

Chapter 17 -- Specific defenses – Immune system immunology

Lymphocytes – a white blood cell involved in specific immune responses.

B cells + T cells

B cells – a type of lymphocyte that acts as one of the main components of the humoral immune system; differentiates into antibody-secreting plasma cells and memory cells.

T cells – a type of lymphocyte, which develops from a stem cell processed in the thymus gland, that is responsible for cell-mediated immunity.

Antibody (Ab) – a protein. Immunoglobulin (Ig) protein produced in response to an antigen.

Antigen (Ag) – something that invokes (or causes) an immune response. Recognition as foreign.

Examples: Any bacteria that does not belong, not normal flora, viruses, chemicals, it does not have to be a living thing, even your “self” can be recognized as foreign.

Types of immunology

Page 426, Figure 17.1

Sometimes immunity is innate (certain disease are species specific)

Artificially acquired (not acquired naturally)

- Active – vaccines cause an immuneresponse. Actually getting a little of the organism which will cause antibodies. Oral – not killed completely. Injected – killed completely.
- Passive – giving preformed antibody. You are giving person immune response. Ex. Phogam

Naturally acquired – natural process

- Active – get disease and make antibodies. Ex. Chicken pox.
- Passive – in utero – in uterus to transfer. Colostrum (breast milk). First fluids, very rich, good for baby.

Rh Factor – classification of red blood cells based on the presence or absence of Rh antigens.

Classic example of passive, artificially acquired immunity:

Rh Incompatibility

Blood types:

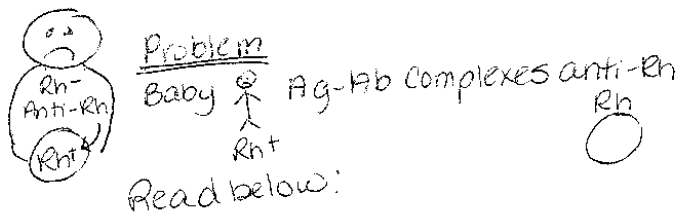
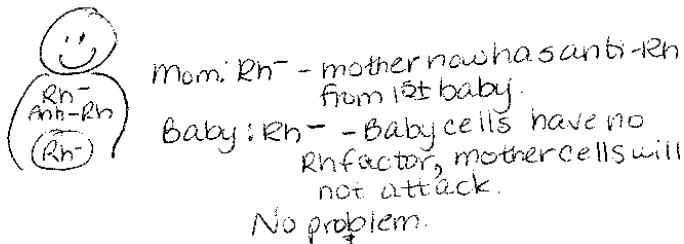
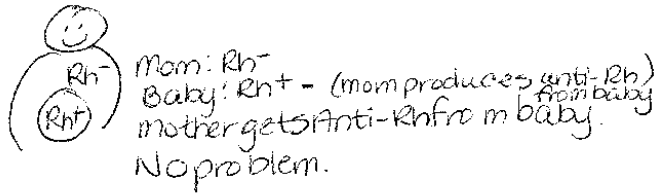
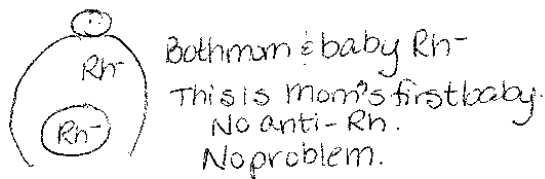
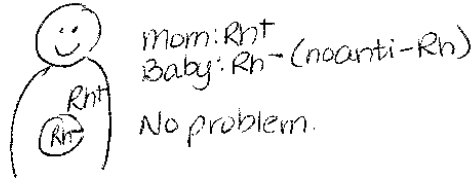
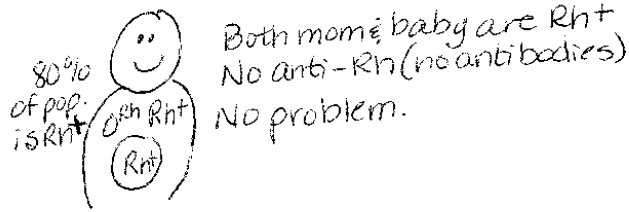
O^A Type A

O^B Type B

O Type O

O^{AB} Type AB

O^{Rh} Rh+



Mother cells will clump with babies blood cells. Will think it is foreign. When baby is born, will have body full of Ab-Ag complex (mom's anti-Rh stuck on baby's red blood cells when born).

Rhogam – passive artificially acquired (prime example). Shot given before (36 weeks) and after delivery to Rh – women. Coats the baby's cells. Mom will not form anti-Rh, or if is formed, will not attack baby.

B-cells

- Humoral immunity. Immunity produced by antibodies dissolved in body fluids, mediated by B-cells.
- Microbial (generally)

T-Cells

- Cellular (cell-mediated) immunity. An immune response that involves T-Cells binding to antigens presented on infected cells; T-cells then differentiate into several types of effector t-cells, including helper and cytotoxic.
- Tissue rejection.

