EXAM QUESTIONS

1. RESIDENT VS TRANSIENT
   Resident -are indigenous to the organism, meaning they have been on/in the organism since birth or for a very long time. Resident bacteria out number your normal cell count by a 10-1 margin
   Transient- are organisms that one may pick up day to day and only reside in or on the host for a short time. This is due to the resident bacteria being able to outcompete the transient (new) bacteria for resources in addition to altering pH, producing toxins and occupying space

2. BACTEREMIA VS SEPTICEMIA
3. COLONIZATION VS CONTAMINATION
4. INNATE VS ADEQUATE
5. WHY KEEP COPY FROM PATIENT OF AN ISOLATED BACTERIA
6. RESOLVING POWER
7. TYPES OF STAINING AND HOW
8. DIFFERENTIAL STAIN !!!!!!!
9. WHAT CHARACTERISTICS OF THE SKIN MAKE IT AN EFFECTIVE MECHANICAL BARRIER ?
10. WHAT IS THE DIFFERENCE BETWEEN INNATE AND ADAPTIVE IMMUNITY ?
11. WHERE DO IMMUNE CELLS COME FROM
12. ANTIBODY/ANTIGEN
13. WHAT STRUCTURES COULD BE ANTIGENIC IN BACTERIA/VIRUS?
14. ANTIBODY BINDING? HOW DOES IT OCCUR?
15. CLASSES OF IGS AND THEIR FUNCTIONS
16. 1ST AND 2ND RESPONSE

S.N.	Bacteremia	**Septicemia**
1.	Bacteremia is the simple presence of bacteria in the blood.	Septicemia is the presence and multiplication of bacteria in the blood.
2.	Bacteremia is not as dangerous as Septicemia.	Septicemia is a potentially life-threatening infection.
3.	Less amount of bacteria are present in blood.	Large amounts of bacteria are present in the blood.
4.	This may occur through a wound or infection, or through a surgical procedure or injection.	It can arise from infections throughout the body, including infections in the lungs, abdomen, and urinary tract.
5.	Toxins are not produced.	Toxins may be produced by bacteria.
6.	Bacteremia usually causes no symptoms or it may produce mild fever.	It shows symptoms like chills, fever, prostration, very fast respiration and/or heart rate.
7.	It can resolve without treatment.	Untreated septicemia can quickly progress to sepsis.
8.	Rapidly removed from the bloodstream by the immune system.	Antibiotics will be used to treat the bacterial infection that is causing septicemia.
9.	Caused by *Staphylococcus, Streptococcus, Pseudomonas, Haemophilus, E. coli*, dental procedures, herpes (including herpetic whitlow), urinary tract infections, peritonitis, *Clostridium difficile* colitis, intravenous drug use, and colorectal cancer.	*Staphylococci*, are thought to cause more than 50% of cases of sepsis. Other commonly implicated bacteria include *Streptococcus pyogenes, Escherichia coli, Pseudomonas aeruginosa, Klebsiella* species and even *Candida* spp.

3. Colonization - bacteria has to attach to establish w multiply
Contamination - bacteria is exposed/ deposited w out multiplication (doesnt have to do much)

LECTURE 2

Sub-clinical - hard to diagnose because no symptoms
Clinical - easy to diagnose because symptoms
Pathogenicity - the ability to produce disease
Virulence- ability to produce disease
Opportunism - do not normally cause disease but can do so when defense mechanism is breached/ compromised

- There are two types of immunity
- Non specific (INNATE) and adaptive

INNATE	ADAPTIVE
nonspecific defense mechanisms that come into play immediately or within hours of an antigen's appearance in the body.	antigen-specific immune response. The adaptive immune response is more complex than the innate. The antigen first must be processed and recognized. Once an antigen has been recognized, the adaptive immune system creates an army of immune cells specifically designed to attack that antigen
mechanisms include physical barriers such as skin, chemicals in the blood, and immune system cells that attack foreign cells in the body.	Adaptive immunity also includes a "memory" that makes future responses against a specific antigen more efficient.
The innate immune response is activated by chemical properties of the antigen.	Depends on past experience, directed against one type

INNATE IMMUNITY

Q. What makes the skin an effective mechanical barrier ?
ANS. 1. Impermeable 2. Dry (bacteria need moisture/water) 3.produces oils and other secretions that have antimicrobial properties

