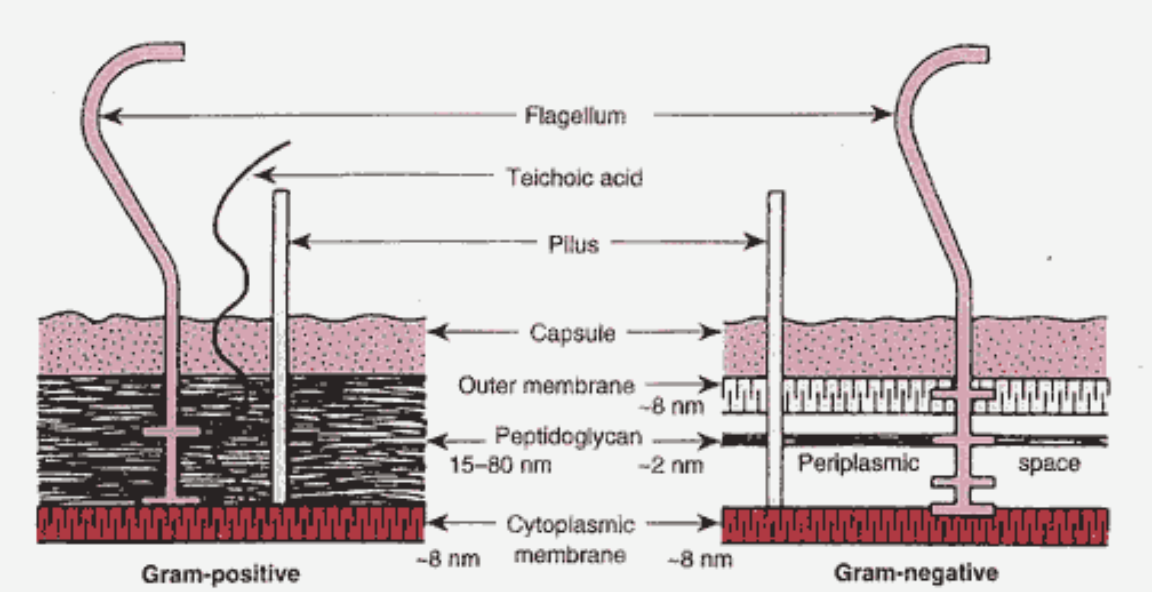


Figure 2–4. Cell walls of gram-positive and gram-negative bacteria. Note that the peptidoglycan in gram-positive bacteria is much thicker than in gram-negative bacteria. Note also that only gram-negative bacteria have an outer membrane containing endotoxin (lipopolysaccharide [LPS]) and have a periplasmic space where β-lactamases are found. Several important gram-positive bacteria, such as staphylococci and streptococci, have teichoic acids. (Reproduced, with permission, from Ingraham JL, Maaløe O, Neidhardt FC: *Growth of the Bacterial Cell*, Sinauer Associates, 1983.)

