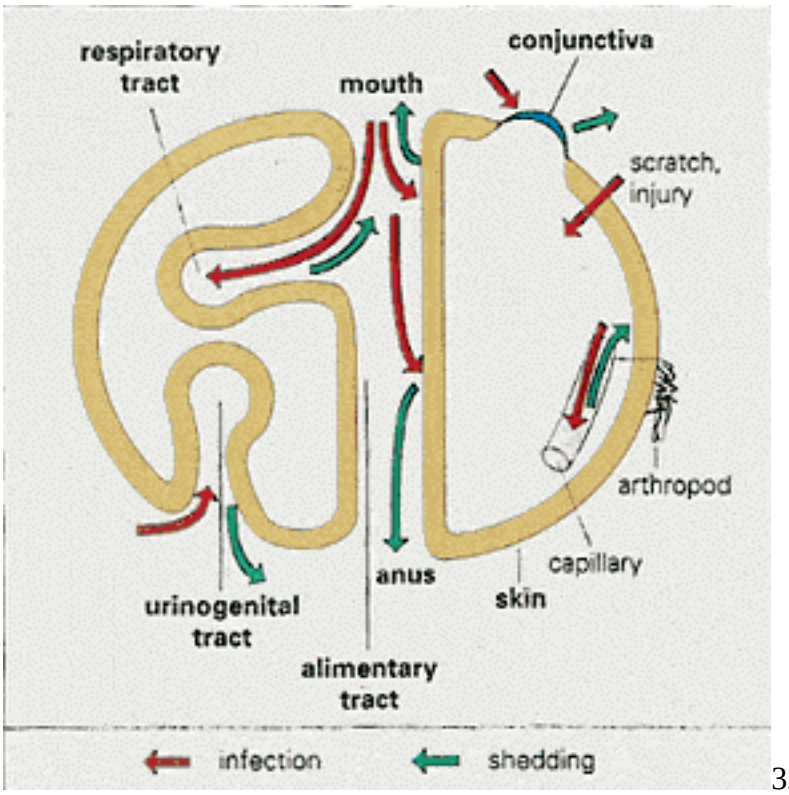
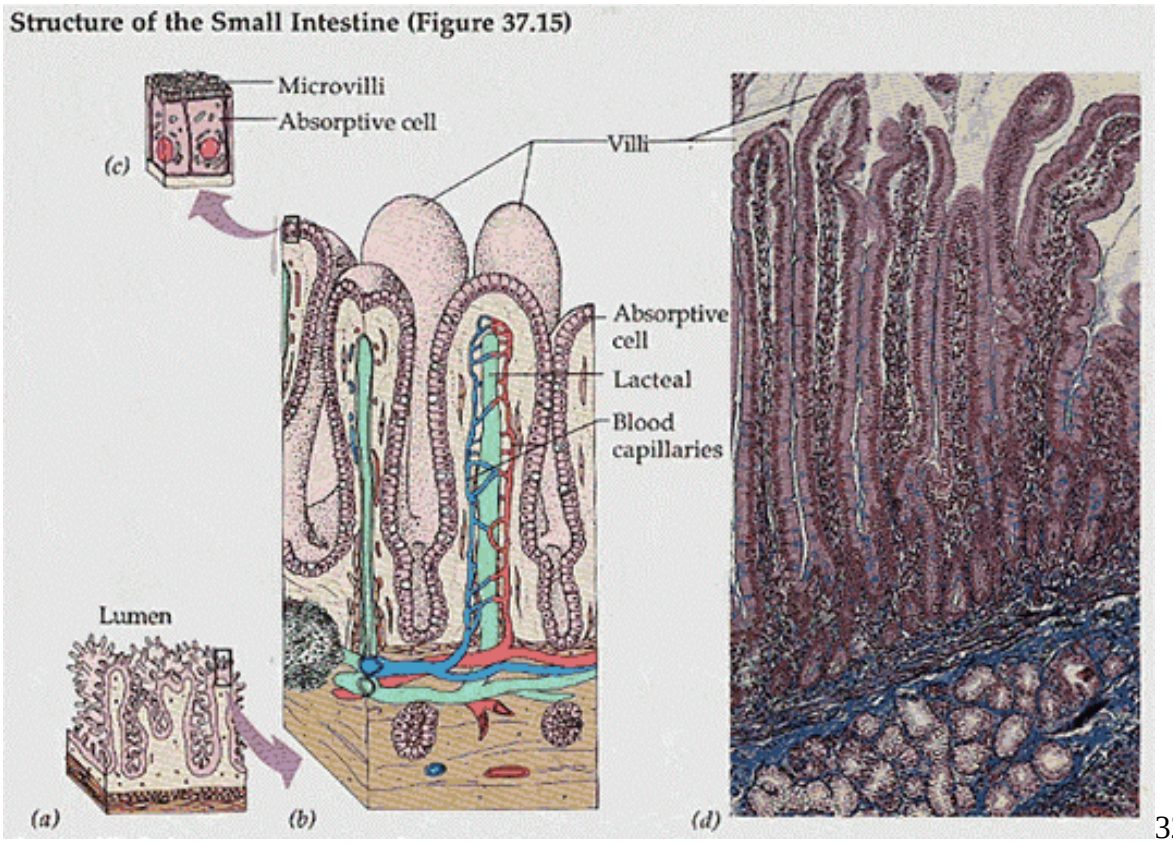

respiratory tract
mouth
conjunctiva
scratch, injury
arthropod
capillary
skin
anus
urinogenital tract
alimentary tract
infection
shedding
32a


