**Lecture: 1 Main points:**

1- Why Immunology is a feared subject.  
2- Main lines of defense of immune system: Skin, Mucosal membranes, Innate and adaptive immune systems.  
3- Innate immune cells, some insights into macrophage function.  
4- Some insights into adaptive immune system including B cell variability, VDJ recombination, and antibody production. We also discussed T cells briefly.  
5- 2 step mechanism of immune activation: Signal one binding of MHC-peptide complex to B or T cell receptor.  
Signal two is the co-stimulatory siganl: B7 family on Antigen presenting cells (APCs) binding to CD28 on T cell.  
6- Understand the wired prorammed nature of the innate immune system and its major role in activating the more specific and refined adaptive immune system.

**Lecture: 2 Innate Immune system**

1- Innate Immune system is our second line of defense and made of: a- Complement system, b- Professional Phagocytes, C- NK cells

2- Complement system utilizes three main pathways: a- Classical b-Alternative c- Lectin-activated pathways

3- While the Classical pathway relies on antibodies, the alternative and lectin pathways do NOT need antibodies to cleave C3 molecules to C3a and C3b.

4- C3b will bind to pathogen surfaces forming C3 convertase, which further allows the formation of Membrane Attack Complexes (MACs).

5- Complement can promote opsonization through iC3b.

6-C3a and C5a act as chemotactic factors.

7- Professional phagocyte include Macrophages and neutrophils. Macrophages exist in various activation statuses.

8- Immune cells utilize a complex system of selelectins and integrins to bind to adhesion molecules on blood vessel endothelial cells and extravasate.

9- NK cells have both Activatory and inhibitory receptors. The balance between both signals helps decide whether the NK cells with kill the target cell they engaged with or not.

**Lecture: 4 B cells and antibodies:**

1- B cells are crucial components of the adaptive immune system and they produce antibodies.

2- As B cells mature they perform several actions to become better at what they do: This includes Antibody class-switch, Somatic Hypermutation, and career choice.

3- Initial antibody produced is IgM and is excellent in Complement activation. Switch then takes place to produce IgG, IgA, or IgE

4- IgG is a great neutralizing antibody and can cross placenta.

5- IgA is found in tissue and secretions, and can be found in colostrum.

6- IgE is ideal to fight parasites through its ability to activate mast cells.

7- Class switch is triggered by cytokine profile present.

8- Somatic hypermutation is the increased mutation rates in mature B cells to produce more variety in the Fab region and ultimately select for B cells that produce antibodies of higher affinity to antigen.

9- Career choice for B cells dictate whether a B cell will become a plasma cell producing antibodies or a memory cell to protect us against future infections of a similar nature.

**Lecture: 6 Ag presentation :**

1- Ag presentation involves an APC (Macrophage, DC, or B cell), presenting a peptide on an MHC class I or class II molecule.  
2-Internal peptides are presented on MHC class I molecules, while exogenous materials are presented on MHC class II.  
3-Ubiquitin-Proteasome pathway is mainly involved in the generation of internal peptides, while the lysosomal proteolytic pathway is responsible for the generation of exogenous peptides.  
4-MHC genes are pleomorphic, and are comprised of lots of variants providing a versatile ground for variety in antigen presentation abilities.   
5-MHC I and I (HLA 1&2) play a key role i organ transplants, andlow compatibility between donor and recipient will likely cause organ rejection.  
6- DCs are activated by cytokines or TLRs, and migrate to lymph nodes upon activation to interact with naive T cell and activate them.  
7-Macrophages usually stay at the fight scene and will provide restimulation for T cells that arrive to the battle site.  
8-B cells are special APCs in that they can be antigen concentrators, as they recognize a specific antigen using their BCR.

**Lecture: 8 T cells and cytokines:**

1- TCR on its own is unable to signal and will do so with the help of CD3 complex, which is considered a Pan-T-Cell marker.  
2- For a T cell to be activated a co-stimulatory signal needs to be provided by an APC. Famous Co-stimulatory families are: B7 and CD40 on APC, and their receptors on T cells are CD28 and CD40L, respectively.  
3- No co-stimulatory signal will casue T cell anergy, and a costiumlatory signal with no TCR engagement will have no effect on T cells.  
4- Th cells has many subsets defined by a profile of cytokines they produce and different functions. Such subsets include: Th0, Th1,Th2,and Th17.  
5- Th cells help immune system components by producing cytokines.  
6- Cytotoxic (Killer) T cells kill target cells by using perforin-Grnazyme B they have stored in them or by using FASL which binds to FAS on target cell and induces apoptosis.  
7- Delayed type hypersensitivity (DTH) seen in tuberculin test is driven by memory Th cells.  
8- Quantiferon gold test is superior to tuberculin test in screening for TB.  
9- CD40L on T cells is also essential to trigger class switch in B cells and in the generation of memory cells. Lack of CD40L in humans will cause hyper IgM immunodeficiency.