Disease Transmission and Epidemiology

I. Introduction

A. Disease-causing microorganisms are called pathogens.

B. Pathogenic microorganisms have special properties that allow them to invade the human body or produce toxins.

C. When a microorganism overcomes the body's defenses, a state of disease results.

II. Pathology, Infection, and Disease

A. Pathology is the scientific study of disease.

B. Pathology is concerned with the etiology (cause), pathogenesis (development), and effects of disease.

C. Infection is the invasion and growth of pathogens in the body with or without disease.

D. A host is an organism that shelters and supports the growth of pathogens.

E. Disease is an abnormal state in which part or all of the body is not properly adjusted or is incapable of performing normal functions.

III. Normal, Transient and Opportunistic Organisms

1. Animals, including humans, are usually germ-free in utero.

2. Microorganisms begin colonization in and on the surface of the body soon after birth.

3. Microorganisms that establish permanent colonies inside or on the body without producing disease make up the normal microbiota.

4. The transient microbiota is composed of microbes that are present for various periods and then disappear.

IV. Relationships Between the Normal Microbiota and the Host

1. The normal microbiota can prevent pathogens from causing an infection; this phenomenon is known as microbial antagonism.

2. The normal microbiota and the host exist in symbiosis (living together).

3. Three types of symbiosis are commensalism (one organism benefits and the other is unaffected), mutualism (both organisms benefit), and parasitism (one organism benefits and one is harmed).

Opportunistic Organisms

Opportunists (opportunistic pathogens) do not cause disease under normal conditions but cause disease under special conditions.

Cooperation Among Microorganisms

In some situations, one microorganism makes it possible for another to cause a disease or to produce more severe symptoms; this is called cooperation.

V. Normal Human Microbiota

A. Harmless/helpful bacteria always there

B. Mouth
1. Strep. Sanguis, S. mutans, Leptotrichia buccalis
2 . Anaerobic acids cause cavities

C. Digestive Tract (stomach and colon)
1. In stom acid – none. Some Under protective mucus, Colon – E. coli, Strep fecalis, Clostridium
2. Inhibition, Cellulose digestion, vitamin production
3. Imbalances cause distress, some foods cause flatus. Ulcers. Perforations!

D. Respiratory Tract (Bronchi and Lungs)
1. Usually none
2. Ciliary Flushing
3. Growth is a problem

E. Skin
1. Staph epidermidis, S. aureus, M. luteus
2. Prevent pathogens
3. Staph infections

F. Urogenital Tract
1. Sterile

G. Blood/ Internal Tissues
1. Sterile

II. Infectious Disease

A. Factors influencing transmission
1. Invasiveness – ease of invasion
2. Pathogenicity – degree of disease causing ability
3. Virulence – Severity of Disease
4. Opportunistic Pathogens – normal flora given an opportunity

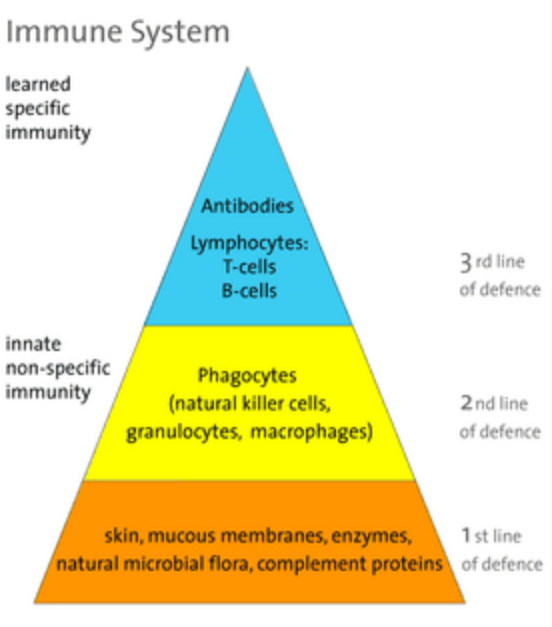
B. How Microbes cause disease

1. Transmission –
a. Common entry points:
1) Resp. Openings
2) Eyes
3) Dig. Tract
4) Urogenital Tract

2. Invasion of specific tissue– organisms are tissue specific

3. Cause Symptoms – some sort of problem
    a. Use of host nutrients
    b. Digestion of tissue – Strep throat
    c. Toxins
        1) Endotoxins – inside cell, released when they die, usually weak. E. coli
        2) Exotoxins – secreted from live cells, survive sterilization, potent Salmonella

III. Host response



Innate resistance

Defn. Response which requires no understanding of organism, immediate

Mechanism:

Cellular response at focal point

1)) Histamines – increase bloodflow to infection

2)) Interferon – blocks infection of healthy cells by a virus

Phagocytosis – monocytes locate and eat invaders

Benefits

Fast response, broad spectrum, localized attack

Drawbacks

Slow to kill, poor descrimination (mistakes) too localized, limited resources

Acquired Resistance

1) Defn. Response which requires the identification of an organism, learned

2) The players:

Macrophage – phagocytotic cell
Helper T-cell – recognize antigen (non-self)
B-Cell – produce antibodies
Killer T-cell – destroy tagged antigen

Mechanism

antigen recognition

1)) Macrophage ingests antigen
2)) Macro digests antigen and puts it's surface proteins on cell memb.
3)) Macro presents pro. To a Helper T cell. (thymus)
4)) Helper T cell analyses the antigens proteins and identifies the Antigenic determinants

Antibody production

1)) Helper T cell calls for an unassigned B Cell which can make an antibody for that determinant.
2)) The Helper T instructs the B cell to rapidly divide to form many plasma cells of the same type.
3)) The plasma cells begin producing antibodies
a)) capable of sticking to any antigen with this specific determinant
b)) Function for 1 – 2 weeks
4)) Some of the B cells survive for months – lifetime as memory cells

Marking and death

1)) Antibodies circulate in blood, stick to antigen. Marked for death.
2)) Killer T cells recognize the marked cells and burst them w/ enzymes

Antibodies

a) Groups

Type	# Antigen sites	Location	Function

IgG	**2**	**Blood, lymph , fetus**	**Daily maintenance**
IgM	**10**	**Blood and Lymph**	**Fights specific infection**
IgA	**4**	**Secretions (tears, saliva, milk, lungs, dig tract.**	**Maintenance and specific infection**
IgE	**2**	**Blood, lymph**	**Involved specifically with allergies (parasites?)**

How HIV and AIDS foil your defenses

HIV vs. AIDS

Mechanism of attack

Enters Blood

1) Body fluids -

b) Invasion of Host cell

c) Destruction of Host cell

3) Results of Viral Actions

a)

b)

c)

d)

4) Common symptoms

a) Opportunistic infections

1) Toxoplasmisis encephalitis

2) pneumocystitis

b) Cancer

1)Kaposis sarcoma

2) Lymphoma

5) Testing

a) Antibody presence

False positive

b)T cell count

c) Viral load

6) Treatments

1) AIDS Cocktail

a) AZT

b) 3TC

c) Protease inhibitor

2) Difficulties

a) Drug effectiveness

b) No proof of cure

c) Vaccine development