Structure of the Small Intestine (Figure 37.15)
Microvilli
Absorptive cell
(c)
Villi
Absorptive cell
Lacteal
Blood capillaries
Lumen
(a)
(b)
(d)
32b

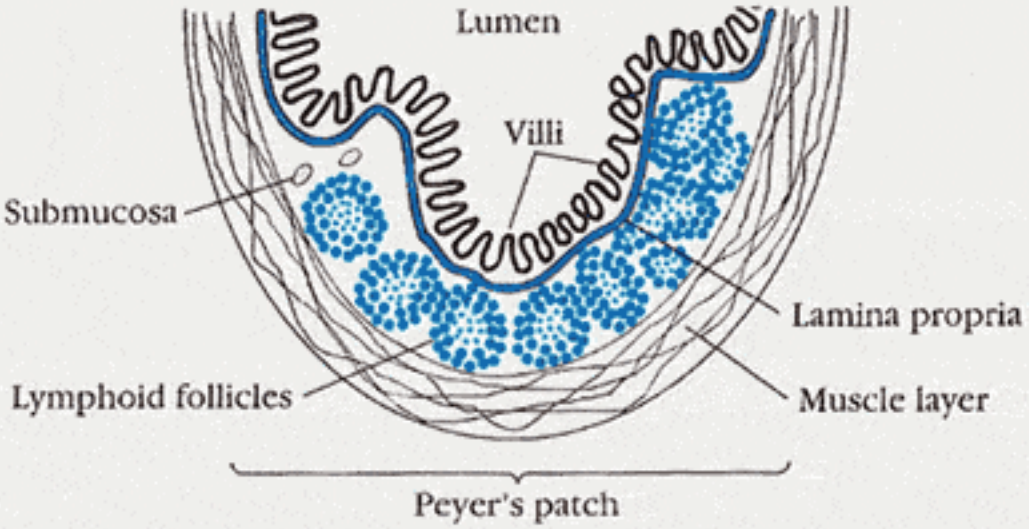


FIGURE 3-23

Cross-sectional diagram of the mucous membrane lining the intestine showing nodule of lymphoid follicles constituting a Peyer's patch in the submucosa. The intestinal lamina propria contains loose clusters of lymphoid cells and diffuse follicles.

