

BIOL 240: General Microbiology

Spring 2020 **WM, 4/29-5/4**

<http://accounts.smccd.edu/staplesn/biol240/>

1. **ONLINE Experiment 16/Bacterial transformation due THIS WEEK!**
-- Complete the questions by **Monday, 5/4!**
2. **NEXT WEEK: Labster Experiment ("17") on Identification of Bacterial Unknown. Due Fri., 5/8.**
3. **QUIZ #7: Final quiz – Experiments 16, 17; Chapters 12, 13, 15, 16, 17.**
– Will open next Wed. **DUE, Monday, 5/11.**
4. **Extra Credit: STEM SPEAKERS, are DONE and posted at the STEM CANVAS page. ALL DUE by THIS FRIDAY, 5/1!!!!**

1

Ch. 16: Nonspecific Host Defenses

OBJECTIVES: Students should be able to:

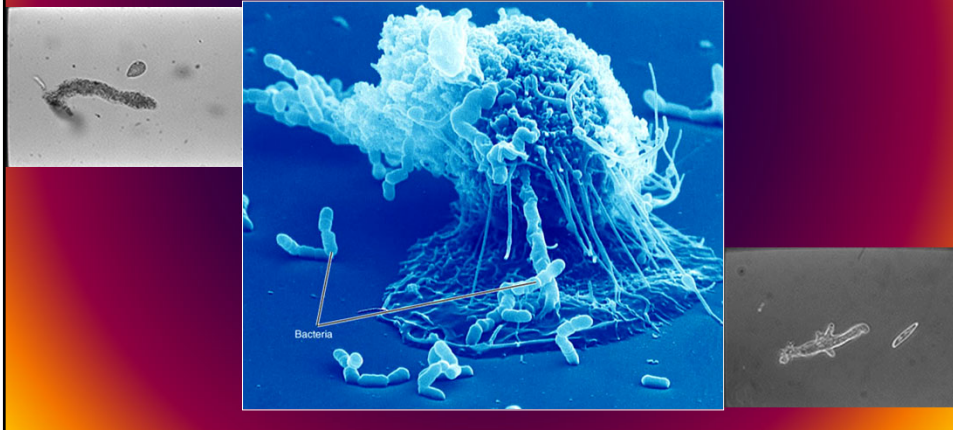
1. **Ch. 16:** Listing examples, explain how **mechanical, chemical, and microbial barriers** protect a host animal. ***
Define the function of each **granulocyte!**
2. Describe the sequence of events that lead to **inflammation** and/or **fever**, and how these processes protect a host from infection.
3. Compare and contrast how **Complement** protects a host from bacteria, and how **Interferons** protect a host from viral infection. Describe 3 major effects of **complement** activation, and 3 effects of **interferon** production.

❖ **Objectives are your HOMEWORK between classes!!! **Read, Review, Draw!!**

2

Chapter 16

Nonspecific Defenses of the Host



3

Nonspecific Defenses of the Host

1. **Susceptibility** = Lack of resistance to a disease.
2. **Resistance** = Ability to ward off disease.
3. **Nonspecific resistance** = Defenses against any pathogen.
4. **Specific resistance** = Immunity, resistance to a specific pathogen.

4

Host Defenses

Figure 16.1

Innate (Nonspecific) Immunity		Adaptive (Acquired) Immunity (Chapter 17)
First line of defense	Second line of defense	Third line of defense
<ul style="list-style-type: none"> • Intact skin • Mucous membranes and their secretions • Normal microbiota 	<ul style="list-style-type: none"> • Natural killer cells and phagocytic white blood cells • Inflammation • Fever • Antimicrobial substances 	<ul style="list-style-type: none"> • Specialized lymphocytes: T cells and B cells • Antibodies
<u>BARRIERS</u>	<u>NONSPECIFIC ATTACK</u>	<u>SPECIFIC ATTACK</u>

5

16.1) Mechanical Factors

1. Skin

- a) Epidermis consists of tightly packed cells with
- b) **Keratin**, a protective protein