Both are lymphocytes. Both develop in bone marrow (stem cells).

Stem cells – Fetal cells that give rise to bone marrow, blood cells and B and T cells.

B-cells – when your body, say, picks up a virus (say the flu), one of two things will happen, B cell is going to recognize on this antigen the surface receptors. On the surface will be glycoprotein receptors, the B-cell will recognize this, is going to cause these cells to produce clumps.

Page 433- Figure 17.8

Someone sneezes on you.

B-cell sees the antigen, recognizes a certain part of the surface, the “name tag” of the antigen called the receptor. There is a “map”. How this is worked out is still being investigated. Clonal collection theory. B-cell that recognizes the antigen, recognizes the surface receptor and starts to clone. Part of the clone is going to go in to your lymphatic system, and is going to sit there and remember the receptors on this virus. Other clone is going to go into your blood stream and they are going to produce antibodies.

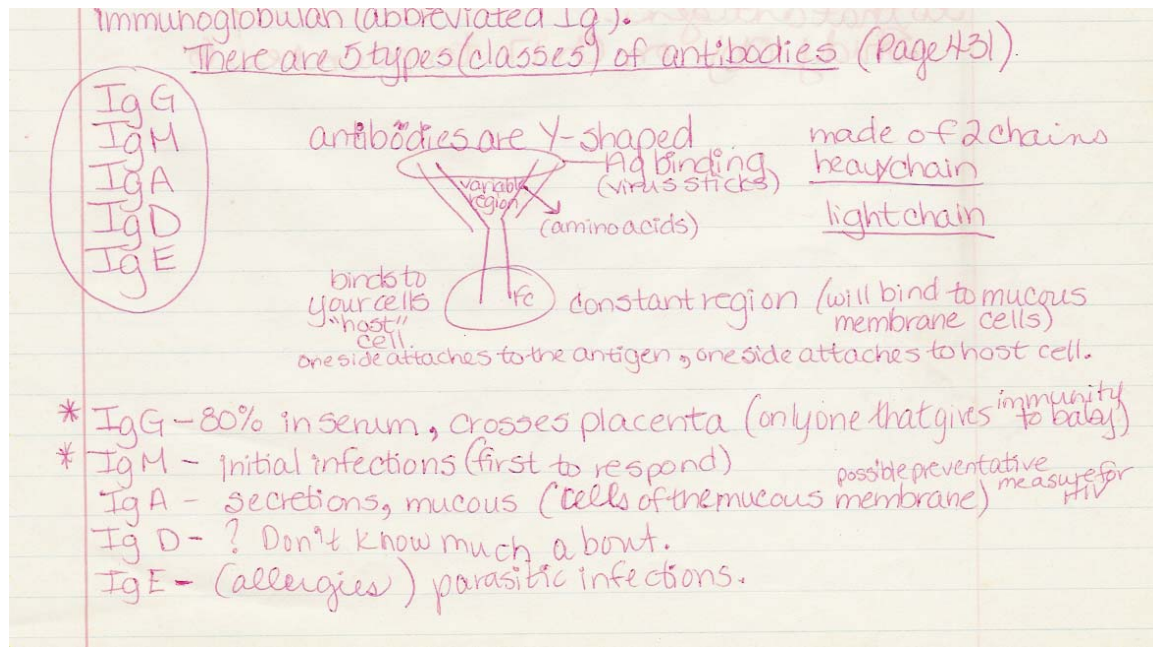
2 things happen to B-cells when they see a foreign agent “antigen”.

They are going to clone:

- Some of the clone will go into your lymphatic system, called memory cells. If you ever see this cold virus again, you will be able to stimulate this “whole army”.
- The rest of the clones are multiplied and they go into your blood stream and they produce antibodies and are called plasma cells.

You always get a different cold when you are sick. When you get a cold, you produce memory cells. Nice system, memory cells stay on, plasma cells hang around until organism is taken care of. Plasma cells put out antibodies. Antibodies are quite important. They are immunoglobulin (abbreviated Ig).

There are 5 types (classes) of antibodies (page 431)



Measles – increase in IgM, then rise to IgG.

2nd exposure – rise in IgG (IgM will not rise much).

Problem with this test is that you could also have a cold, it can mess up the indications.

Titer – how much Ab in your blood (serum)

Figure 17.10 – page 435

Anamnestic response – rise in IgG upon second exposure to Ag. (Also memory response) A rapid rise in antibody titer following exposure to an antigen after the response to that antigen.

Cellular mediated immunology – an immune response that involves T-cells binding to antigens present on infected cells; T-cells then differentiate into several types of effector T-cells, including cytotoxic and suppressor T-cells.

Cellular immunity

- T-Cell response
- Involves T-cells
- Several types of T-lymphocytes
- Helper T – stimulate B and T immunity. Do not kill, just stimulate. Helps out B & T cells. T_H – specialized T-Cells that often interact with an antigen before B cells interact with the antigen.
- Suppressor T – T_S – T-Cells that are thought to end an immune response after an antigen is no longer present. Suppressor T response. Do not kill, just stimulate.

