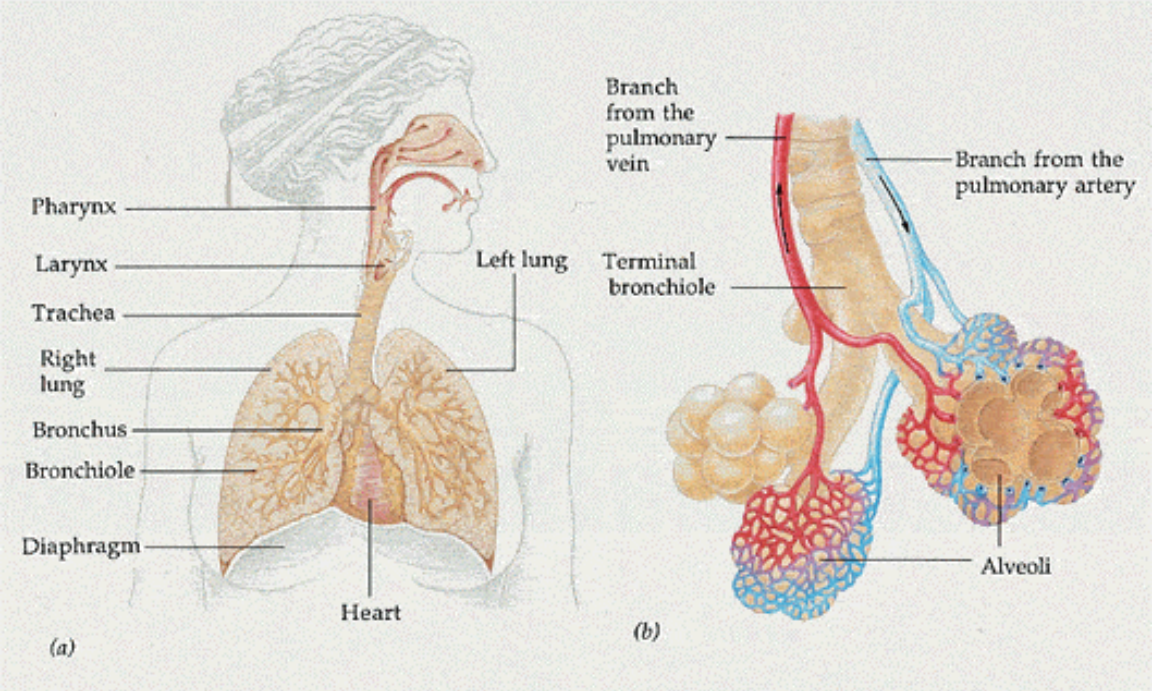
