## **Immune Topics**

- 1) microbes
- 2) lymphatic system
- 3) immunity
- 4) allergy
- 6) diseases

# Pathogens

= tiny, invisible disease causing agents, kill by fast growth

bacteria

- single cell prokaryote with unique cell wall
- antibiotics breakdown cell wall, eg penicillin
- strep, TB, gonorrhea, syphilis

#### virus

- chemicals, acellular, come alive when reproducing
- colds, flu, measles, chicken pox, polio, AIDS, herpes

### prions

- protein only infectious particles / chemicals
- neural degeneration, eg CJD, mad cow disease, scrapie

# **Lymphatic Components**

- 1) lymphatic vessels drainage
- 2) primary lymphatic organs
  - a) red bone marrow B & T cells
  - b) thymus T cells
- 3) secondary lymphatic organs
  - a) spleen clean blood, fight infection
  - b) lymph nodes matures B & T cells
  - c) tonsils fight infection in throat
  - d) Peyer's patch intestines
  - e) appendix drain intestines; can burst

# Immunity

### = ability to fight infection

### infection

= attack by infectious microbes\* (pathogens)

### path of infection:

- 1) infection: microbes enter and multiply
- 2) immunity (3 levels of body defence)
- 3) result: survival (intact or perm. injury) or death

### \*not all microbes are pathogens

# **Innate vs Adaptive Def.**

#### innate defences:

- = regular defences (1st, 2nd)
- non-specific to pathogen
- always available; born with it
- no "memory"
- local effects

#### adaptive defences:

- = special defences (3rd)
- specific to pathogen
- acquired as needed
- memory present
- systemic effects

# **3 Defence Levels**

**1st level: innate defences (external)** - physical & chemical defences - eg skin, tears, acids, mucus **2nd level: innate defences (internal)** - cells & processes - eg inflammation, complement sys. **3nd level:** acquired defences - specific to pathogen - eg antibody-mediated immunity cell-mediated immunity

### 1st Level - innate, external defences

innate defence

- protective physical & chemical barriers

review:

- 1) skin (oil)
- 2) tears, saliva, earwax (lysozyme)
- 3) mucus
- 4) digestive & vagina acids
- 5) vomiting, urination, defecation
- 6) resident bacteria (mouth, anus)

### 2nd Level - innate, internal defences

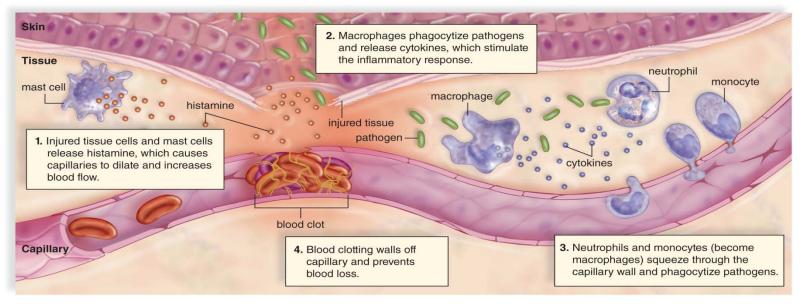
innate defence

- protective cells & processes
- 1) phagocytes (macrophages & WBC)
- 2) inflammation
- **3) complement system (protective proteins)**
- 4) natural killer cells
- 5) interferons

## **Inflammation Steps**

- 1) mast cells release histamine
- 2) macrophages ingest microbes, release cytokines
- 3) WBC ingest pathogens
- 4) blood vessel wall clot, reduce blood loss

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## **NK Cells & Interferon**

### NK cells:

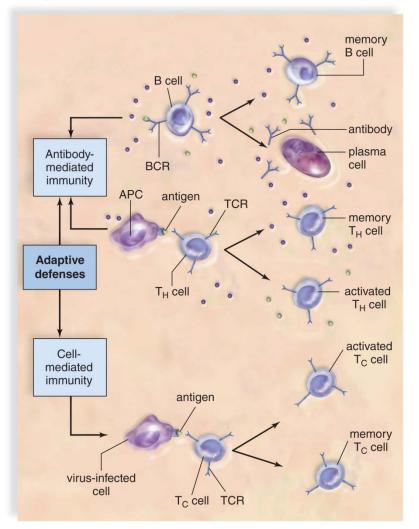
- not phagocyte
- prod. perforin to perforate microbe membrane
- kills bacteria

#### **Interferon:**

- protein by infected cells to protect non-infected cells
- "interferes" with microbe reproduction
- kills viruses, eg hepatitis C

## **3rd Level - adaptive defences**

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- 2 types:
- 1) antibody-mediated (B cell)
- 2) cell-mediated (T cell)

#### antigens:

chemically recognized
pieces of bacteria,
virus, mold, or worms

# **Antibody-Med. Immunity**

### = humoral immunity (B cells)

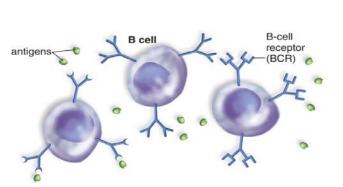
### **B** Cell types:

- 1) plasma B cells make antibodies, destroy pathogens
- 2) memory B cells remember, future fast response

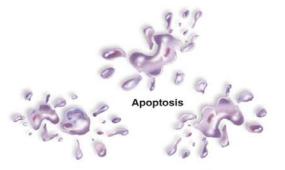
#### target pathogens:

 intact bacteria, bacterial toxins, RBC, free viruses

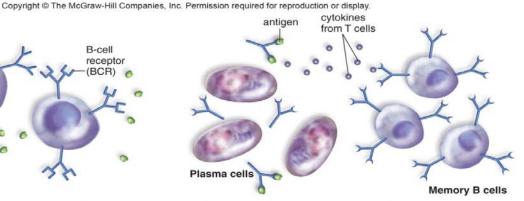
# **Antibody-Med. Immunity Steps**



a. Activation–When a B-cell receptor binds to an antigen activation occurs.



c. **Apoptosis**–Apoptosis, or programmed cell death, occurs to plasma cells left in the system after the infection has passed.



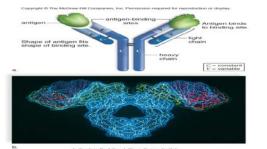
 b. Clonal expansion-During clonal expansion, cytokines secreted by helper T cells stimulate B cells to clone mostly into plasma cells or memory cells.

a) activate: antigen binds to B cell
b) clone (copies are made)

a) plasma B cell
b) memory B cells

c) destroy: antibodies attack

pathogens (5 ways)



## Antibodies

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Table 7.1	Antibodies	
Class	Presence	Function
lgG	Main antibody type in circulation; crosses the placenta from mother to fetus	Binds to pathogens, activates complement, and enhances phagocytosis by white blood cells
IgM	Antibody type found in circulation; largest antibody; first antibody formed by a newborn; first antibody formed with any new infection	Activates complement; clumps cells
lgA	Main antibody type in secretions such as saliva and milk	Prevents pathogens from attaching to epithelial cells in digestive and respiratory tract
lgD	Antibody type found on surface of immature B cells	Presence signifies readiness of B cell
IgE	Antibody type found as antigen receptors on mast cells in tissues	Responsible for immediate allergic response and protection against certain parasitic worms

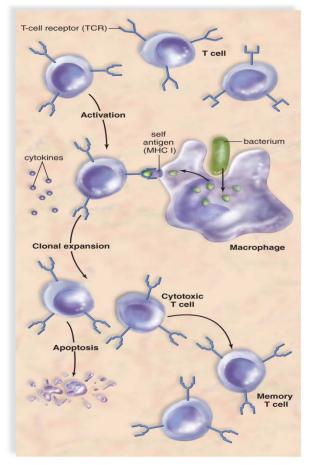
### Summarize 5 Ig's: location & target

eg IgE - mast cells - allergy & parasitic worms

note: Ig blood levels indicate source of infection

## **Cell-Mediated Immunity**

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= cellular immunity (T cells)

**T Cell types:** 

- 1) cytotoxic T killer
- 2) helper T activate B cells
- 3) memory T remember

target: virus, parasite, cancer, grafts & transplants

# **Acquired Immunity**

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a. Antibodies (IgG) cross the placenta.



b. Antibodies (IgG, IgA) are secreted into breast milk.



c. Antibodies can be injected by a physician. a: © John Lund/Drew Kelly/Blend Images/Corbis RF; b: © Digital Vision/Getty RF; c: © Photodisc Collection/Getty RF

#### active immunity

vaccines (weakened pathogens) -> self prod.
of antibodies

passive immunity
antibodies are given &
pass thru:
- placenta

- mother's milk
- injections

## **Allergy - 3 Levels**

### = hypersensitivity to ordinary substances, hair, food, pollen

level	time	IG	example .
1) immed. allergic response	sec	IgE	hay fever food allergy
2) anaphy- lactic shock	sec	IgE	bee stings pennicilin shots
3) delayed allergic resp.	days	T cells	poison ivy TB test

# **Rheumatoid Arthritis**

### bumpy knuckles:

- 1) B cell antibodies
- destroy synovial membranes
- 2) inflammation
- more damage & swelling
- 3) fingers, wrists, toes, joints
- stiffen and painful
- 4) cartilage fuse
- joint deformity, loss of motion
- 5) progressively worse

treatment: pain meds & mild exercise





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# **Tissue Transplant Issues**

- 1) organ bank systems data sharing
- 2) scarcity of donor tissues
- 3) cross-matching: accept donor tissues
- 4) surgery easy
- 5) recipient immune system
  - a) accept tissue antigens
  - b) life-long immuno-suppressants
  - cortico-steroids: suppress inflammation
  - cytotoxic meds: kill fast growing cells
  - long term: impaired immune system  $\rightarrow$  easy to get infected
  - balance: prevent rejection of transplant; preserve own immune function

## **Immune Diseases**

**Describe the cause & effects of:** 

- 1) allergies
- 2) autoimmune disorders
  - a) lupus erythematosus
  - b) rheumatoid arthritis
- 3) immune deficiency
  - a) AIDS