Chapter 16 Innate Immunity: Nonspecific Defenses of the Host

The cond	cept of immunity
• Immu	unity – ability to protect against from microbes and their
	Includes protection against environmental agents such as pollen, drugs, chemicals, etc
0	Aka,
• Susce	eptibility – or lack of
• Two	general mechanisms of immunity
	() immunity
0	() immunity
	tive Immunity
0	Involve recognition of
	 Adapts of adjusts to fight a microbe
0	1.1
0	**
0	Involves white blood cells called ""
	B cells, T cells
 Innate 	e Immunity
	Always present and available to provide rapid response
0	
0	· . · · · ·
0	
	Skin, mucous membranes
0	of defense include cellular and molecular components
	 Inflammation, fever, phagocytic white blood cells
0	
	LINE OF DEFENSE: Skin and mucous membranes
Physical	factors
• Skin	
0	1 3
	 Removes overlying microbes
0	1
0	<u></u>
	■ In conditions, skin infections common
0	I ************************************
	 Some fungi can hydrolyze keratin in skin
0	
	Through burns, cuts, stabs, breaks
Muco	ous membranes
0	Inhibit of many microbes
	 Line GI tract, respiratory tract, genitourinary tract
0	Mucus – slightly fluid composed of
	• invading microbes
	■ Some pathogens can in mucus → <i>Treponema pallidum</i>

Lacrin	nal apparatus	
0	Manufactures and drains away	
0	Continual helps wash away microbes	
Saliva	, urine cleanses like	
	, urine cleanses like in nose inhaled air and traps microbes	
Cilia i	n respiratory tract move trapped microbes up into throat – "	
0	and speed up process	
Defeca	ation and also expel microbes	
nemical		
Sebun	n – substance in skin produced by in skin	
0	Forms film over skin	
0	Contain unsaturated acids	
	 Inhibits growth of some pathogens and fungi, lower pH (pH)
0	Some bacteria can sebum, lead to acne	
Sweat	, or perspiration, contains lysozyme – that breaks down cell wall	
0	Lysozyme also found in tears, saliva, tissue fluids, nasal secretions	
	c juice – mixture of, enzymes, mucus found in	_
0	Kills most bacteria and	
0	Some bacteria survive in food particles	
Norma	al protect microbioto vio " " (p. 424)	
	LINE OF DEFENSE	
ECOND	LINE OF DEFENSE	
ECOND ormed e Forme	LINE OF DEFENSE clements in blood cd elements – in blood	
ECOND ormed e Forme	clements in blood ed elements — in blood Leukocytes — ()	
ECOND ormed e Forme o wpes of w	clements in blood ded elements – in blood Leukocytes – () white blood cells	
ECOND ormed e Forme over of weather	LINE OF DEFENSE clements in blood cd elements – in blood Leukocytes – () white blood cells ophils	
ECOND ormed e Forme orpes of w Neutro	LINE OF DEFENSE clements in blood	
Formed e Forme orpes of w Neutro	lements in blood d elements — in blood Leukocytes — () white blood cells ophils Highly Active in stages of infection	
Formed e Forme over of we Neutro o	lements in blood de elements — in blood Leukocytes — () white blood cells ophils Highly Active in stages of infection Can leave bloodstream and move into to fight infection	
Formed e Forme vpes of w Neutro Basop	clements in blood ded elements —	
Formed e Forme over sof we have the composition of	lements in blood d elements — in blood Leukocytes — () white blood cells ophils Highly Active in stages of infection Can leave bloodstream and move into to fight infection hils Release, important in	
Formed e Forme vypes of w Neutro Basop Eosino	lements in blood de elements — in blood Leukocytes — () white blood cells ophils Highly Active in stages of infection Can leave bloodstream and move into to fight infection hils Release, important in ophils	
Formed e Formed o Vipes of W Neutro O Basop C Eosino	DLINE OF DEFENSE Column	
Formed e Formed e Very pes of w Neutro O Basop O Eosino	Column C	
Formed e Formed o Opes of w Neutro O Basop O Eosino	Column C	
Formed e Formed o Vipes of W Neutro O Basop O Eosino	Column C	
Formed e Formed e Formed o Opes of w Neutro O Basop O Eosino O O Dendr	lements in blood de elements —	
Formed e Formed e Formed o Vipes of W Neutro O Basop O Eosino O Dendr	Column C	
Formed e Formed e Formed o Vipes of W Neutro O Basop O Eosino O Dendr	lements in blood de elements —	
Formed e Formed e Formed o Vipes of W Neutro O Basop O Eosino O Dendr O Monoo	Column C	
Formed e Formed e Formed o Vipes of W Neutro O Basop O Eosino O Dendr O Monoo	lements in blood de elements —	