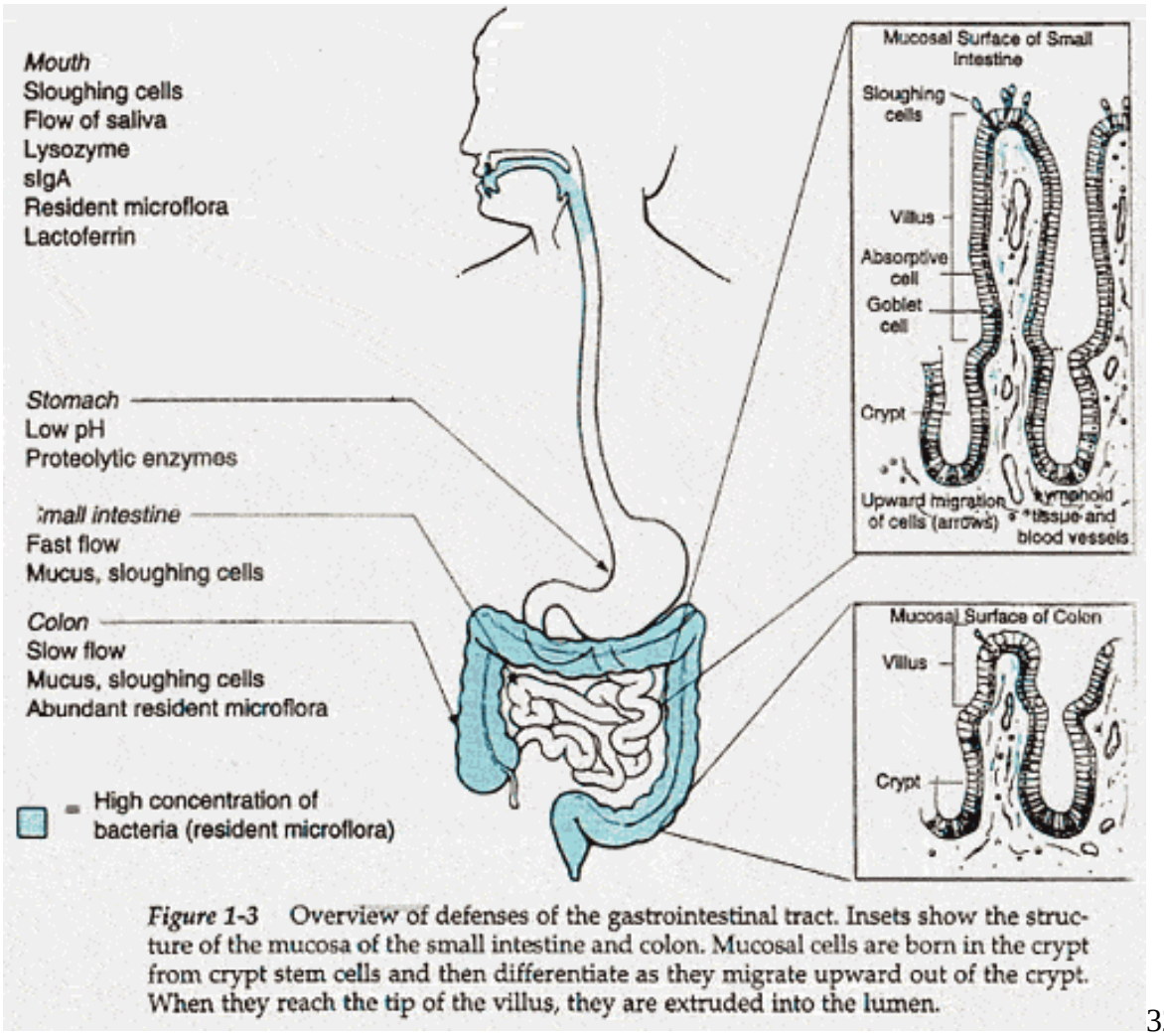
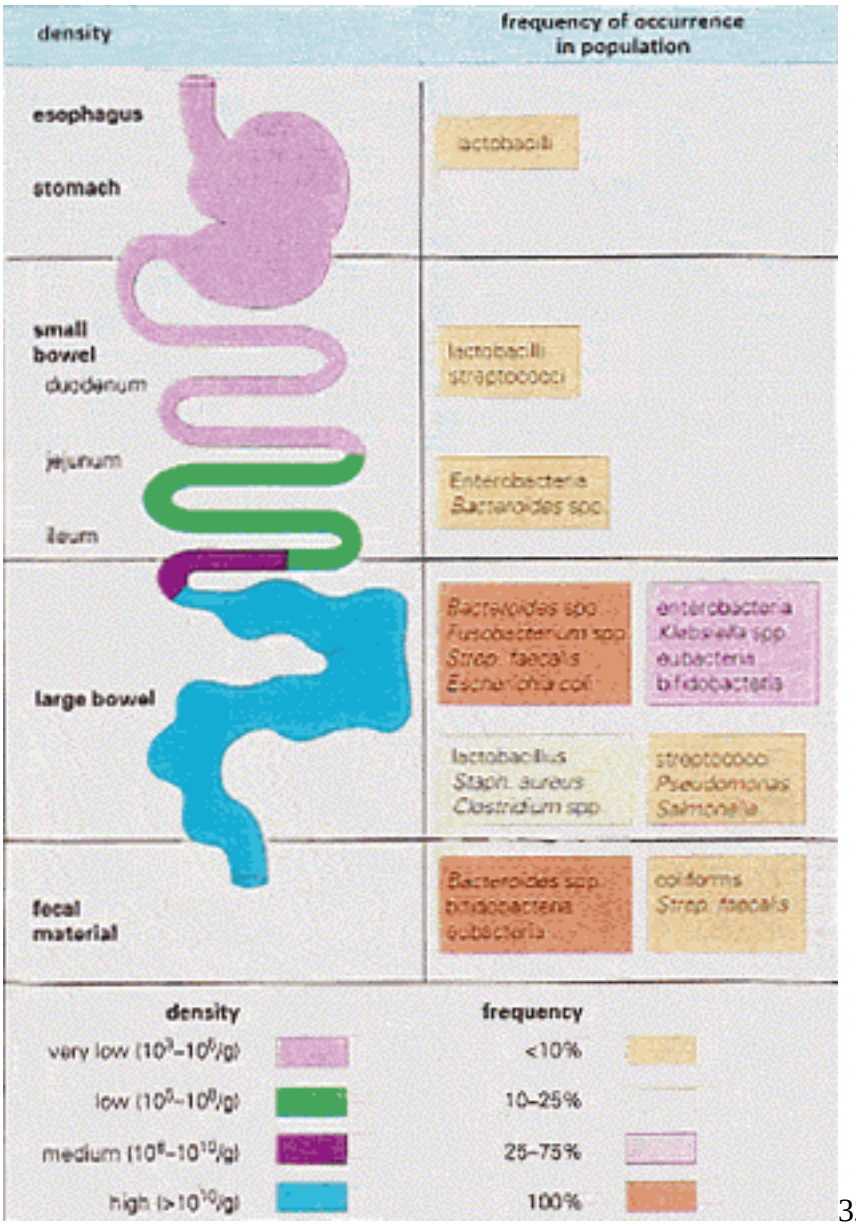


Figure 1-3 Overview of defenses of the gastrointestinal tract. Insets show the structure of the mucosa of the small intestine and colon. Mucosal cells are born in the crypt from crypt stem cells and then differentiate as they migrate upward out of the crypt. When they reach the tip of the villus, they are extruded into the lumen.


density
frequency of occurrence in population
esophagus
stomach
lactobacilli
small bowel
duodenum
jejunum
ileum
lactobacilli
streptococci
Enterobacteria
Bacteroides spp.
large bowel
Bacteroides spp.
Fusobacterium spp.
Strep. faecalis
Escherichia coli
enterobacteria
Klebsiella spp.
eubacteria
bifidobacteria
lactobacillus
Staph. aureus
Clostridium spp.
streptococci
Pseudomonas
Salmonella
fecal material
Bacteroides spp.
bifidobacteria
eubacteria
coliforms
Strep. faecalis
density
very low (10^3–10^5/g)
low (10^5–10^8/g)
medium (10^8–10^{10}/g)
high (>10^{10}/g)
frequency
<10%
10–25%
25–75%
100%
32e