2. Mucous membranes

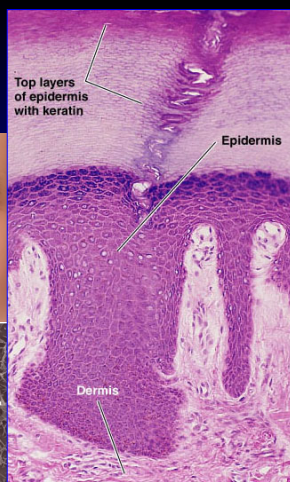
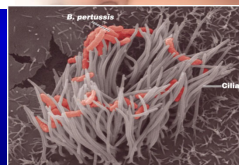
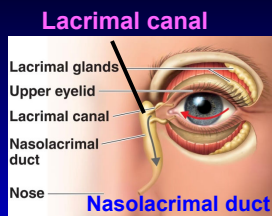
- 3. **Ciliary escalator:** Microbes trapped in mucus are transported away from the lungs

- 4. **Lacrimal apparatus:** Washes eye; **lysozyme**

- 5. **Saliva:** Washes microbes off; **lysozyme**

- 6. **Urine:** Flows out

- 7. **Vaginal secretions:**
 - Flow out



Computer-enhanced 10 μm

6

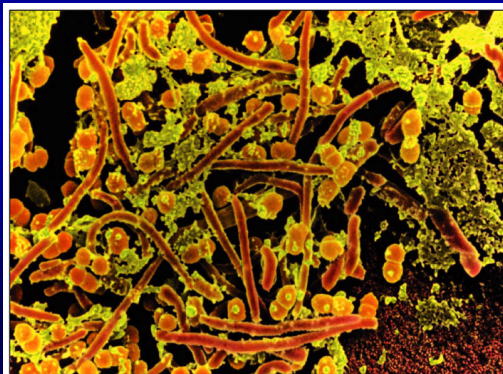
16.2) Chemical Factors

1. Fungistatic fatty acid in sebum
2. Low pH (3-5) of skin, vaginal secretions
3. Lysozyme in perspiration, tears, saliva, urine, and tissue fluids
4. Low pH (1.2-3.0) of **gastric juice (HCl)**
5. Transferrins in blood bind & bind iron
6. NO (nitrous oxide) inhibits ATP production

7

16.3) Normal Microbiota

- Microbial antagonism / competitive exclusion:
 - *Normal microbiota compete with pathogens.*



MOUTH

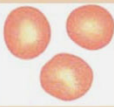
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8

16.4) Formed Elements In Blood

Table 16.1 Formed Elements in Blood

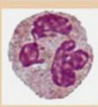
I. Erythrocytes (Red Blood Cells)
4.8-5.4 million per μL or mm^3
Function: Transport of O_2 and CO_2




LM 4 μm

II. Leukocytes (White Blood Cells)
5000-10,000 per μL or mm^3


A. Granulocytes (stained)

- 1. Neutrophils (PMNs)**
(60-70% of leukocytes)
Function: Phagocytosis


LM 4 μm

- 2. Basophils (0.5-1%)**
Function: Production of histamine


LM 3 μm

- 3. Eosinophils (2-4%)**
Functions: Production of toxic proteins against certain parasites; some phagocytosis


Differential White Cell Count

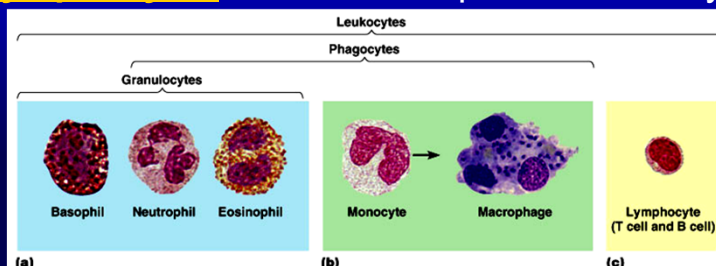
- Percentage of each type of white cell in a sample of 100 white blood cells

Neutrophils, PMNs	60-70%
Basophils	0.5-1%
Eosinophils	2-4%
Monocytes	3-8%
Lymphocytes	20-25%

9

A. White Blood Cells

- 1. Neutrophils:** Phagocytic
- 2. Basophils:** Produce histamine (IgE-R); ~**mast cells** in tissues.
- 3. Eosinophils:** Toxic to parasites, some phagocytosis
- 4. Monocytes:** Phagocytic as mature **macrophages**
 - a) Fixed macrophages:** in lungs, liver, bronchi
 - b) Wandering macrophages:** roam tissues
- 5. Natural Killer Cells:** destroy target cells. Like CTLs.
- 6. Lymphocytes:** Involved in specific immunity (T, B)