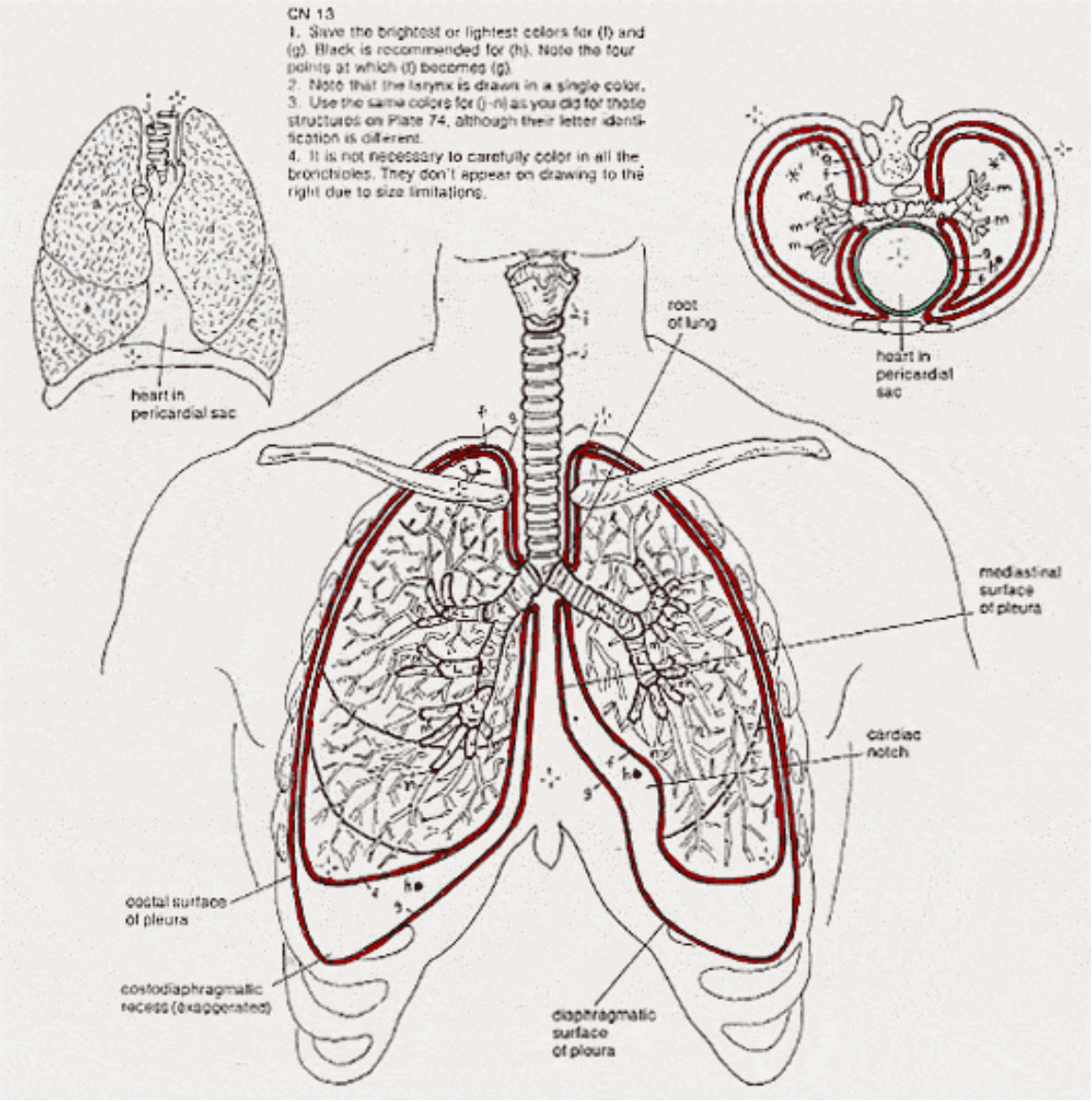