 Lymphocytes 		
Natural killer (NK)	cells, T cells, B cells	
NK cells kill	cells,	cells
 Any cell that 	ıt displays "	cells representation contains cells
	ell, not microbe inside	
 T cells, B cells play 	key role in	
• Leukocytosis –	in	in response to infection
 Can double, triple, 	quadruple	
• Leukopenia –	in	in response to infection
 Due to impairment 	of white blood cell	
• Differential white blood ce	ll count –	of WBC in blood
Phagocytes		
• Phagocytosis – ingestion of	f or	by a cell
• Phagocytes –	that perform	
o Neutrophils, macro		
Actions of phagocytic cells		
• During infection,	and	(macrophages) migrate
to infected area		
• inc	crease in initial stages of	infection
 Highly phagocytic 		
• As infection progresses,	dominate	
 Clean up remaining 	g live bacteria, clear up dead cel	1
The mechanism of phagocytosi	is	
• 4 main phases of phagocyte	osis	
 Chemotaxis, adhere 	ence, ingestion, digestion	
• Chemotaxis – chemical	of phagocytes to	microbes
 Attracted to microb 	ial products, damaged tissue ce	lls, various chemicals
• Adherence –	of phagocyte's plasma	a membrane to microbe
• Ingestion –	of microbe via plasi	ma membrane
 Projections of plasm 	na membrane called "	,,
 Microbe internalize 	ed in vesicle called	
Inside is aci	dic, pH 4	
• Digestion –	of microbe	
 Phagosome fuses w 	vith lysosomes →	
	contains many enzymes that	bacteria
Takes 10 to		
	material expelled fro	om cell
Microbial evasion of phagocyt		
• Inhibition of		
	ot, can't phagocyt	
	genes, capsules of S. pneumonio	ae
• Some are phagocytosed, bu	ıt	
 Leukocidins and str 	reptolysins kill phagocytes	

•		from phagosome
	0	Produce "membrane attack complexes"
	0	Live and replicate phagocyte
	0	Can escape from phagocyte by cell
		Listeria monocytogenes, Trypanosoma cruzi
•		
	0	val inside inside to replicate
	0	Mycobacterium tuberculosis can prevent fusion of phagosome
T	flamma	
		response to infection
		cterized by redness, pain, heat, swelling
•	Acute	inflammation –,
		Cause of inflammation
		Boils caused by S. aureus
•	Chron	ic inflammation – lasting, less intense
		Cause of inflammation
		Tuberculosis caused by M. tuberculosis
•	Functi	ons:
	0	To the injurious agent
	0	Limit the effects on body by injurious agent
		 Occurs if destruction, removal not possible
		or replace damaged tissue
Sta	ages of i	inflammation
•		ilation and increased permeability of blood vessels
		Tissue damage results in vasodilation, increase in permeability of blood vessels
	0	Vasodilation – (increase in diameter) of blood vessels
		Increases to area
		Increases to areaResponsible for "" (redness),
	0	Increased permeability allows WBC, chemicals to pass from to injured area
		Responsible for "" (swelling)
	0	release in response to injury
		 Released by cells,
	0	Blood clots around injury prevents
•	Phago	cyte migration and phagocytosis
	0	Blood flow eventually brings to site of infection
		invading microorganisms
	0	In response to bacteria, neutrophils first, followed by monocytes
	0	often die after killing many cells
		 Cell debris contribute to
•	Tissue	repair
	0	Replacement of or cells
	0	Speed of repair depends on tissue
		 Skin heals fast, cardiac (heart) muscle heals slow

	temperature	
response to in		
Most commonly caused by	, infection infection of body "thermostate"	ons
Certain chemicals trigger a "		ıt" to a
temperature		
0		
Chill – response to		
	ering, increased metabolism, constriction	of blood vessels
Crisis – response to	body temperature	
o Skin gets warm, perspir		
Fever is helpful up to certain de	-	
	production, metabolism,	repair, etc
Complications include:		
o – rapid	I heart rate, may compromise weak hearte	ed
o Increased	– dehydration, electrolyte imbala	ance, acidosis
o in you	ung children	
o Delirium		
o Coma	000	
o°C ($\underline{}$ = death	
Destroy microbes by:	ve system consisting of	in blood
Complement system – defensiv Destroy microbes by: O(_ O Triggering O Helping with) of bacteria	in blood
Complement system – defensiv Destroy microbes by: O(_ O Triggering O Helping with ading complement system) of bacteria	in blood
Complement system – defensiv Destroy microbes by: O(_ O Triggering O Helping with adding complement system Some capsules prevent complements) of bacteria	
Complement system – defensive Destroy microbes by: O	mentan lengthen surface glycolipids to preven	
Complement system – defensive Destroy microbes by: O	ment an lengthen surface glycolipids to preven	ıt
Complement system – defensive Destroy microbes by: O	mentan lengthen surface glycolipids to preven	ıt
Complement system – defensive Destroy microbes by: O	ment an lengthen surface glycolipids to preven	ıt
Complement system – defensiv Destroy microbes by:	ment not bacteria an lengthen surface glycolipids to prevent ant" ase that produced by	ıt complement protei
Complement system – defensiv Destroy microbes by:	ment not bacteria ment an lengthen surface glycolipids to prevent ant" ase that	ıt complement protei
Complement system – defensive Destroy microbes by: O	ment not bacteria an lengthen surface glycolipids to prevent ant" ase that produced by	ıt complement protei
Complement system – defensiv Destroy microbes by: O	ment	t complement protei cells
Complement system – defensiv Destroy microbes by: O	ment	t complement protei cells
Complement system – defensiv Destroy microbes by:	ment	t complement protei cells
Complement system – defensiv Destroy microbes by: O	ment	t complement protei cells

•	Some (Hepatitis B virus) do not induce	e a great interferon re	esponse	
•	Can serve as potential	_ drugs,	drugs	
Aı	ntimicrobial peptides			
•	Newly discovered, may be most impor	tant component of _		immunity
•	Small			
	\circ 10 – 20 amino acids			
•	Bind to	causing cell ly	vsis	
•	Produced by	and		