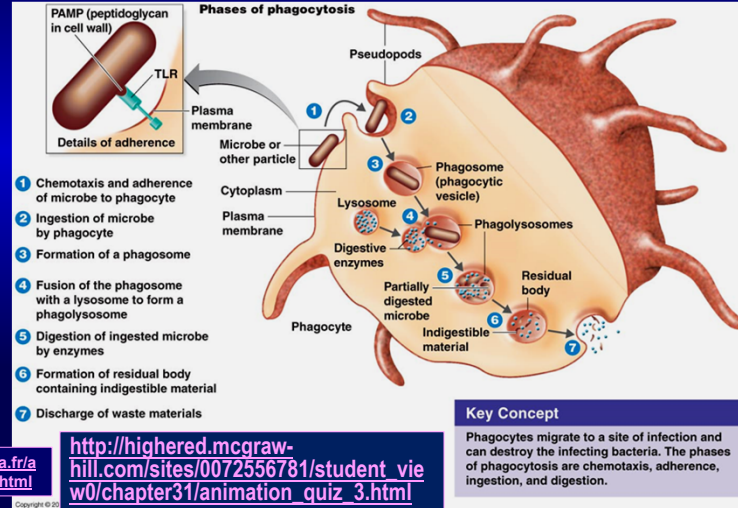
<http://www.sp.uconn.edu/~terry/Common/phago053.html>

10

B. Phagocytosis

Figure 16.8a

- **Phago** = eat; **Cyte** = cell (“cell eating”)
- Ingestion of microbes or particles by a cell, performed by phagocytes.



http://www.edumedia.fr/a/82_l2-phagocytosis.html

http://highered.mcgraw-hill.com/sites/0072556781/student_vie_w0/chapter31/animation_quiz_3.html

11

Microbial Evasion of Phagocytosis



• Inhibit adherence: M protein, capsules	<i>Streptococcus pyogenes</i> , <i>Strep. pneumoniae</i>
• Kill phagocytes: Leukocidins	<i>Staphylococcus aureus</i>
• Lyse phagocytes: <u>Membrane attack complex</u>	<i>Listeria monocytogenes</i>
• Escape phagosome	<i>Shigella</i> species (spp.)
• Prevent phagosome-lysosome fusion	HIV
• Survive in phagolysosome	<i>Coxiella burnetti</i>

12

16.5) Inflammation

1. Redness – *rubor* [latin]
2. Pain - *dolor*
3. Heat - *calor*
4. Swelling (edema) - *tumor*
5. Acute-phase proteins activated (complement, cytokine, kinins)
6. Vasodilation (histamine, kinins, prostaglandins, leukotrienes)
7. Margination and emigration of WBCs
 - *Diapedesis* – leave BV and enter tissues
8. Tissue repair

13

Chemicals Released by Damaged Cells

• Histamine	Vasodilation, increased permeability of blood vessels
• Kinins	Vasodilation, increased permeability of blood vessels
• Prostaglandins	Intensify histamine and kinin effect
• Leukotrienes	Increased permeability of blood vessels, phagocytic attachment

14

Inflammation

(a) Tissue damage

- Chemicals such as histamine, kinins, prostaglandins, leukotrienes, and cytokines (represented as blue dots) are released by damaged cells.
- Blood clot forms.
- Abscess starts to form (dark yellow area).

(b) Vasodilation and increased permeability of blood vessels

- Margination—phagocytes stick to endothelium.
- Diapedesis—phagocytes squeeze between endothelial cells.
- Phagocytosis of invading bacteria.

(c) Phagocyte migration and phagocytosis

(d) Tissue repair

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http://faculty.riohondo.edu/rbethel/videos/micro_inflammation.swf

- <http://www.studiodaily.com/2006/07/cellular-visions-the-inner-life-of-a-cell/>
- Narrated, detailed (super speed): <http://multimedia.mcb.harvard.edu>
- Discovery Ch. Special, Curiosity: "Battlefield Cell"!!! http://www.dailymotion.com/video/x150im3_battlefield-cell_shortfilms