- Cytotoxic T – T_C – Specialized T-Cells that destroy infected cells presenting antigens. “Natural killer” (NK) cells. These actually destroy pathogens directly.

Page 441 – Figure 17.15 – The central role of helper T-Cells.

T-cells are involved in tissue rejection, cancer. When your cells go out of control and cancer first starts it's your T-Cells that can take care of it. When your T-Cells can't take care of it, that's when it can grow into a tumor and cause problems. But you do have an immune system and many times when you have cells that might become cancer, are checked by T-Cells. One of the ways this is done (the way) is through this mechanism. Need to know this.

17.15

Microorganism carries the antigen (the receptor). Phagocyte (white blood cell). Not necessarily a T-Cell, could be a neutrophil, a phagocyte, or a macrophage. Here is the T-Cell so the phagocyte which is usually a macrophage or a neutrophil is going to eat up the bacterial cell. When it chews it up, it's going to have these lysozymes that will be released. These are the cellular name tags. These lysozymes are actually going to be brought to the surface of the macrophage that ate it. When the T-cells see this name tag on the macrophage, it says, “Hey, you ate something”, and it turns the T-cells on. It stimulates the T-cell. The T-cell does not get stimulated to produce cytokines (small proteins released from human cells in response to bacterial infection; directly or indirectly may induce fever, pain, or T-cell proliferation) to stimulate other cells. Cytokines that are released include interleukin. Interleukin is being explored as a possible therapy for AIDS because of this activity of T-Cells.

Helper T-cells when stimulated by this antigen will release cytokines. One of the cytokines is Interleukin 2. This cytokine stimulates other T-cells and it also stimulates B-cells. A patient with AIDS that has a drop in helper T, is not only going to have a problem with cellular immunity but also B-cells and humoral immunity (immunity produced by antibodies dissolved in body fluids, mediated by B-cells). Which means antibodies will be down. This person is severely immune compromised. B-cells are going to be affected because helper T-cells are going to be ineffective by the virus. This person is at a loss. An AIDS patient has complement left to help them.

Complement – A group of serum proteins involved in phagocytosis and lysis of bacteria. So complement is one of the few things that AIDS patients have left to help them out.

The helper T activates. What is the problem if the only thing that was recognized by the helper T was just the antigen? What would happen in your body? Remember the phagocyte just presents the antigen, the helper T might recognize it only as foreign. In the case of cytotoxic T-Cells, they kill the antigen.

Self molecule also called MHC – Major histocompatibility complex. The genes that code for the histocompatibility antigens; also known as human leukocyte antigen (HLA) complex. If we did not have these on the surface of all our cells, whether it be

macrophages, body cells, whatever, we would be recognized as foreign by our own immune system. Because we have this MHC, when the bacteria is eaten up, the nametag receptor of the bacteria is presented next to the MHC, so that the helper T looks at it and says, "Oh this is your cell presenting an antigen."

If it happens to be a cytotoxic T it will not kill your phagocytes. Now people who are on transplant waiting lists, are on these lists waiting for somebody who has a similar MHC, and the similarity between MHC's of the donor and recipient, the closer that is the less T-cell problems you are going to have. That's because it's the T-cell that's going to be responsible for transplant rejection. There are immuno-suppressant drugs, one of which being cyclosporine. This drug has recently revolutionized transplant therapy, and what it does is suppresses the T-cell response. So you end up having people that are walking the line between immuno-suppression where they are suppressing their immune system, but if they get sick they have to be careful. They are suppressing their immune system from rejecting the foreign tissue subjected by the transplant but if they get sick it is no good because they tend to get sicker than the average person. People on cyclosporine have to be very careful. The more things technologically advance in terms of transplants, and immunotherapy, the more things you have to look out for.

Only cytotoxic kill, the rest of the T-cells are going to help by stimulating more T and B which then go to the plasma cells which produce antibodies. Cytotoxic T-cell is going to attack the cell directly. Now this phagocyte is considered an:

APC – Antigen Presenting Cell. A macrophage or dendritic cell that engulfs an antigen and presents fragments to T-cells. Chews up bacteria and presents the nametag to the T-cell. That turns on the T-cell, the T-cell produces cytokines which turn on B and more T. Interleukin 1 is around but the major cytokine in T-cell stimulation is Interleukin 2. Interleukin 1 stimulates T_H cells in presence of antigens, chemically. Only T-cells that kill are cytotoxic.

Suppressor T-cells suppress just like cyclosporine does.

Problems encountered by people that have infected T-cells. Things like HIV.

HIV virus hits CD4 receptors. Usually refers to helper T cells.

You can use interleukin to stimulate cytotoxic T-cells in AIDS patients.

Source: class notes and textbook:

Tortora, Gerard J., Funke, Berdell R., Case, Christine L.

Microbiology: An introduction, Fifth edition. Redwood City, CA. The Benjamin/Cummins Publishing Co., Inc., 1995