Chapter 16

Innate Immunity: Nonspecific Defenses of the Host

Fever

- Advantages
  - Increases transferrins
  - Increases IL-1 activity
  - Produces interferon

The Concept of Immunity

- 16-1 Differentiate innate and adaptive immunity.
- 16-2 Define Toll-like receptors.

The Concept of Immunity

- Susceptibility: lack of resistance to a disease
- Immunity: ability to ward off disease
- Innate immunity: defenses against any pathogen
- Adaptive immunity: immunity or resistance to a specific pathogen

An Overview of the Body's Defenses

The Concept of Immunity

- Host Toll-like receptors (TLRs) attach to pathogen-associated molecular patterns (PAMPs)
- TLRs induce cytokines that regulate the intensity and duration of immune responses

First Line of Defense: Skin and Mucous Membranes

- 16-3 Describe the role of the skin and mucous membranes in innate immunity.
- 16-4 Differentiate physical from chemical factors, and list five examples of each.
- 16-5 Describe the role of normal microbiota in innate immunity.

Physical Factors

- Skin
  - Epidermis consists of tightly packed cells with
• Keratin, a protective protein

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13 Physical Factors
• Mucous membranes
• Mucus: traps microbes
• Ciliary escalator: transports microbes trapped in mucus away from the lungs

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16 Physical Factors
• Lacrimal apparatus: washes eye
• Saliva: washes microbes off
• Urine: flows out
• Vaginal secretions: flow out

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18 Chemical Factors
• Fungistatic fatty acid in sebum
• Low pH (3–5) of skin
• Lysozyme in perspiration, tears, saliva, and urine
• Low pH (1.2–3.0) of gastric juice
• Low pH (3–5) of vaginal secretions

19 Normal Microbiota and Innate Immunity
• Microbial antagonism/competitive exclusion: normal microbiota compete with pathogens or alter the environment
• Commensal microbiota: one organism (microbe) benefits, and the other (host) is unharmed
  • May be opportunistic pathogens

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• Identify one physical factor and one chemical factor that prevent microbes from entering the body through skin and mucous membranes. 16-3
• Identify one physical factor and one chemical factor that prevent microbes from entering or colonizing the body through the eyes, digestive tract, and respiratory tract. 16-4
• Distinguish microbial antagonism from commensalism. 16-5

21 Second Line of Defense
• 16-6 Classify leukocytes, and describe the roles of granulocytes and monocytes.
• 16-7 Define differential white blood cell count.
• 16-8 Differentiate the lymphatic and blood circulatory systems.

24 Differential White Cell Count
• Percentage of each type of white cell in a sample of 100 white blood cells

26 The Lymphatic System

28
• Compare the structures and function of monocytes and neutrophils. 16-6
• Describe the six different types of white blood cells, and name a function for each type. 16-7
• What is the function of lymph nodes? 16-8

29 Second Line of Defense
• 16-9 Define phagocyte and phagocytosis.
• 16-10 Describe the process of phagocytosis, and include the stages of adherence and ingestion.
• 16-11 Identify six mechanisms of avoiding destruction by phagocytosis.

32 Phagocytosis
• Phago: from Greek, meaning eat
• Cyte: from Greek, meaning cell
• Ingestion of microbes or particles by a cell, performed by phagocytes

35 Phagocytosis

36 Microbial Evasion of Phagocytosis

38 • What do fixed and wandering macrophages do? 16-9
• What is the role of TLRs in phagocytosis? 16-10
• How does each of these bacteria avoid destruction by phagocytes? Streptococcus pneumoniae, Staphylococcus aureus, Listeria monocytogenes, Mycobacterium tuberculosis, Rickettsia 16-11

39 Second Line of Defense

• 16-12 List the stages of inflammation.
• 16-13 Describe the roles of vasodilation, kinins, prostaglandins, and leukotrienes in inflammation.
• 16-14 Describe phagocyte migration.
• 16-15 Describe the cause and effects of fever.

40 Inflammation

• Activation of acute-phase proteins (complement, cytokine, and kinins)
• Vasodilation (histamine, kinins, prostaglandins, and leukotrienes)
• Redness
• Swelling (edema)
• Pain
• Heat

41 Chemicals Released by Damaged Cells

42 Phagocyte Migration and Phagocytosis

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46 Fever

• Abnormally high body temperature
• Hypothalamus is normally set at 37°C
• Gram-negative endotoxins cause phagocytes to release interleukin-1 (IL-1)
• Hypothalamus releases prostaglandins that reset the hypothalamus to a high temperature
• Body increases rate of metabolism, and shivering occurs, which raise temperature
• Vasodilation and sweating: body temperature falls (crisis)

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• What purposes does inflammation serve? 16-12
• What causes the redness, swelling, and pain associated with inflammation? 16-13
• What is margination? 16-14
• Why does a chill indicate that a fever is about to occur? 16-15

48  Antimicrobial Substances
• 16-16 List the major components of the complement system.
• 16-17 Describe three pathways of activating complement.
• 16-18 Describe three consequences of complement activation.

49  The Complement System
• Serum proteins activated in a cascade
• Activated by
  • Antigen–antibody reaction
  • Proteins C3, B, D, P and a pathogen

50  The Complement System
• C3b causes opsonization
• C3a + C5a cause inflammation
• C5b + C6 + C7 + C8 + C9 cause cell lysis

51  Effects of Complement Activation
• Opsonization, or immune adherence: enhanced phagocytosis
• Membrane attack complex: cytolysis
• Attract phagocytes

52  The Complement System
• C3b causes opsonization
• C3a + C5a cause inflammation
• C5b + C6 + C7 + C8 + C9 cause cell lysis

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58  Some Bacteria Evade Complement
• Capsules prevent C activation
• Surface lipid–carbohydrate complexes prevent formation of membrane attack complex (MAC)
• Enzymatic digestion of C5a

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• What is complement? 16-16
• List the steps of complementation activation via (1) the classical pathway, (2) the alternative pathway, and (3) the lectin pathway. 16-17
• Summarize the major outcomes of complement activation. 16-18

60  Antimicrobial Substances
16-19 Define interferons.
16-20 Compare and contrast the actions of IFN-α and IFN-β with IFN-γ.
16-21 Describe the role of iron-binding proteins in innate immunity.
16-22 Describe the role of antimicrobial peptides in innate immunity.

**Interferons (IFNs)**
- IFN-α and IFN-β: cause cells to produce antiviral proteins that inhibit viral replication
- IFN-γ: causes neutrophils and macrophages to phagocytize bacteria

**Innate Immunity**
- Transferrins
  - Bind serum iron

**What is interferon?** 16-19
**Why do IFN-α and IFN-β share the same receptor on target cells, yet IFN-γ has a different receptor?** 16-20
**What is the role of siderophores in infection?** 16-21
**Why are scientists interested in AMPs?** 16-22