Other types of innate immunity

1. Mucous membranes ; cilia in RT, LYSOSOMES and pH
2. Iron-binding proteins; some bacteria require iron to grow example **transferrin and lactoferrin**
3. Phagocytes; **PMNs**. macrophages and **monocytes**
4. Complement ; group of proteins circling the blood * can bind to **antibody** making it more attractive (process called obsinizing) ****** can recognize certain things in bacteria and will form a circle creating a MAC pore in the bacteria making it leak out and die

Q. What is the difference between innate and specific immunity ?
INNATE; protects against any intruder, does not discriminate
ADAPTIVE/SPECIFIC; directed against one type , **depends on past exposure

SPECIFIC IMMUNITY (ADAPTIVE)
TWO TYPES 1. HUMORAL
2. CELL- MEDIATED (CMI)

HUMORAL IMMUNITY; Circulating antiBODIES

ANTIBODY

- A **protein** that binds specifically to its antigen
- Igs/ immunoglobulins
- ***produced by **B-lymphocytes** when stimulated by antigen presenting ***T-cells***
- Recognizes 1.capsules 2. Toxins 3. some viral proteins

ANTIGEN

- "NON-SELF"
- Protein, glycoprotein,lipoprotein, polysacc

ANTIBODY BINDING

Antibody= immunoglobulins produced in response to a **stimulation** by an **antigen** reacting **specifically** with it

- Distinguishes self from nonself

- Constant and variable regions (constant is bottom of y and variable is top)
- Variable is responsible for **antigen** recognition

5 classes of immunoglobulins

1. igG = host defense, crosses placenta and protects newborns
2. igD = role unknown
3. igA= host defense, found in secretions (saliva, milk, respiratory, GI) *DIMER
4. igM= host defense, **early immune response**, pentamer
5. igE= hypersensitivities, defends against parasites

1st and 2nd immune responses

1st

1. Antibody production triggered on first antigen introduction
2. *LATENT* period of several days
3. *Circulating* antibody detectable after 5-10 days
4. Antibody in serum is maximum -21 days and then drops

2nd

1. **BASIS FOR IMMUNIZATION**
2. Occurs when antibody is introduced for the 2nd,3rd,4th time…
3. *LAG*, rapid antibody increase (2-3 days), slow decrease
4. Booster injections to maximize antibody levels

SEROLOGICAL REACTION

- Detects the presence of ANTIBODIES in a serum sample
- The antigen and antibody reacts = **AGGLUTINATION**
- **Antibody titration**
- **Detects unknown microorganisms using ANTISERA**

CMI (CELL MEDIATED RESPONSE)

******T CELLS ARE NOT ANTIBODIES**

- Helper , suppressor, cytotoxic are generated from memories of T-cells
- Exposure to antigen induces response from trained T-cells
- **essential for defense against INTRACELLULAR organisms- parasites-tumors and other foreign substances.
- Immunosuppressive medication for transplant recipients

DISORDERS OF THE IMMUNE SYSTEM

1. **Allergy and hypersensitivity** = over reaction to presence of antigens in absence of true infections * can be fatal **ANAPHYLAXIS**

