c. Use **red ink** to indicate the sites of TCR somatic hypermutations.

d. Use **normal pencil** to superimpose the location of an MHC I plus peptide on top of your TCR.



6 pts.

6) List as many ways as you can think of which our immune systems kill bacterial infections.

The key was killing:

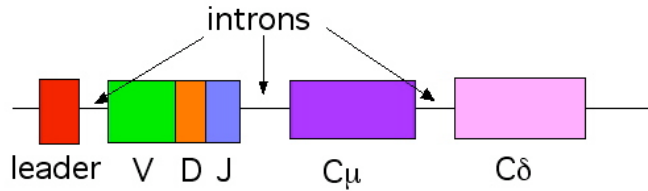
attack complex of the complement system

phagocytosis and digestion

oxidative burst or secretion of other toxic compounds (NK cells especially)

8 pts.

7) Draw a picture of a Ig heavy chain gene after somatic recombination that could be used to produce an IgD molecule.



8 pts.

8) Draw a picture of a T helper cell interacting with a macrophage in such a way that the T helper cell will be activated. Label all the important parts.