15

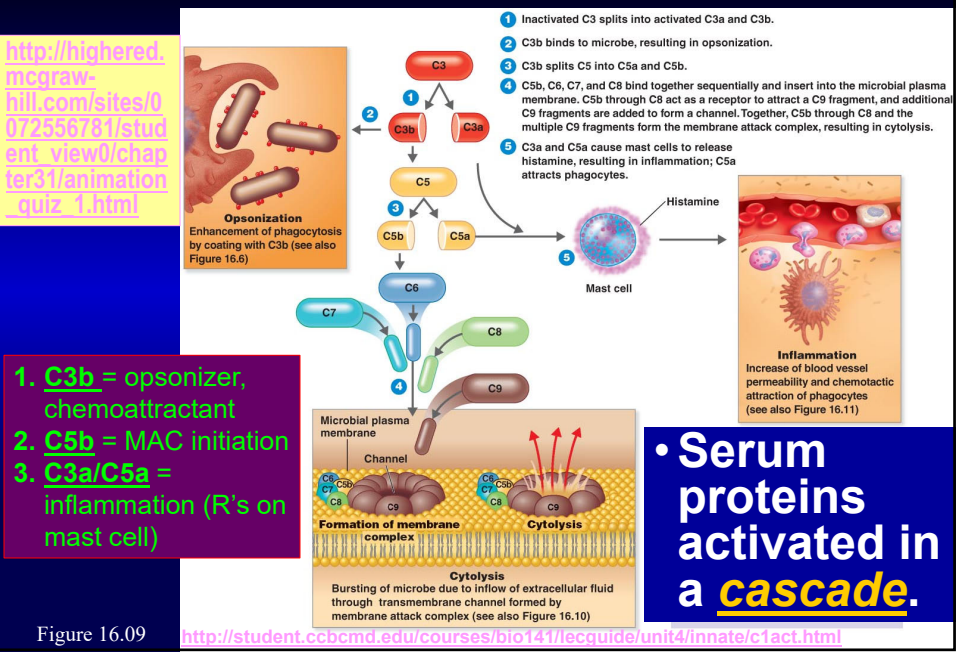
16.6) Fever: Abnormally High Body Temperature

- Hypothalamus normally set at 37°C
 - Body's thermoregulatory center ("thermostat")
- Gram-negative endotoxin cause phagocytes to release interleukin 1
- Hypothalamus releases prostaglandins that reset the hypothalamus to a high temperature
- Body increases rate of metabolism and shivering to raise temperature
- When **IL-1** is eliminated, body temperature falls. (**Crisis** = turning point)

http://faculty.riohondo.edu/rbethel/videos/micro_inflammation.swf

16

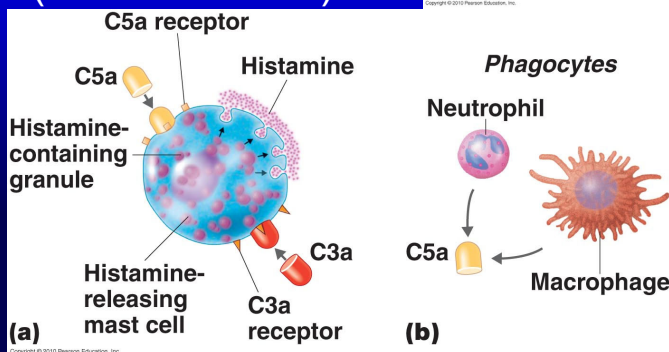
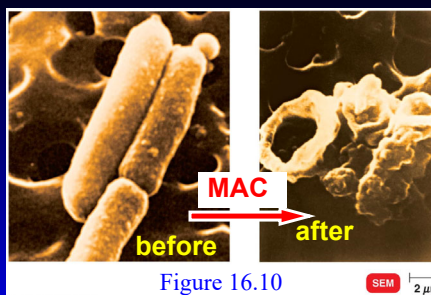
16.7) The Complement System:



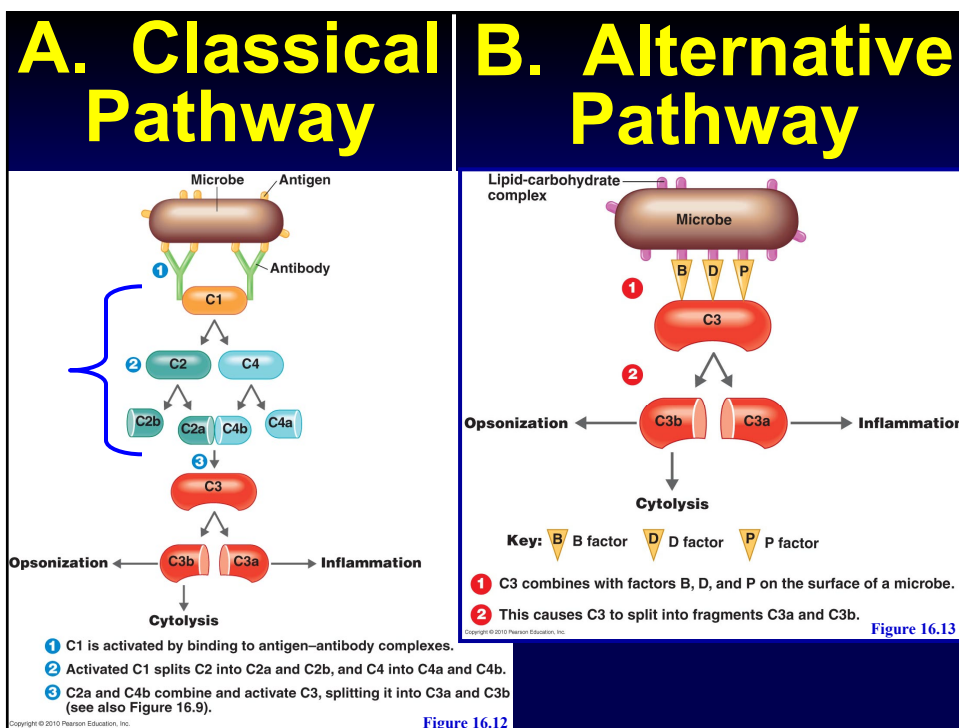
17

Effects of Complement Activation

- Opsonization** or immune adherence: enhanced phagocytosis
- Membrane attack complex (MAC) cytolysis**
- Attract phagocytes
• (chemo-TAXIS!)



18



19

Some Bacteria Evade Complement

1. Capsules prevent **C** activation.
2. Surface lipid-carbohydrates prevent **MAC** formation.
 - Too long to reach PM surface!
3. Enzymatic digestion of **C5a** (**C5a protease**).

http://highered.mheducation.com/sites/0072507470/student_view0/chapter22/animation_activation_of_complement.html

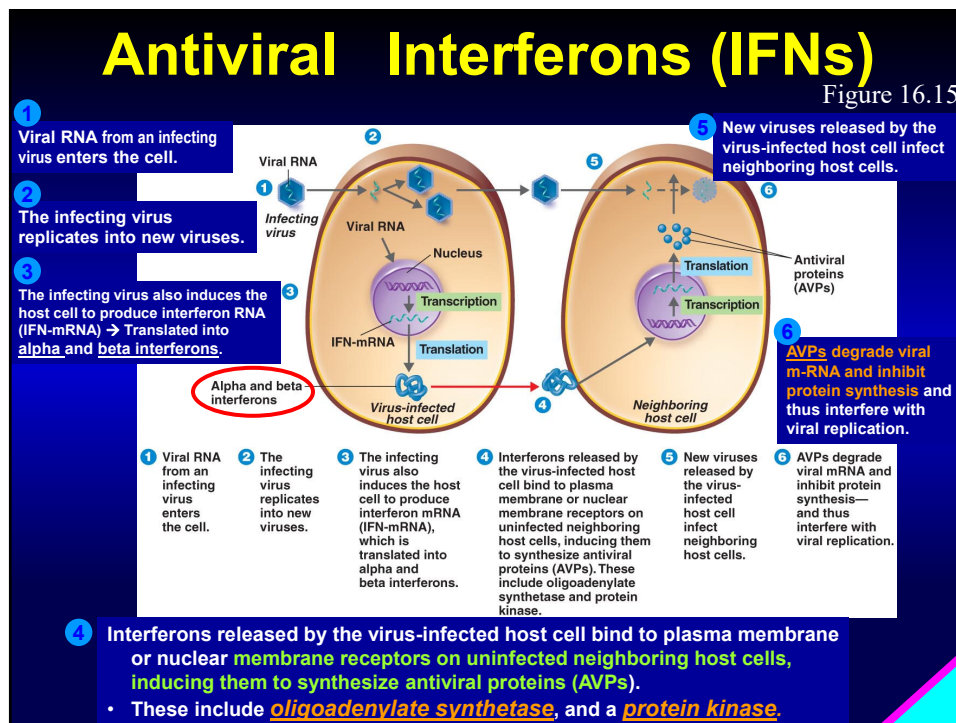
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16.8) Interferons (IFNs)

- **Alpha IFN** & **Beta IFN**: Cause cells to produce *antiviral proteins* that inhibit viral replication.
- **Gamma IFN**: Causes neutrophils and macrophages to *phagocytize bacteria*.

http://highered.mcgraw-hill.com/sites/0072556781/student_view0/chapter31/animation_quiz_2.html

21



22