12a

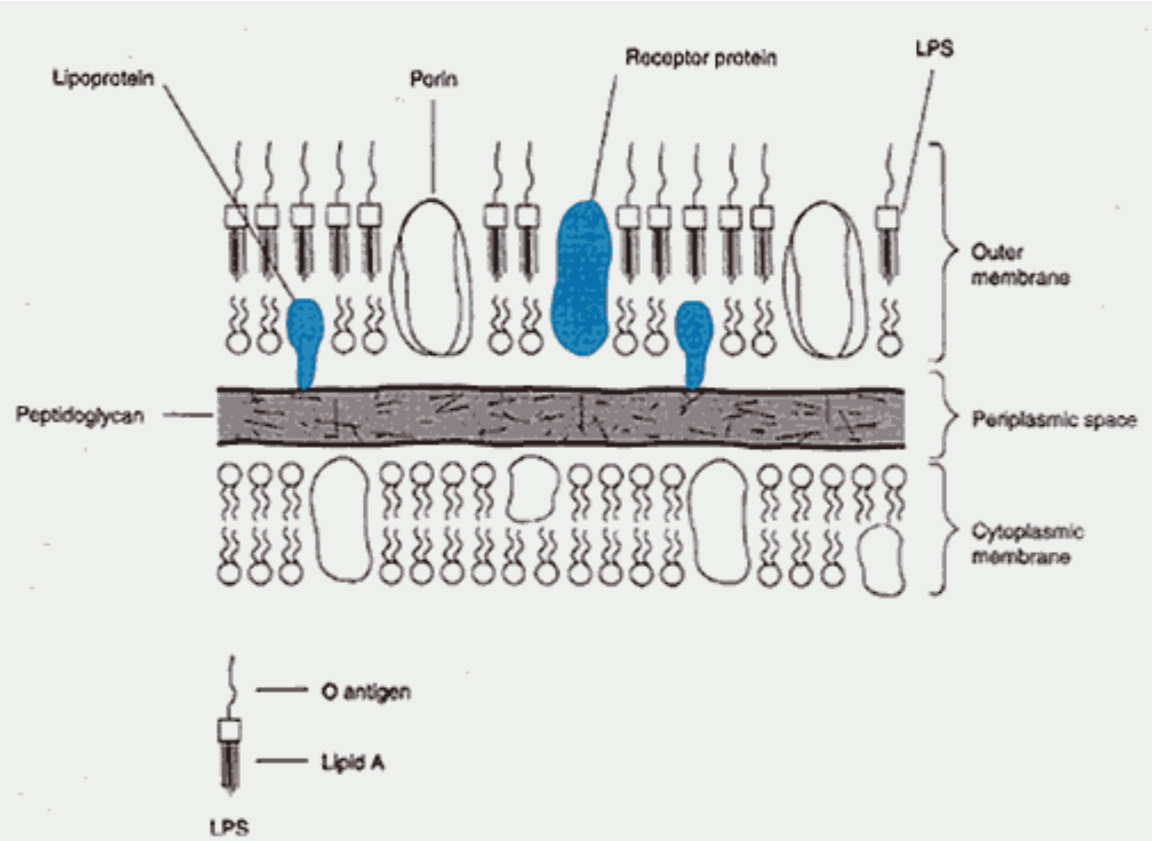
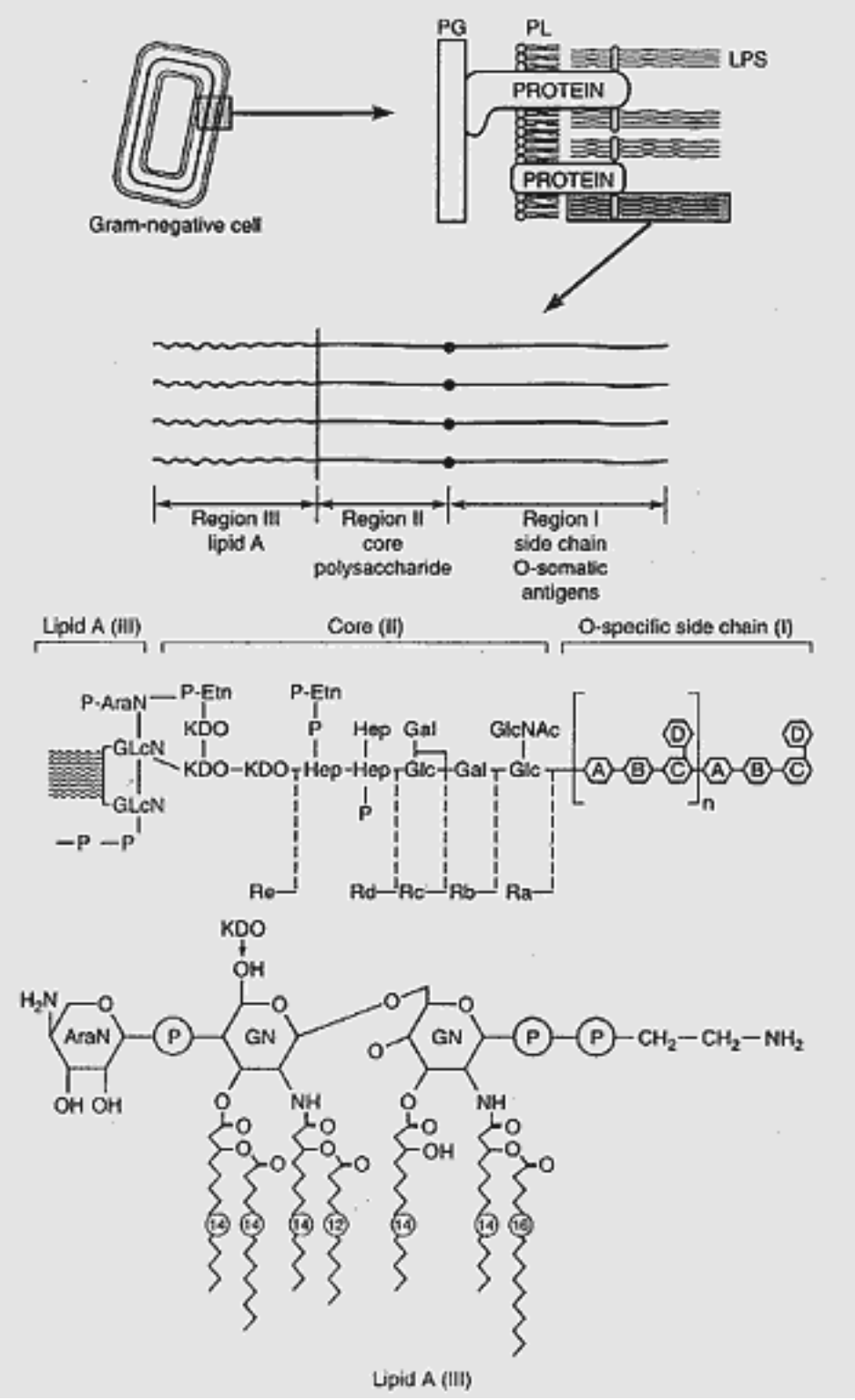


Figure A1-3 Structure of the cell surface of a gram-negative bacterium.

12b


PG
PL
LPS
PROTEIN
PROTEIN
Gram-negative cell
Region III
lipid A
Region II
core
polysaccharide
Region I
side chain
O-somatic
antigens
Lipid A (III)
Core (II)
O-specific side chain (I)
P-AraN
P-Etn
KDO
GLcN
GLcN
—P —P
KDO–KDO
P-Etn
P
Hep–Hep
P
Hep
Gal
Glc–Gal–Glc
GlcNAc
n
Re
Rd
Rc
Rb
Ra
KDO
OH
AraN
GN
GN
P
P
P
$CH_2—CH_2—NH_2$
H_2N
OH OH
NH
NH
OH
14
14
14
12
14
14
16
Lipid A (III)
12c