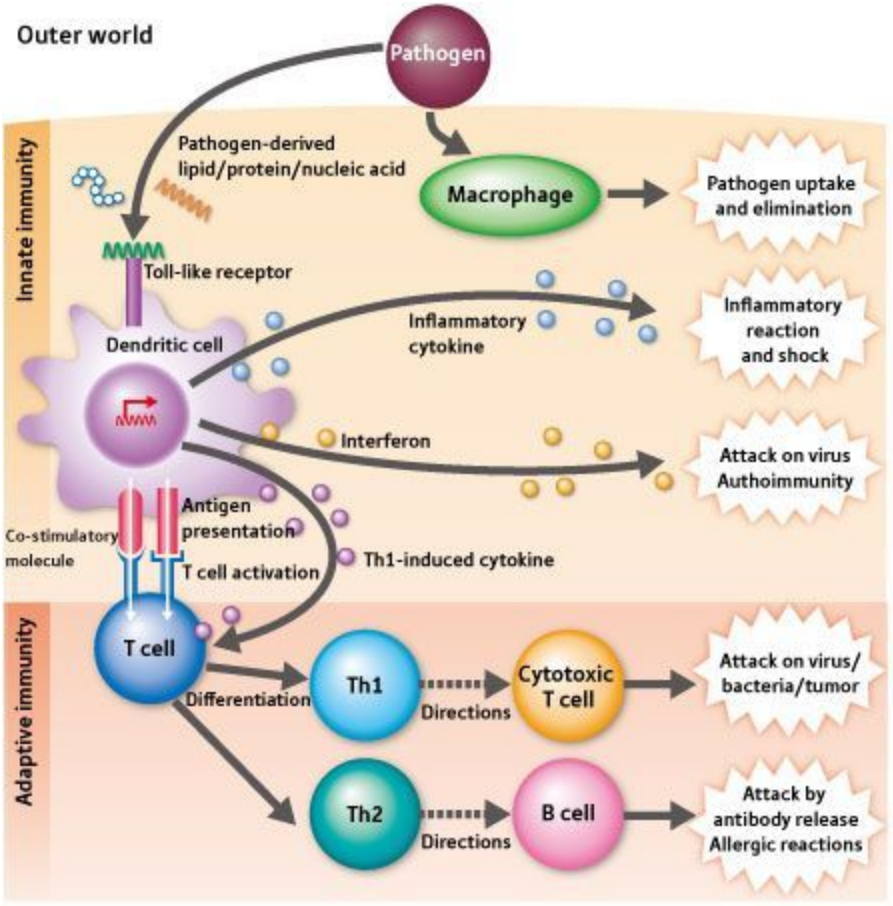
2. **Autoimmune diseases**= immune system reacts to its own "self" antigens **AUTOANTIBODIES **EXAMPLES; type 1 diabetes, lupus, rheumatoid arthritis

3. **Immunodeficiency state** =inability to produce antibodies and or dysfunction of CMI**ex congenital disease, AIDS

4. **Graft rejections=** normal reaction to "non self", controlled by immunosuppressive medication

IMMUNIZATIONS ; 1. PASSIVE 2. ACTIVE

1. Passive
    - The administration of PRE-FORMED ****antibody** against a ***SPECIFIC*** microbial agent
    - igG animal origin = short lived//risk of hypersensitivity
    - igG human origin= short lived// no risk
    - ***GAMMA GLOBULIN*** (igG)- pooled from a large group of blood donors and has antibodies to many common infections
    - ***HYPERIMMUNE GLOBULIN*** (igG)- specific for a particular microbe



2. Active
   - Stimulates immune system by administration of **ANTIGENS*****
   - LONGER LASTING

1. Live attenuated vaccines - subclinical / mild illness mimicking the disease
   - Local (igA) and humoral (igG) immunity
   - *Rapid immunity development*
   - Serious illness in immunocompromised individuals

2. Killed vaccines / subunit / toxoids
   - Antigens without the infectivity
   - Might require boosters
   - Adjuvant w/ toxoids

- Polysacc vaccines can be conjugated to protein vaccines***?

3. Recombinant vaccines

-DNA recombinant technology

- Attenuates microorganism
- **HEP B VACC

4. ADsorbed vaccines

- Vaccine mixed w/ inorganic salts to slow down absorption and longer lasting immunity
- Tetanus, diphtheria