Pharynx
Larynx
Trachea
Right lung
Bronchus
Bronchiole
Diaphragm
Left lung
Heart
(a)
Branch from the pulmonary vein
Terminal bronchiole
Branch from the pulmonary artery
Alveoli
(b)

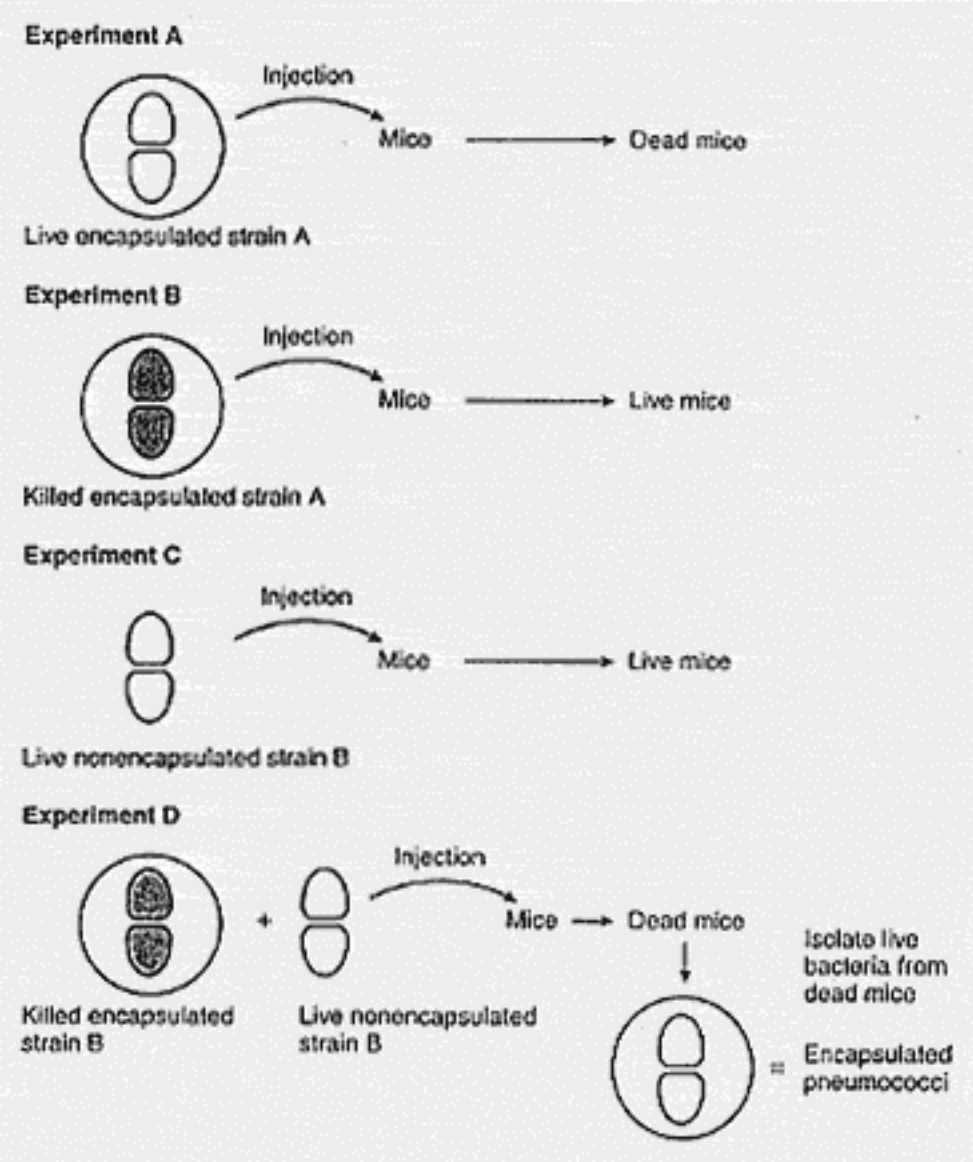
16a


CN 13
1. Save the brightest or lightest colors for (f) and (g). Black is recommended for (h). Note the four points at which (f) becomes (g).
2. Note that the larynx is drawn in a single color.
3. Use the same colors for (j–n) as you did for those structures on Plate 74, although their letter identification is different.
4. It is not necessary to carefully color in all the bronchioles. They don't appear on drawing to the right due to size limitations.
heart in pericardial sac
root of lung
heart in pericardial sac
mediastinal surface of pleura
cardiac notch
costal surface of pleura
costodiaphragmatic recess (exaggerated)
diaphragmatic surface of pleura