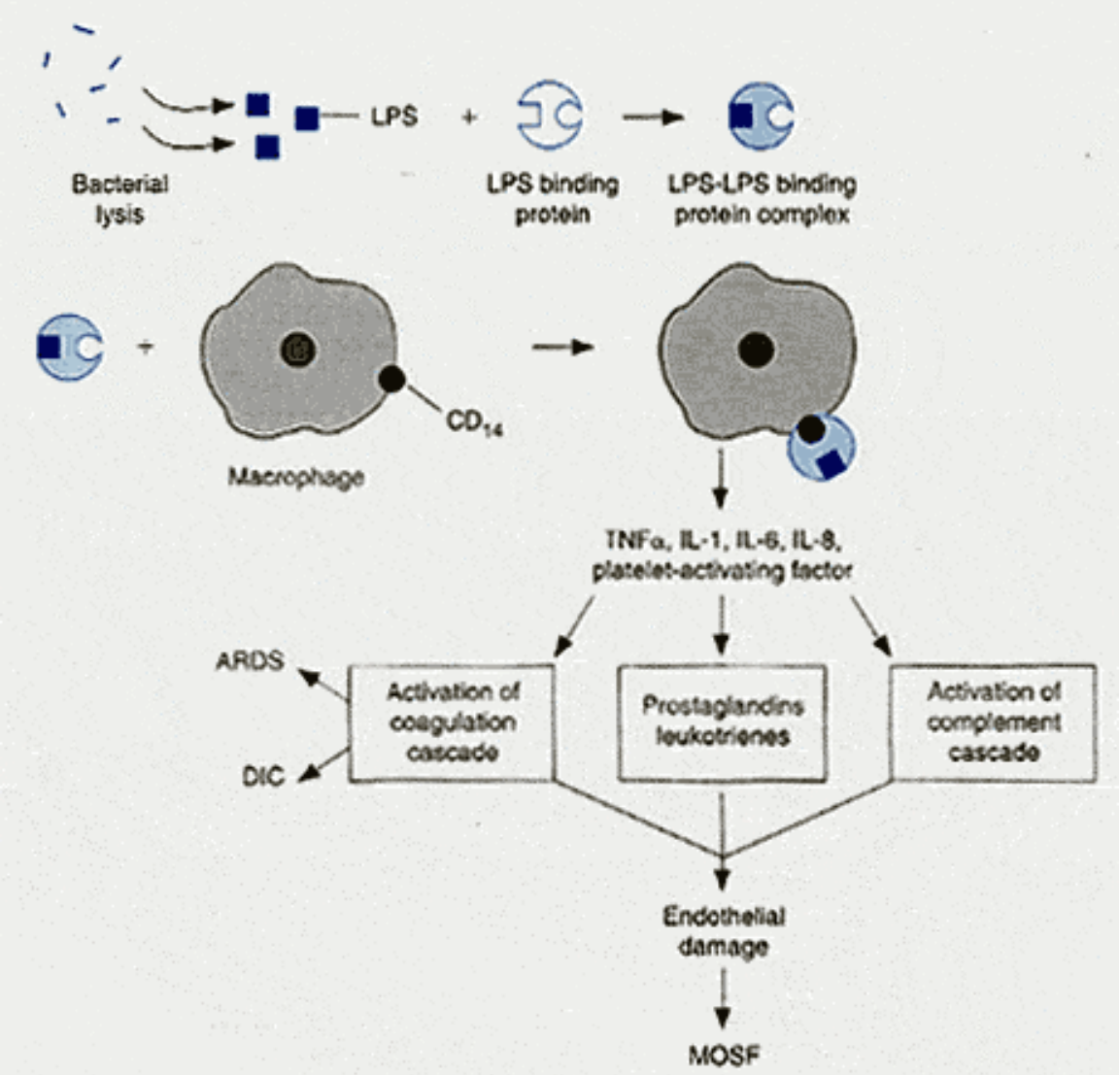
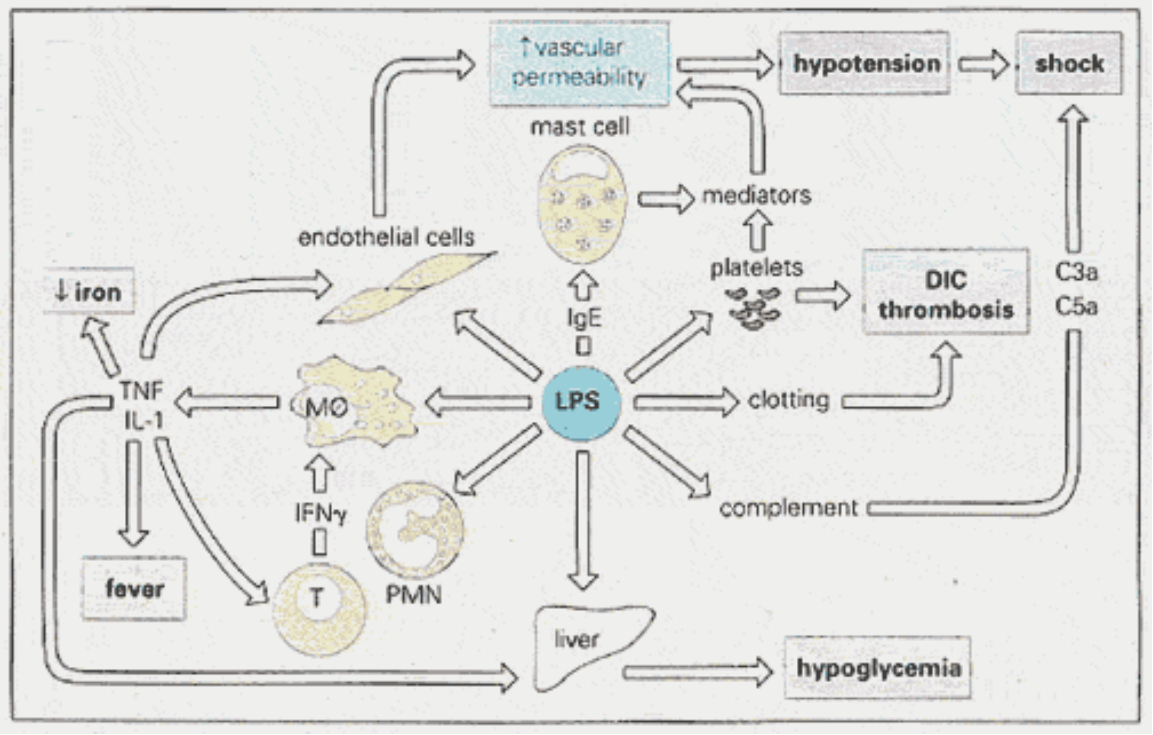


Figure 4-7 Triggering of cytokine release by gram-negative LPS and the role in septic shock. (TNFα, tumor necrosis factor alpha; IL-1, interleukin-1; IL-6, interleukin-6; IL-8, interleukin-8; DIC, disseminated intravascular coagulation; ARDS, acute respiratory distress syndrome; MOSF, multiple organ system failure.)