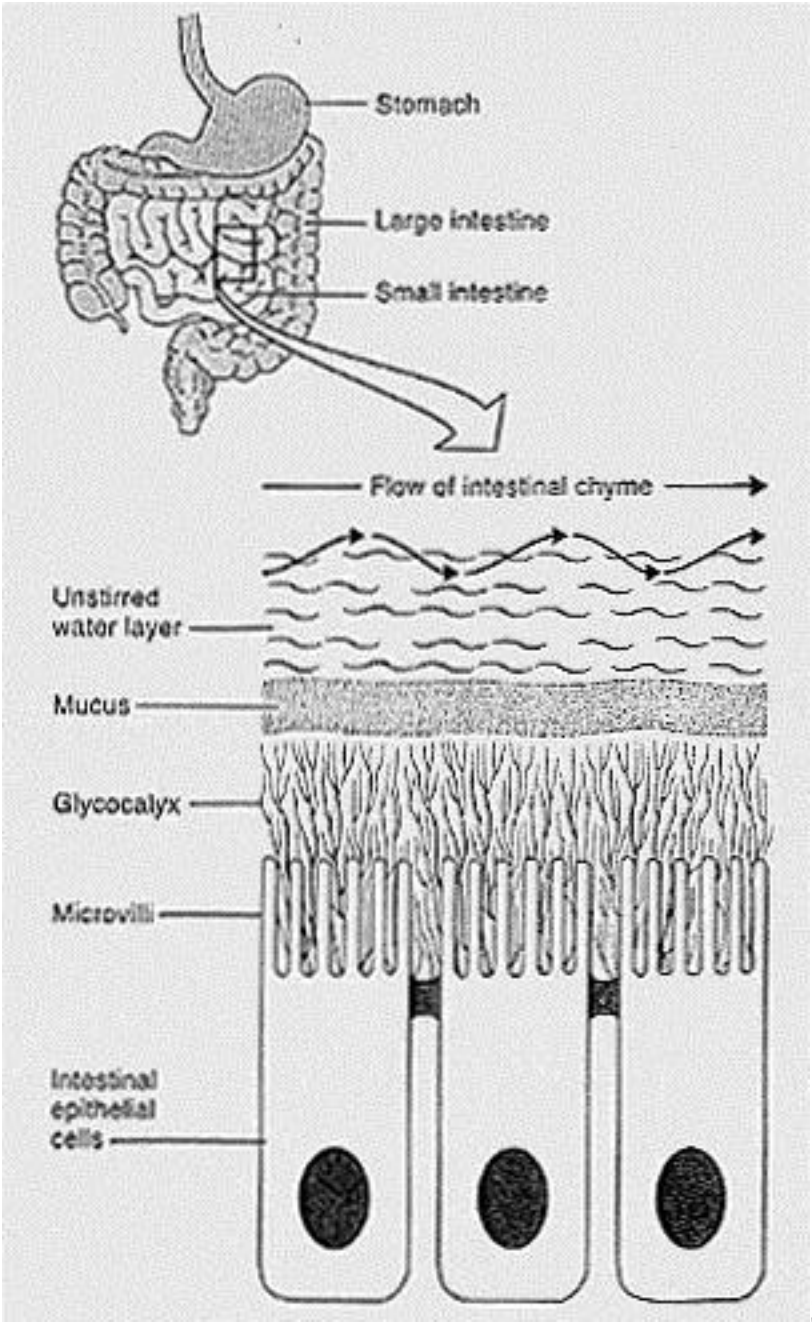

Stomach
Large intestine
Small intestine
Flow of intestinal chyme
Unstirred water layer
Mucus
Glycocalyx
Microvilli
Intestinal epithelial cells
32f

Table 30–1. Important features of acute viral diseases.

	Local Infections	Systemic Infections
Specific disease example	Respiratory (rhinovirus)	Measles
Site of pathology	Portal of entry	Distant site
Incubation period	Relatively short	Relatively long
Viremia	Absent	Present
Duration of immunity	Variable—may be short	Usually lifelong
Role of secretory antibody (IgA) in resistance	Usually important	Usually not important

