Viruses. Retroviruses: totally different from other ssRNA genome viruses: they make dsDNA (reverse transcription) & integrate as provirus. Antigenic drift vs Antigenic shift in influenza A viruses. Hemagglutinin & Neuraminidase: important surface antigens on the surface (envelope) of influenza that distinguish one strain from another

AIDS: & HIV: abbreviations for what? What kind of virus is HIV?(retrovirus) What kind of genome does it have?(ssRNA) What enzyme does it provide to convert its RNA into DNA?(reverse transcriptase, an RNA-dependent DNA polymerase) Where does the HIV genome go once it is made into DNA?(it integrates into the infected cell’s genome as a provirus) What cell surface marker does HIV use to enter a cell?(CD4) What cells are CD4+?(T helper lymphocytes; macrophages) What kills most AIDS patients?(opportunistic infections or cancers due to immunodeficiency, their immune systems fail) What part of the world currently has the highest AIDS prevalence rates?(southern Africa) How is HIV primarily transmitted there?(heterosexual sex) What “problem” does HIV have that makes it difficult to design a vaccine?(high mutation rate due to inaccurate polymerase activity of RT)


Viruses & cancer: prefix onco-; what is cancer/neoplastic transformation? What is the difference between a proto-oncogene and an oncogene? 3 mechanisms by which viruses can cause cancer: DNA tumor viruses (integration into host DNA leads to inappropriate regulation of proto-oncogenes: normal cell genes involved, for example, in control of cell division; strong viral promoters over-express proto-oncogenes); retroviruses (which carry an extra gene with them, a true oncogene which is a mutated proto-oncogene; by itself, an oncogene can cause cell transformation into cancer); and chronic tissue damage, with constant cell division for repair over decades; each cell division risks a mistake in DNA replication; takes decades for cancer to result (example: hepatitis B virus). HPV (human papilloma viruses): cervical cancer; new vaccine available, standards for use not yet established.

Antiviral drugs: Nucleoside analogs: what are they “analogs” of, how do they work, why are they selectively toxic? Neuaminidase inhibitors: specifically target influenza virus (Tamiflu). Interferons (natural proteins of the immune system). Anti-retrovirals: know all 4 categories: nucleoside analogs, entry inhibitors, non-nucleoside RT inhibitors (which, like nucleoside analogs, target the viral polymerase, but these affect RT by a different mechanism than competitive inhibition); and protease inhibitors. These are administered to patients in combinations (HAART; why?). Understand what the HIV protease does.


Protozoa. Single cell eukaryotes. Giardia (cyst vs trophozoite, how transmitted); Toxoplasma (how transmitted: role of cats, eating contaminated meat; why the disease is important: unborn babies).

Plasmodium (malaria): Anopheles mosquito is the vector. Basic life cycle: mosquito bite; blood; liver; erythrocytes; synchronized red cell lysis & fever; gametocytes back to mosquito.

Ch. 16 Innate Immunity
Be able to distinguish between innate (nonspecific) and adaptive (specific) immune defenses. List some innate defenses against microbial infection: e.g., skin, sweat, coughing, lysozyme, stomach acid. How does transferrin protect against infection? Blood comes from stem cells in bone marrow. #WBC in blood increases with infection. Myeloid vs lymphoid lineages. Know key features each leukocyte type (don’t
confuse leukocyte with lymphocyte). Bacterial capsules protect against phagocytosis. Opsonization increases phagocytosis. What happens during phagocytosis (adherence, pseudopodia, role of capsules, fusion with lysosomes). What cell type tackles worms too big to phagocytose? (eosinophils) Natural killer (NK) cells kill by inducing apoptosis (cell suicide/programmed cell death)

**Lymph** (what is it? Where is it? What is lymphadenopathy?). What happens to bacteria that enter a lymph node? 4 cardinal signs of inflammation. What causes redness and swelling at the site of injury or infection? Increased blood flow and leakiness. Define diapedesis. What is pus? Bacteria that kill WBCs make lots of pus (e.g., S. pyogenes). What is a pyrogen? Why is fever good? What cells make alpha & beta interferons? (viraly infected ones) What cells are affected by alpha&beta interferons? (adjacent uninfected cells) What nucleic acid structure should not be found in normal cells and is a good marker of viral infection? (dsRNA) Who makes IFN-gamma? (lymphocytes & NK, not infected)

**Complement** is a cascade (not a metabolic pathway; what is the difference?). Consists of proteins found in blood. Name 3 things complement can do. Complement is considered innate/nonspecific (in its action) but it can also be triggered by the adaptive (specific) immune response (classical pathway involving antibodies). What activates the alternative pathway? What is the most important molecule in the cascade? (C3) What does C9 do? (membrane attack complex; complement-mediated cell lysis) What does C3b do? Define opsonization.