12d



12e

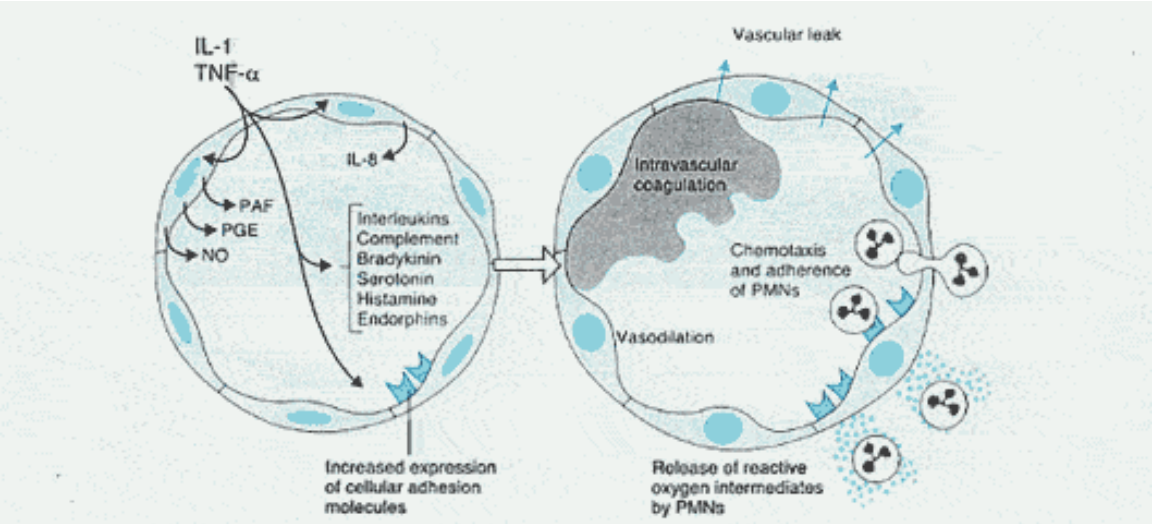
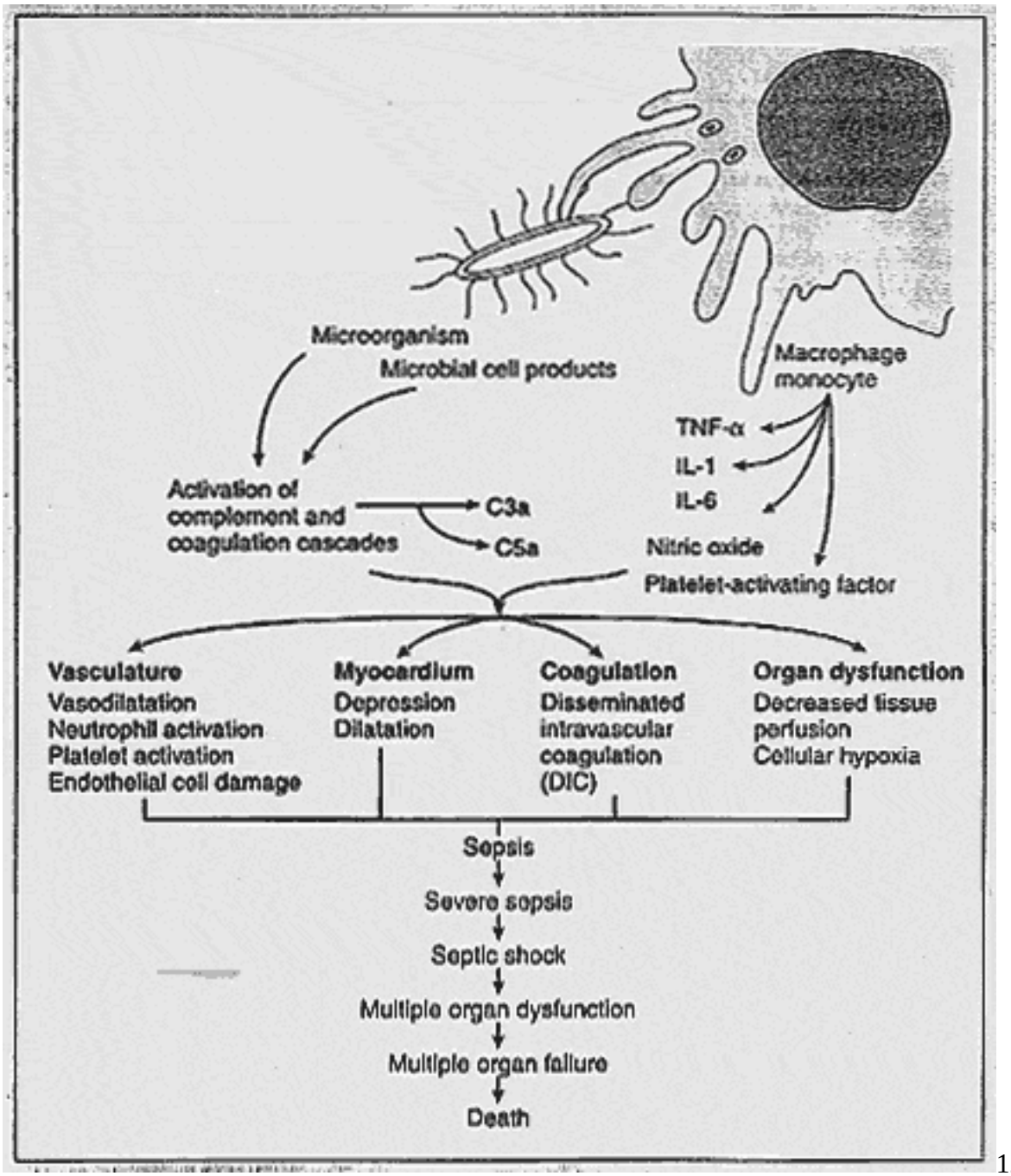