32g

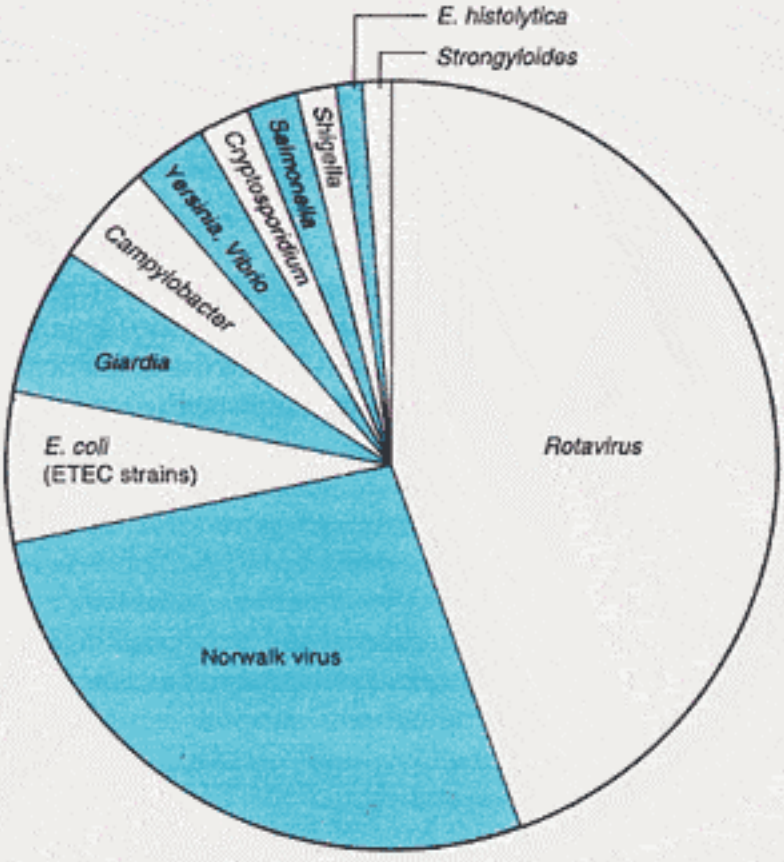