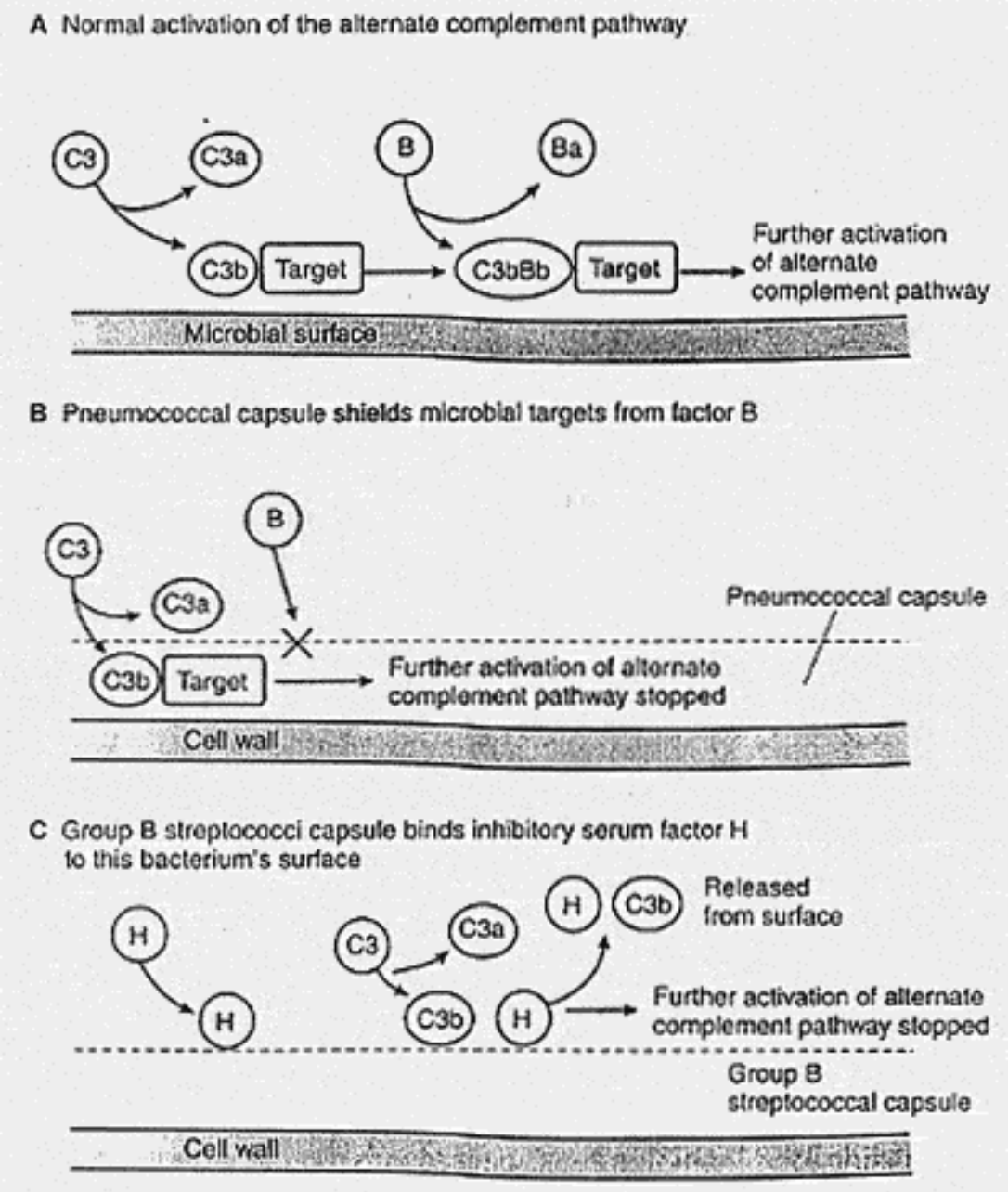
16b

Microorganism	*Capsule*
Streptococcus pneumoniae	Polysaccharide (approximately 90 types known)
Streptococcus pyogenes	Hylauronic acid
Bacillus anthracis	Polypeptide (polyglutamic acid)
Haemophilus influenzae type b	Polyribosylribitol phosphate (PRP or PRRP)
Neisseria meningitidis	Polysaccharides (capsule of each group has a different polysaccharide composition—note that the group B capsule contains sialic acid)
Salmonella typhi	Polysaccharide (called Vi antigen)
Klebsiella pneumoniae	Polysaccharide (referred to as K antigen)
Pseudomonas aeruginosa	Polysaccharide (called alginate)
Cryptococcus neoformans	Polysaccharide
Escherichia coli	K polysaccherides (over 80 different *E. coli* capsular polysaccharides have been identified; these polysaccharides are referred to as K antigens, e.g., the K1 antigen is a sialic acid polymer)

16c


Experiment A
Injection
Mice
Dead mice
Live encapsulated strain A
Experiment B
Injection
Mice
Live mice
Killed encapsulated strain A
Experiment C
Injection
Mice
Live mice
Live nonencapsulated strain B
Experiment D
Injection
Mice
Dead mice
Isolate live bacteria from dead mice
Killed encapsulated strain B
Live nonencapsulated strain B
Encapsulated pneumococci

16d


A Normal activation of the alternate complement pathway
C3
C3a
B
Ba
C3b
Target
C3bBb
Target
Further activation of alternate complement pathway
Microbial surface
B Pneumococcal capsule shields microbial targets from factor B
C3
B
C3a
Pneumococcal capsule
C3b
Target
Further activation of alternate complement pathway stopped
Cell wall
C Group B streptococci capsule binds inhibitory serum factor H to this bacterium's surface
H
H
C3
C3a
H
C3b
Released from surface
C3b
H
Further activation of alternate complement pathway stopped
Group B streptococcal capsule
Cell wall