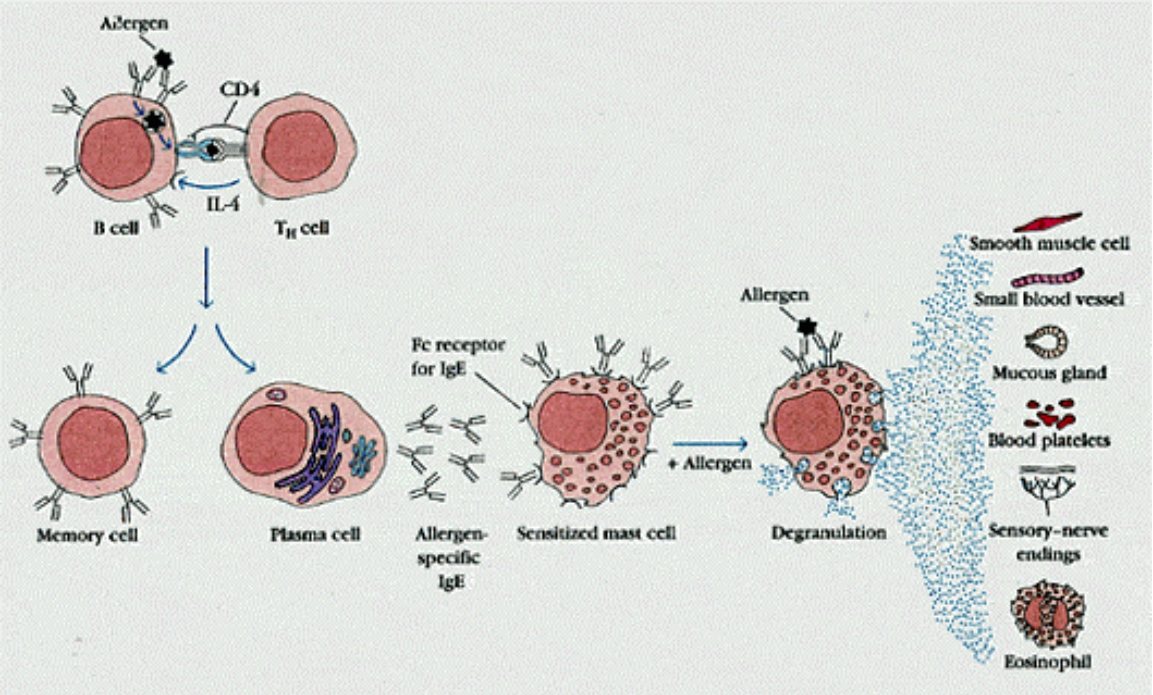
Figure 63.1. Vasculature effects in sepsis. The release of IL-1 and TNF-α causes the outpouring of a number of cytokines and other effectors. In turn, this leads to intravascular coagulation, vasodilation, increased neutrophil adherence to vessel walls, and vascular leakage.

12f


Microorganism
Microbial cell products
Macrophage monocyte
TNF-α
IL-1
IL-6
Activation of complement and coagulation cascades
C3a
C5a
Nitric oxide
Platelet-activating factor
Vasculature
Vasodilatation
Neutrophil activation
Platelet activation
Endothelial cell damage
Myocardium
Depression
Dilatation
Coagulation
Disseminated intravascular coagulation (DIC)
Organ dysfunction
Decreased tissue perfusion
Cellular hypoxia
Sepsis
Severe sepsis
Septic shock
Multiple organ dysfunction
Multiple organ failure
Death
12g

Mediator	Type	Initiation Time	Reaction	Examples
Antibody	I (immediate, anaphylactic)	2-30 min	IgE antibody is induced by allergen and binds via its Fc receptor to mast cells and basophils. After encountering the antigen again, the fixed IgE becomes cross-linked, inducing degranulation and release of mediators (eg, histamine, serotonin, etc.)	Systemic anaphylaxis: Common allergens include: drugs (penicillin) and insect venom from bees and wasps) Localized anaphylaxis: Hayfever (upper respiratory) Asthma (lower respiratory) Food allergies (hives-skin eruptions)
Antibody	II (cytotoxic)	5-8 hours	Antigens on cell surface combine with antibody (IgM or IgG); this leads to complement-mediated lysis	Blood-transfusion reactions (ABO or Rh) Autoimmune hemolytic anemia
Antibody	III (immune complex)	2-8 hours	Large amounts of antigen-antibody immune complexes are deposited in tissues, complement is activated, and polymorpho-nuclear cells are attracted to the site, causing inflammation and tissue damage	Acute poststreptococcal glomerulonephritis Various autoimmune diseases (including rheumatoid arthritis, and systemic lupus erythematosus)
Cell	IV (delayed)	24 to 72 hours	T_H1 lymphocytes sensitized by antigen release cytokines that activate macrophages which, in turn, release various mediators which cause direct cellular damage.	Contact dermatitis to poison ivy and poison oak Tuberculin type hypersensitivity