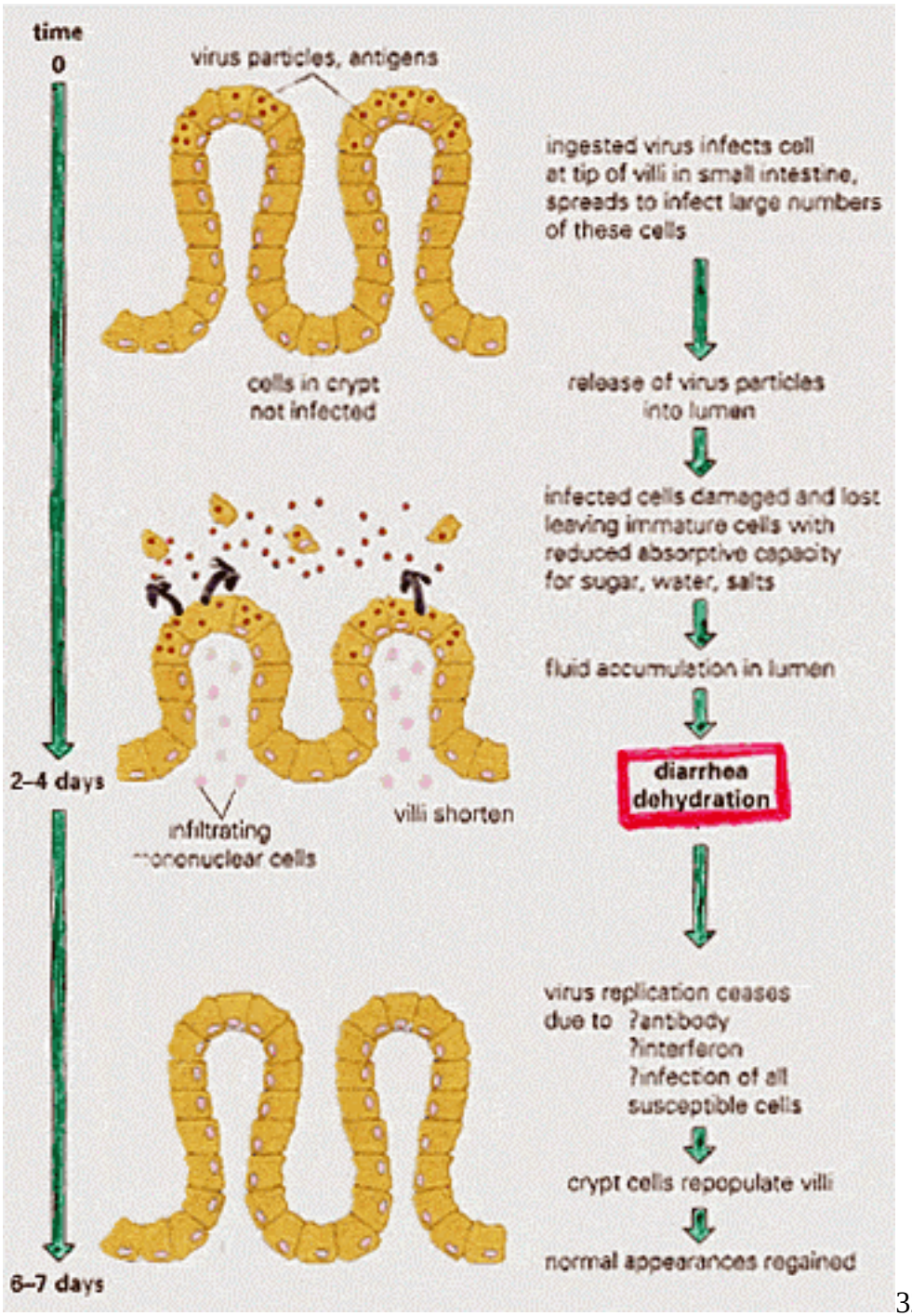
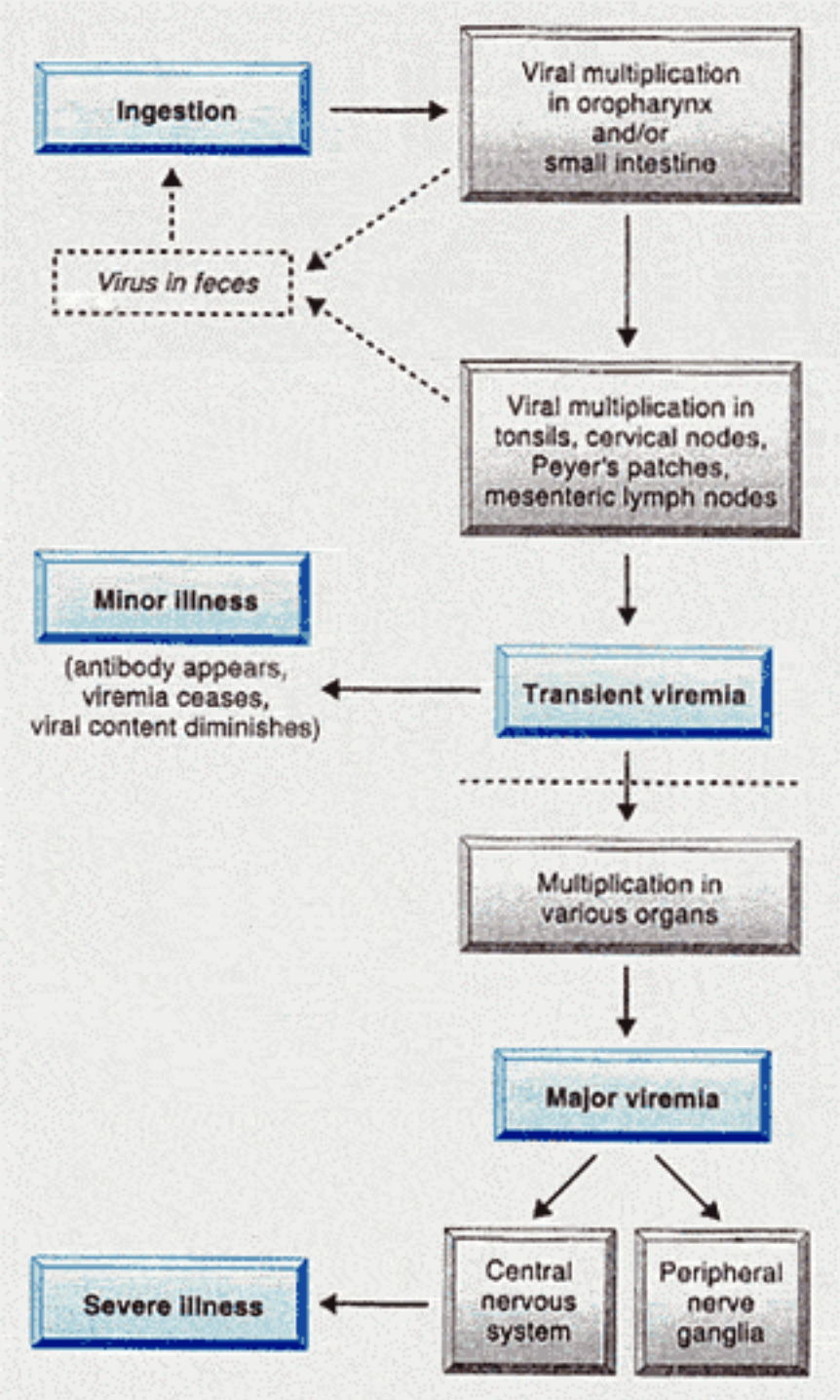