**Ch. 17 Specific immunity**: Humoral (B lymphocytes: bone marrow: antibody production; esp. important vs bacteria); Cellular (T lymphocytes: thymus: help & cytotoxic activity; esp. important vs viruses). Difference between antigen & antibody. What kind of molecules make the best antigens? (big proteins with lots of epitopes) Ig = immunoglobulin; antibodies are these kinds of proteins. Variable (antigen-binding) and constant regions. Know IgG & IgM: structure, functions, when they appear in the immune response. Name 3 things that antibodies can do (neutralize, opsonize, lyse) Name one way you can get antibodies other than having them made by your own B cells. (passive immunization: mother to baby) Does this kind of immune protection generate immunologic memory? (no) Does antigen exposure influence the specificity of a particular B cell’s surface Ig? (no, specificity is determined by random genetic changes early in B cell development) Each B lymphocyte has a unique immunoglobulin gene and produces antibodies with one unique specificity. Does antigen exposure select for the B cell clones producing the Ig with the strongest binding to antigen? (yes) What happens when antigen binds to surface Ig on an immature B cell? (clonal deletion for self tolerance) on a mature B cell? (clonal expansion and differentiation) What is a plasma cell? Anamnestic = secondary = memory immune response. What is required to make memory B cells? (antigen stimulation and T cell help) Which Ig class comes first in any humoral immune response? (IgM) How is antibody diversity generated when we cannot possibly have enough unique genes to code for all the different antibodies we make? (recombination & mutation) Which part of the Ig molecule is variable? (antigen-binding arms) constant? (stem) If self-tolerance mechanisms fail, what results? (autoimmune disease) How does the immune system distinguish between self and foreign (non-self)? (clonal deletion of self-reactive lymphocytes early in their development) What antigen-specific receptors do T cells have on their surface? (TcR) B cells? (surface immunoglobulin, or membrane-bound antibody) Two kinds of T cells (CD4+ helper: secrete cytokines/lymphokines/interleukins; cytotoxic: killers; CD8) Cytotoxic T cells are crucial for fighting intracellular infections (esp. viruses); they can kill with perforin. What kind of molecule does the T cell receptor bind to? (peptide antigen presented by MHC on a cell surface; this activates the T cell). **Class I MHC:** universal; all cells; presents samples of the internal environment so a T cytotoxic cell can find intracellular pathogens (eg viruses) & kill the infected cell. **Class II MHC:** professional antigen presenting cells (e.g., B cells, macrophages); presents peptide antigens derived from phagocytosis/endocytosis of the external environment to activate T helper cells. Loss of T cell help (death of CD4+ cells) is what causes the immunodeficiency in AIDS.
Vaccines: What do vaccines do for you? (produce immunologic memory without getting sick) 4 kinds: live attenuated, inactivated, subunit, toxoid). Live attenuated vaccines are usually the most effective (protective immunity in the largest % of vaccine recipients, and immunity lasts the longest); what is the hazard they pose? (immunocompromised people get exposed; reversion to pathogenic phenotype). What are booster shots for? Polio vaccines: understand why we switched from the live, oral vaccine to the dead, injectable form even though the live vaccine is “better”. Conjugate vaccines: name one organism vaccinated against with this new type of vaccine. Who benefits most? (babies) What are they directed against? (polysaccharide antigens of bacterial capsules) What type of specific immunity can polysaccharides induce? (humoral only) What is achieved by linking the polysaccharide antigen to a protein? (enlist T cell help, get memory) Why are conjugate vaccines polyvalent? (different isolates of the same bacterial species have unique capsule compositions) How can unimmunized people be protected by immunizing others? (herd immunity) Why do many people have an inaccurate perception of the risk/benefit of vaccines? (because they’ve never experienced the diseases) Rotavirus vaccine story.

Why is the transfusion reaction to the ABO blood group antigens so fast & severe? (preformed IgM antibodies formed due to exposure to cross-reactive antigens on gut bacteria) Why is ABO mismatch NOT important during pregnancy? (IgM doesn’t cross the placenta) How can hemolytic disease of the newborn be prevented in a Rh- mother? (passive immunization: treat with anti-Rh antibodies at the time of delivery to prevent sensitization so mother never makes IgG)