12h



12i

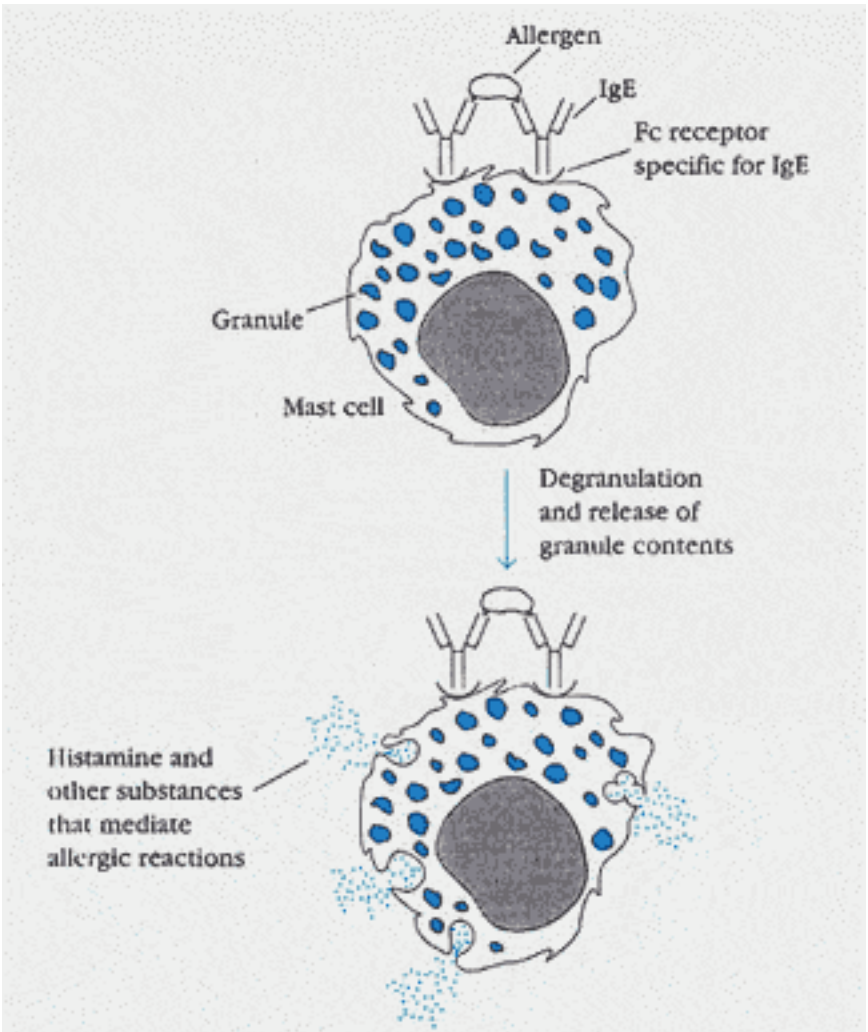
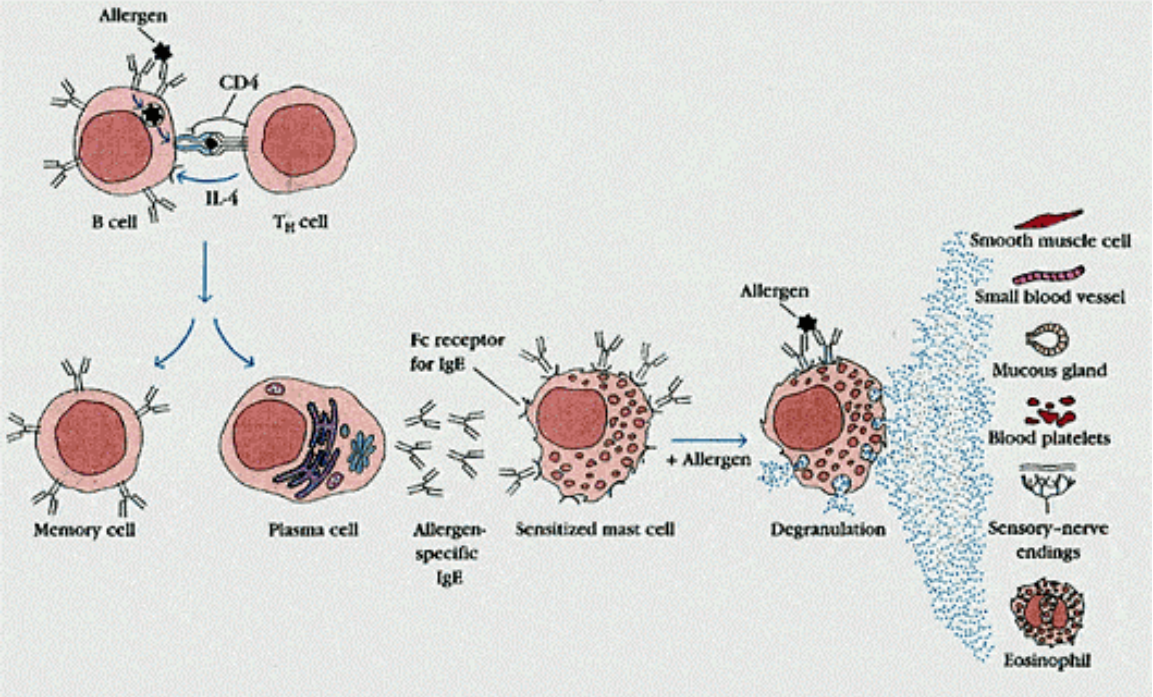


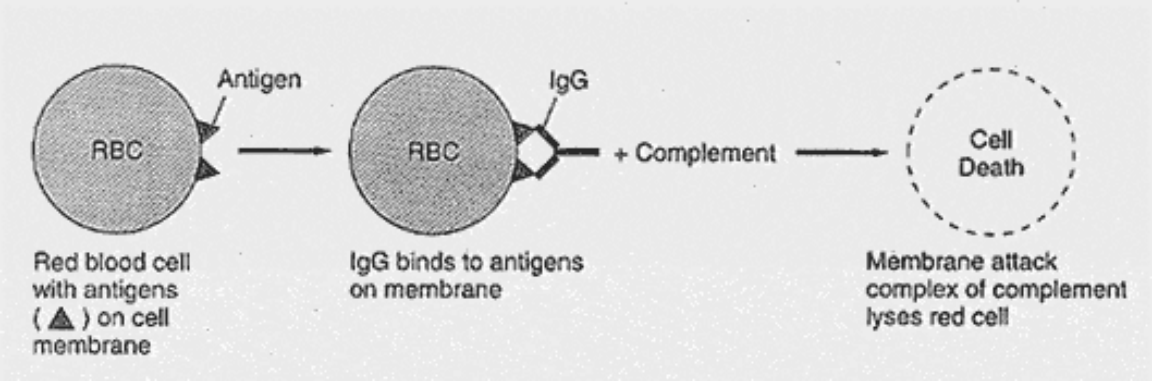
FIGURE 5-18

Allergen cross-linkage of receptor-bound IgE on mast cells induces degranulation, causing release of substances (blue dots) that mediate allergic manifestations.

12j


Allergen
CD4
IL-4
B cell
T_H cell
Memory cell
Plasma cell
Fc receptor for IgE
Allergen-specific IgE
Sensitized mast cell
+ Allergen
Allergen
Degranulation
Smooth muscle cell
Small blood vessel
Mucous gland
Blood platelets
Sensory-nerve endings
Eosinophil

12k


Antigen
RBC
IgG
RBC
+ Complement
Cell Death
Red blood cell with antigens (▲) on cell membrane
IgG binds to antigens on membrane
Membrane attack complex of complement lyses red cell