Figure 57.5. Distribution of agents of diarrhea in residents of the U.S.

32h


time
0
virus particles, antigens
ingested virus infects cell at tip of villi in small intestine, spreads to infect large numbers of these cells
cells in crypt not infected
release of virus particles into lumen
infected cells damaged and lost leaving immature cells with reduced absorptive capacity for sugar, water, salts
fluid accumulation in lumen
diarrhea dehydration
2–4 days
infiltrating mononuclear cells
villi shorten
virus replication ceases due to ?antibody ?interferon ?infection of all susceptible cells
crypt cells repopulate villi
normal appearances regained
6–7 days

32i


Ingestion
Viral multiplication in oropharynx and/or small intestine
Virus in feces
Viral multiplication in tonsils, cervical nodes, Peyer's patches, mesenteric lymph nodes
Minor illness
(antibody appears, viremia ceases, viral content diminishes)
Transient viremia
Multiplication in various organs
Major viremia
Central nervous system
Peripheral nerve ganglia
Severe illness
32j

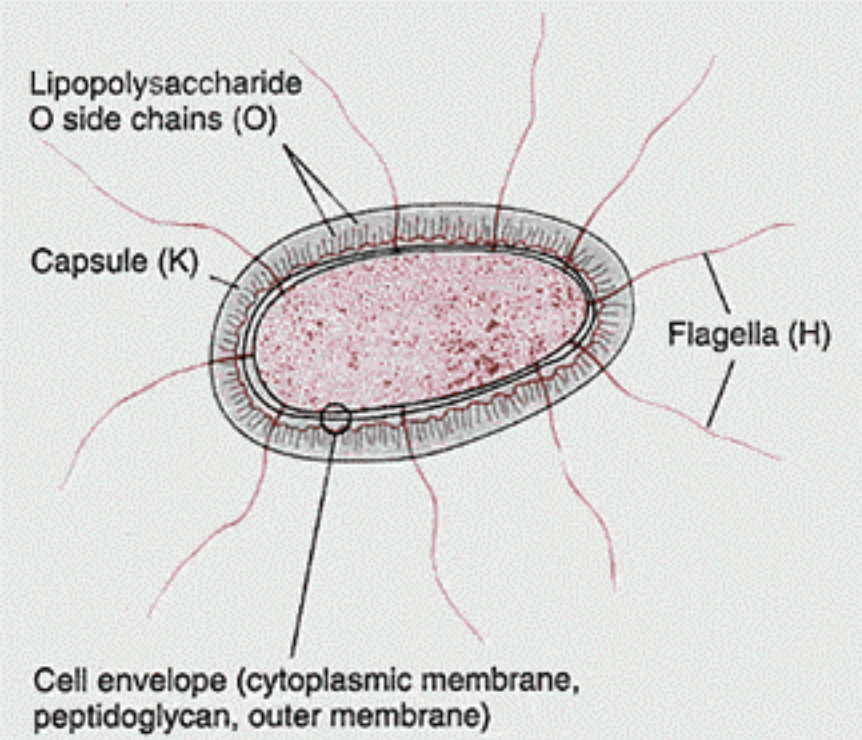


Figure 16–1. Antigenic structure of Enterobacteriaceae.

32k

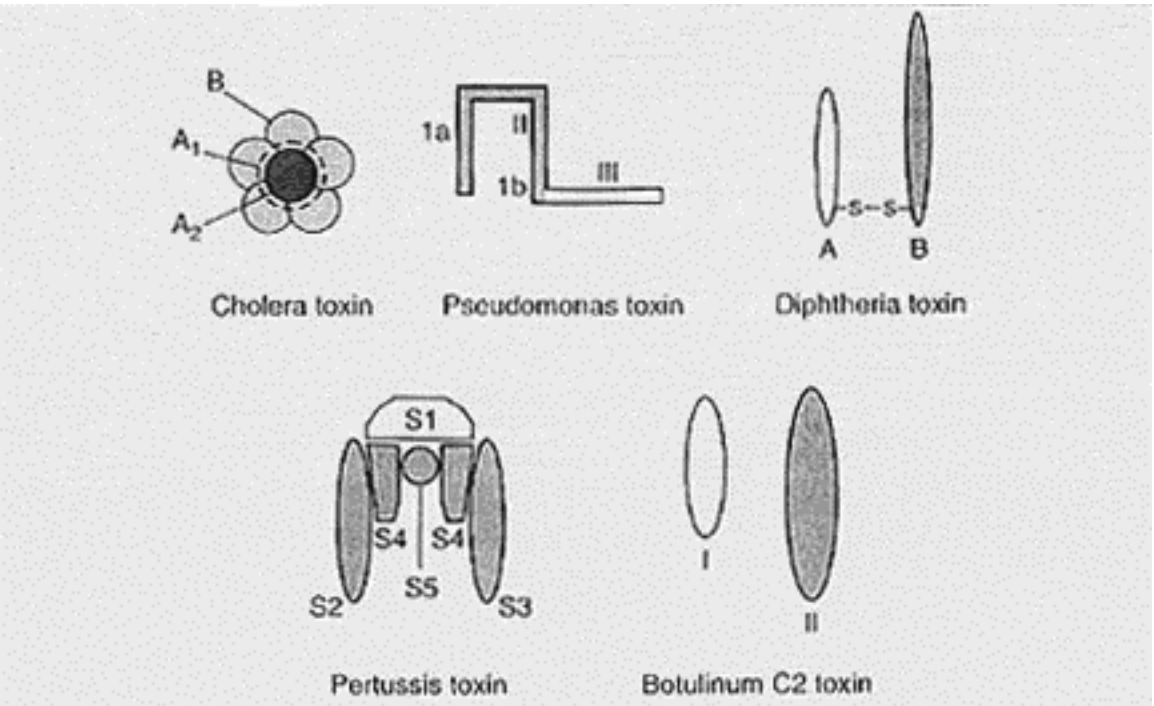


Fig. 8.5 Schematic structure of some ADP-ribosylating toxins. The hatched regions are the binding/translocation-facilitating parts. Cholera toxin is represented in plan view with A1 below the plane. Not to scale. (Taken with permission of authors (I. H. Madshus and H. Stenmark) and publisher (Springer-Verlag GmbH & Co. KG, Heidelberg, Germany) from Figure 1 in 'Entry of ADP-ribosylating toxins into cells' in *Current Topics in Microbiology and Immunology* (1992) **175**, 3, edited by Klaus Aktories.