5. Conjugate

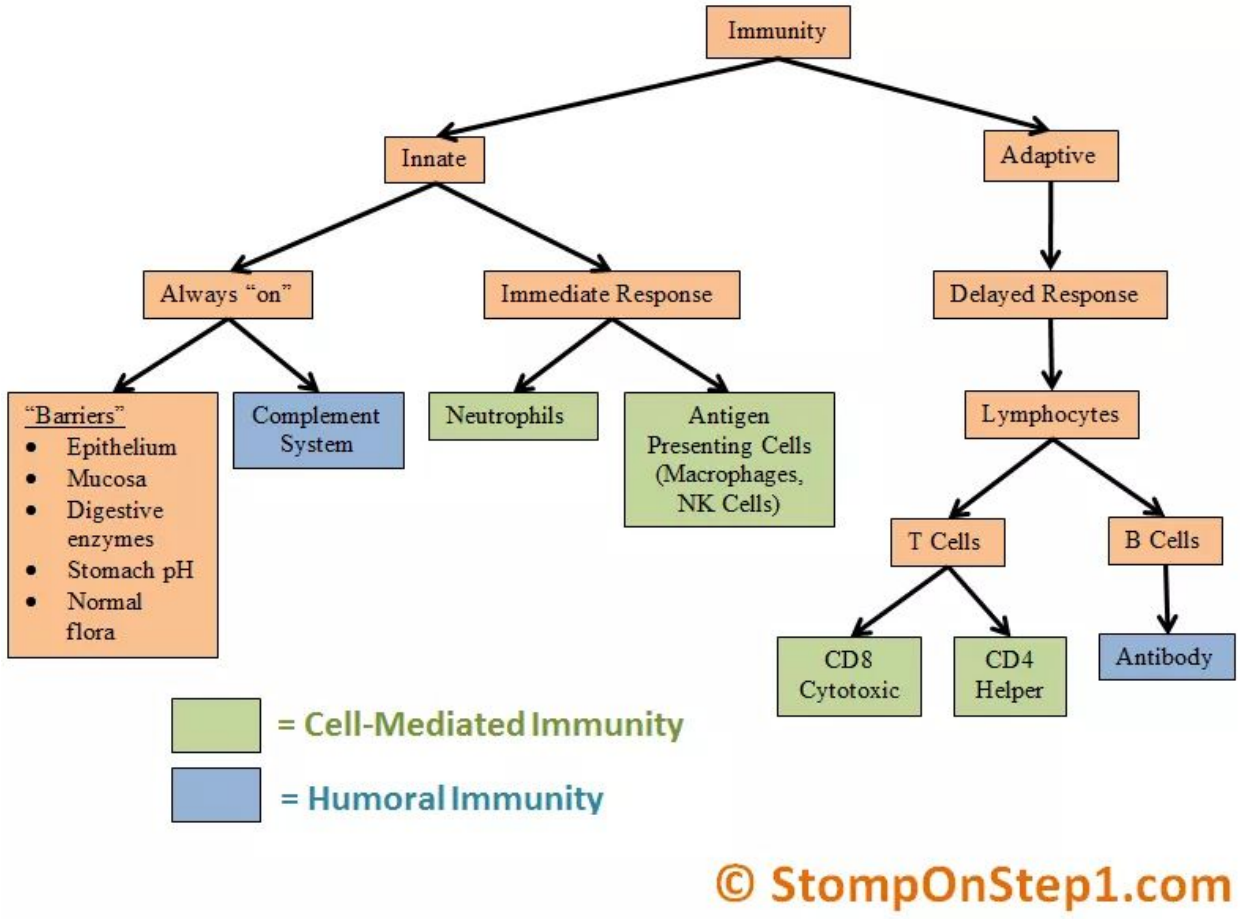
- *Designed for poorly antigenic microO*
- Conjugate antigen of interest to immunogenic, non-toxic protein?????????
- Haemophilus influenzae type b

6. Combined vaccines

- For ease of administration

7. Combined active/passive

- Immediate protection after possible exposure to microbe
- Hyperimmune igs and vaccine injected in DIFFERENT SITES
- Tetanus, hep B, rabies



Antibiotic resistance
> first antibiotic 1929
> sir Alexander Fleming
> penicillin used to treat staphyl/strept ococci in 1946
> resistance developed almost immediately

Antibiotic therapy
-Effective chemo depends on selective toxicity
(effective on toxins, does not affect host)
-exploits pathogen processes not seen in hosts (cell walls,metabolism etc.)
- route of administration
-monitoring effects
-adverse effects (on GI tract, kidneys , renal system, liver etc)

**** 3 MAJOR WAYS OF RESISTANCE**

1. **ALTERATION IN DRUG TARGET**
2. **PRODUCTION OF INACTIVATING ENZYMES**
3. **DECREASED UPTAKE OF ANTIBIOTIC**

RESISTANCE OCCURS WHEN

1. When a susceptible organism no longer has antibiotic agents
    - Due to intrinsic reasons = characteristics of microO, antibiotics mechanism of action
    - Acquired = new / added (due to selection or mutation in bacteria) VERTICAL evolution and exchange in genetic material (HORIZONTAL evolution)

The chromosome's role in antibiotic resistance

1. **Mutations lead to change in site of antibiotic target !!! PROTEIN FOR BACTERIAL STILL WORKS**
2. **Regulatory genes - turn on alternative paths/ turn on efflux mechanisms**
3. **Change in cell permeability**

Decreasing antimicrobial resistance

1. Withhold antibiotics
2. Use narrow spectrum antibiotics
3. Base decision on broadness of spectrum on severity
4. Prevention of infection - hygiene, HANDWASHING
5. Education - when needed, how to take, proper duration !!
6. EARLIER detection of therapeutic failure = suitable for patients w/ antibiotic resistance pathogens