12l

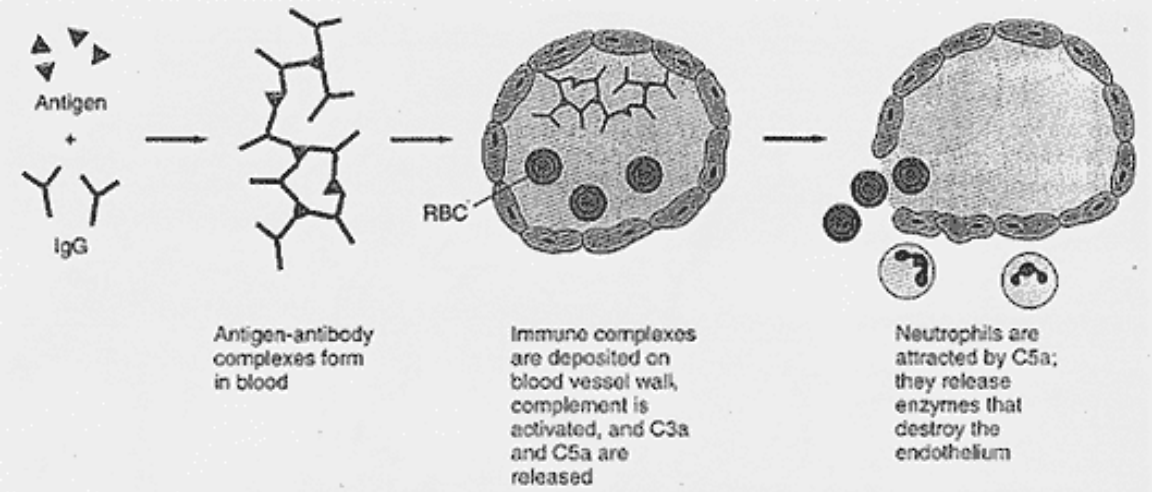
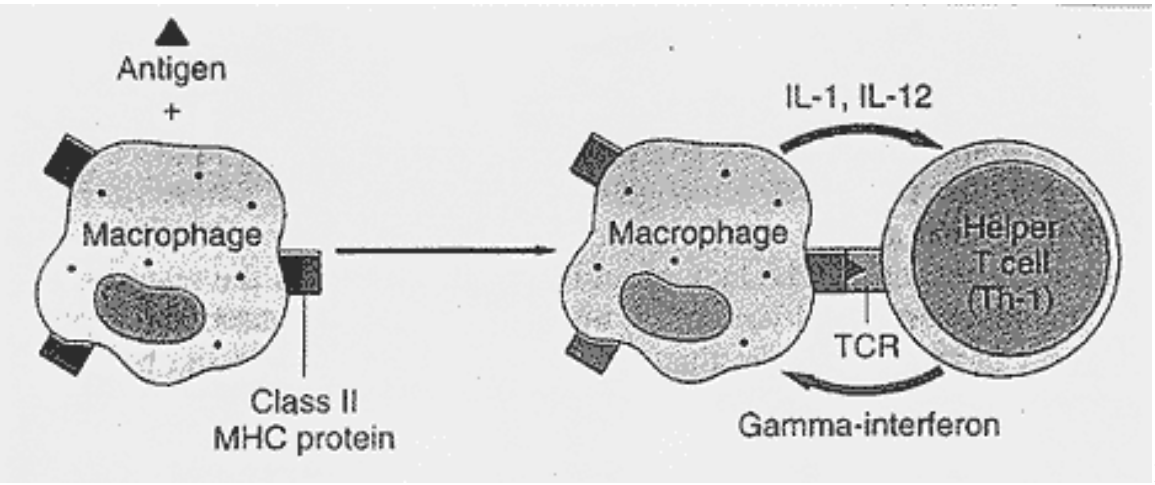


Figure 65–3. Immune complex hypersensitivity.