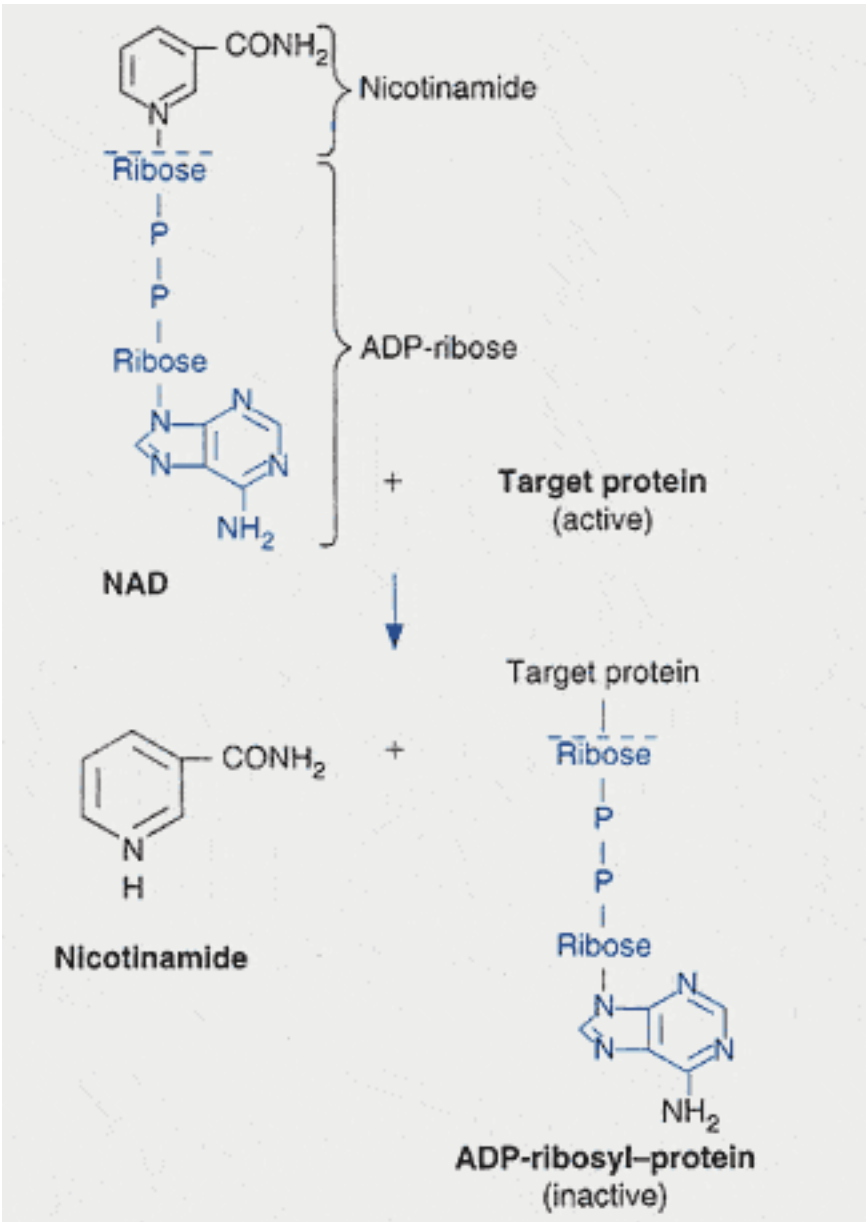
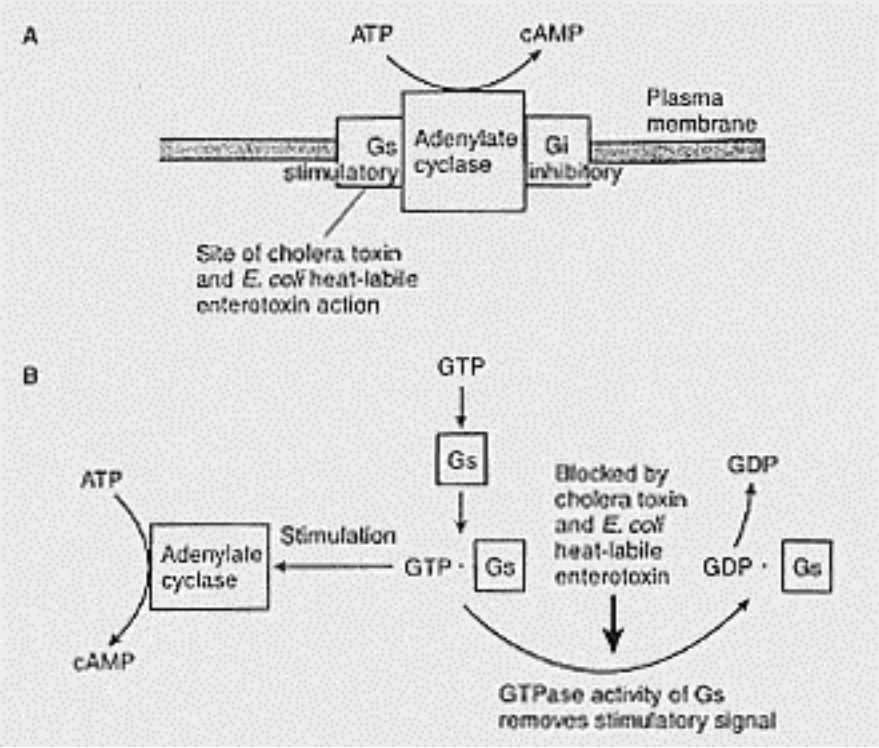


Figure 4-2 ADP-ribosylation of a target host protein. The ADP-ribosyl group is removed from NAD (dashed line) and covalently attached to a host cell target protein.

32m



32n

Table 5-1 Environmental signals that can control the expression of coordinately regulated virulence determinants in bacteria.[a]

Organism	Environmental signal(s)
Bordetella pertussis	Temperature, SO_4, nicotinic acid
Corynebacterium diphtheriae	Iron
Escherichia coli	Iron, temperature, carbon source
Listeria monocytogenes	Temperature
Pseudomonas aeruginosa	Iron, osmolarity
Salmonella typhimurium	Osmolarity, starvation, stress, pH, growth phase
Shigella spp.	Temperature
Staphylococcus aureus	Growth phase
Vibrio cholerae	Osmolarity, pH, temperature, amino acids, CO_2, iron
Yersinia spp.	Temperature, Ca^{2+}

[a] Adapted from J. J. Mekalanos. 1992. Environmental signals controlling expression of virulence gene determinants in bacteria. J. Bacteriol. 174:1–7.

32o