16e

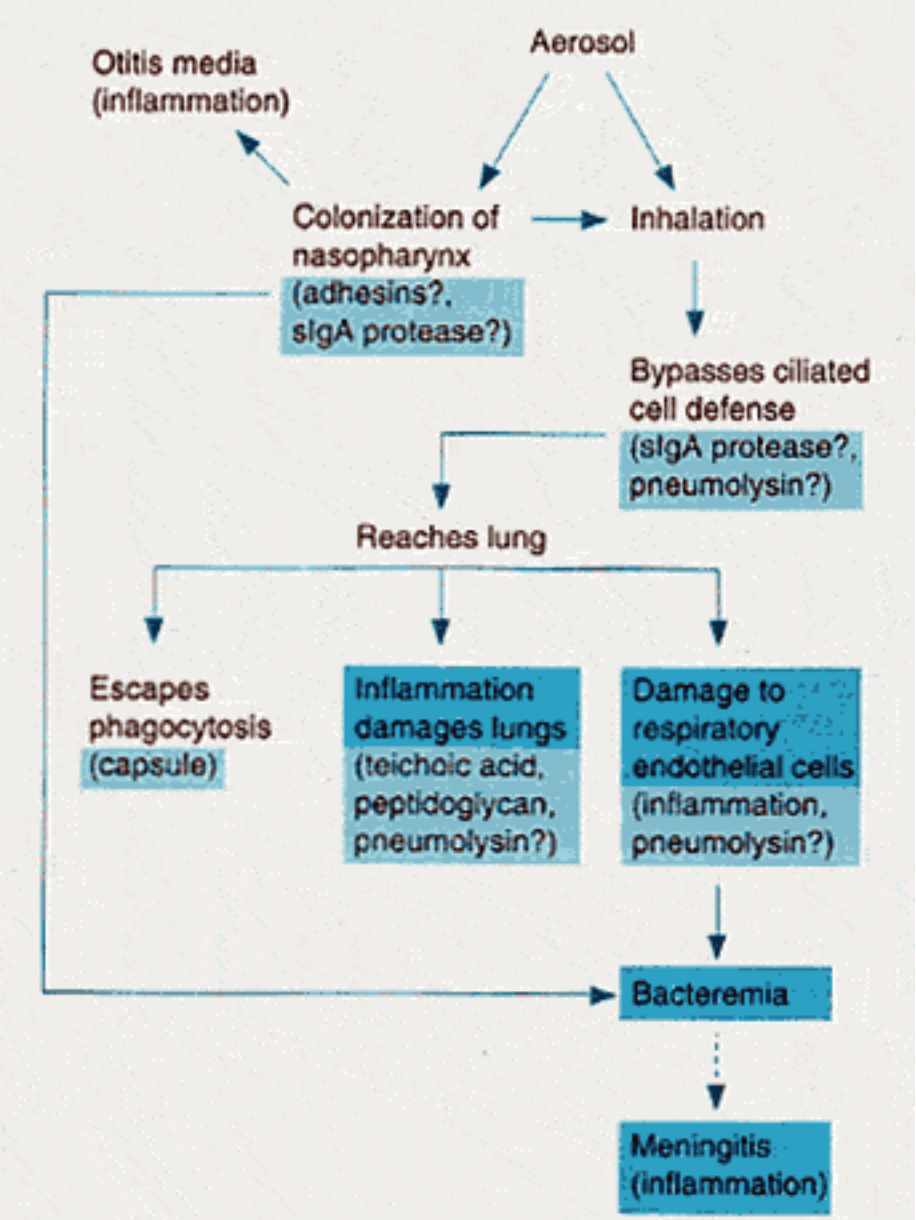


Figure 27-1 Steps in the pathogenesis of pneumococcal disease and associated virulence factors. (Virulence factors for which there is still not good evidence of in vivo importance but that might be significant in the disease process are indicated by "?.")

16f

TABLE 23-5 ***Streptococcus pneumoniae* Virulence Factors**

VIRULENCE FACTOR	BIOLOGICAL EFFECT
COLONIZATION AND MIGRATION	
Protein adhesin	Binds to epithelial cells
Secretory IgA protease	Disrupts secretory IgA-mediated clearance
Pneumolysin	Possibly destroys ciliated epithelial cells
TISSUE DESTRUCTION	
Teichoic acid	Activates alternative C pathway
Peptidoglycan fragments	Activates alternative C pathway
Pneumolysin	Activates classic complement pathway
Hydrogen peroxide	Allows reactive oxygen intermediates to cause damage
Phosphorylchorine	Binds phosphodiesterase-activating factor, allowing bacteria to enter host cells
PHAGOCYTIC SURVIVAL	
Capsule	Antiphagocytic
Pneumolysin	Suppresses phagocytic oxidative burst

16g