12m



12n

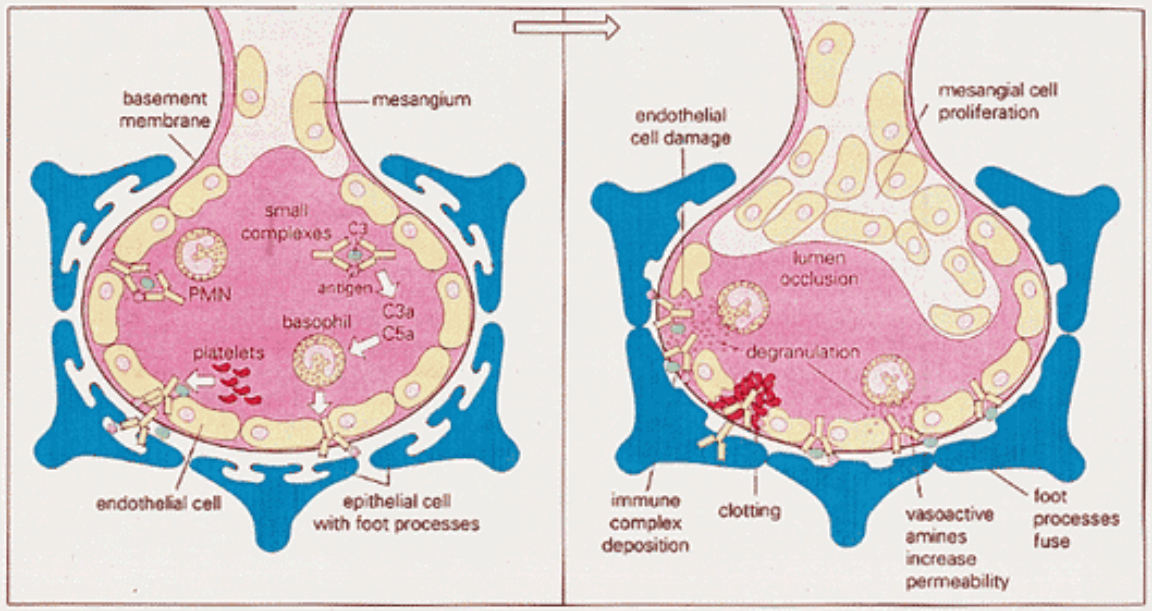
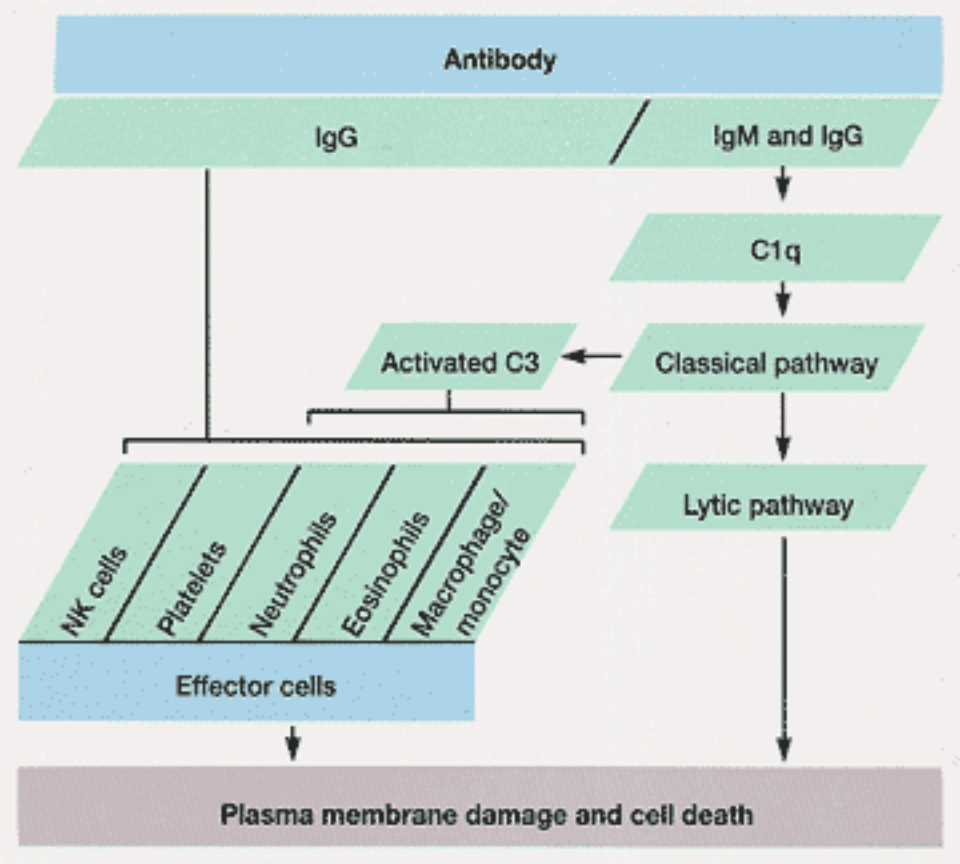
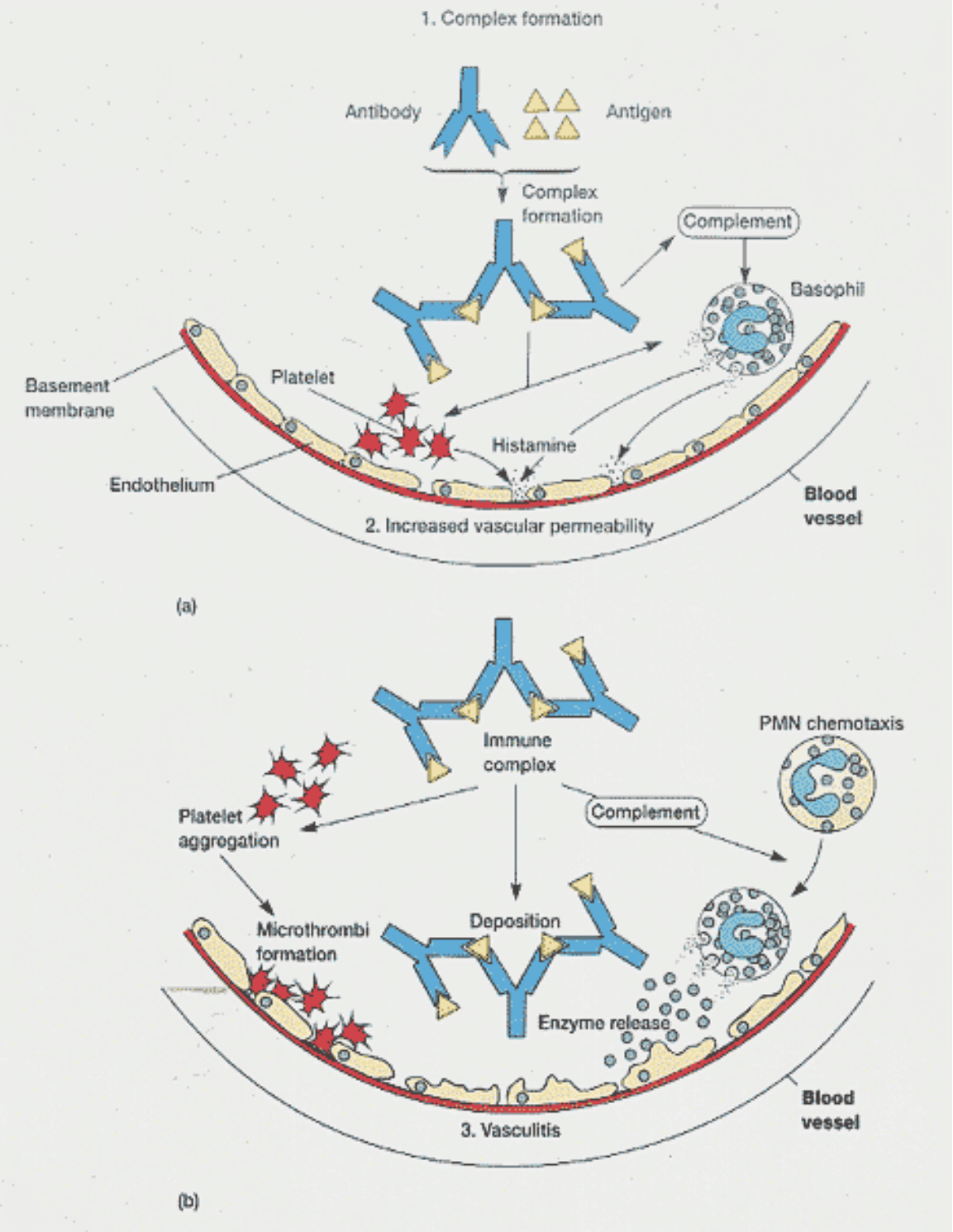


Fig. 12.10 Immune complex-mediated tissue damage. Type III hypersensitivity results in the deposition of immune complexes in the blood vessel walls, particularly at sites of high pressure, filtration or turbulence such as the kidney. (PMN, polymorphonuclear leukocyte.)

12o



12p


1. Complex formation
Antibody
Antigen
Complex formation
Complement
Basophil
Basement membrane
Platelet
Histamine
Endothelium
Blood vessel
2. Increased vascular permeability
(a)
Immune complex
PMN chemotaxis
Platelet aggregation
Complement
Microthrombi formation
Deposition
Enzyme release
Blood vessel
3. Vasculitis
(b)

12q