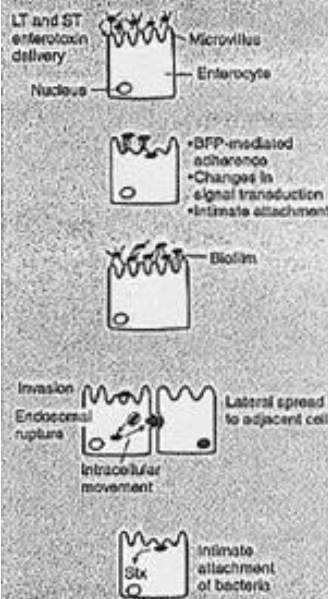


TABLE 12-7
Five Distinct Groups of *E. coli* that Cause Intestinal Disease



Disease	Virulence Factors			
	Adherence	Toxins	Siderophores	Invasion
Enterotoxigenic <i>Escherichia coli</i> (ETEC)	Colonization factors of adherence (CFAs) Type 1 pili	Endotoxin Heat-labile enterotoxin (LT) Heat-stable enterotoxin (STa)	Enterochelin	Noninvasive
Enteropathogenic <i>E. coli</i> (EPEC)	Bundle-forming pili (BFP) Type 1 pili Intimin	Endotoxin	Enterochelin	Poorly invasive
Enteraggregative <i>E. coli</i> (EAEC)	Mucus-associated autoagglutination Type 1 pili	Endotoxin Cytotoxin (enteroaggregative ST-like toxin [EAST])	Enterochelin	Noninvasive
Enteroinvasive <i>E. coli</i> (EIEC)	Type 1 pili Afimbrial adhesins	Endotoxin	Enterochelin	Type III secretion system Very invasive
Enterohemorrhagic <i>E. coli</i> (EHEC)	Type 1 pili Afimbrial adhesins	Shiga toxin Endotoxin	Enterochelin Heme uptake system	Probably poorly invasive

33a

TABLE 29-1 Gastroenteritis Caused by *Escherichia coli*

SITE OF ACTION	DISEASE	PATHOGENESIS
ENTEROTOXIGENIC <i>E. COLI</i> (ETEC) Small intestine	Traveler's diarrhea; infant diarrhea in underdeveloped countries; watery diarrhea, vomiting, cramps, nausea, low-grade fever	Plasmid-mediated heat-stable and/or heat-labile enterotoxins that stimulate hypersecretion of fluids and electrolytes
ENTEROINVASIVE <i>E. COLI</i> (EIEC) Large intestine	Disease in underdeveloped countries; fever, cramping, watery diarrhea followed by development of dysentery with scant, bloody stools	Plasmid-mediated invasion and destruction of epithelial cells lining colon
ENTEROPATHOGENIC <i>E. COLI</i> (EPEC) Small intestine	Infant diarrhea with fever, nausea, vomiting, nonbloody stools	Plasmid-mediated adherence and destruction of epithelial cells
ENTEROHEMORRHAGIC <i>E. COLI</i> (EHEC) Large intestine	Hemorrhagic colitis with severe abdominal cramps, initial watery diarrhea, followed by grossly bloody diarrhea; little or no fever; hemolytic uremic syndrome	Mediation by cytotoxic shiga-like toxins (SLT-I, SLT-II), which disrupt protein synthesis; toxins encoded by lysogenic bacteriophages
ENTEROAGGREGATIVE <i>E. COLI</i> (EAaggEC) Small intestine	Infant diarrhea in underdeveloped countries; persistent watery diarrhea with vomiting, dehydration, and low grade fever	Plasmid-mediated aggregative adherence that prevents fluid absorption

33b

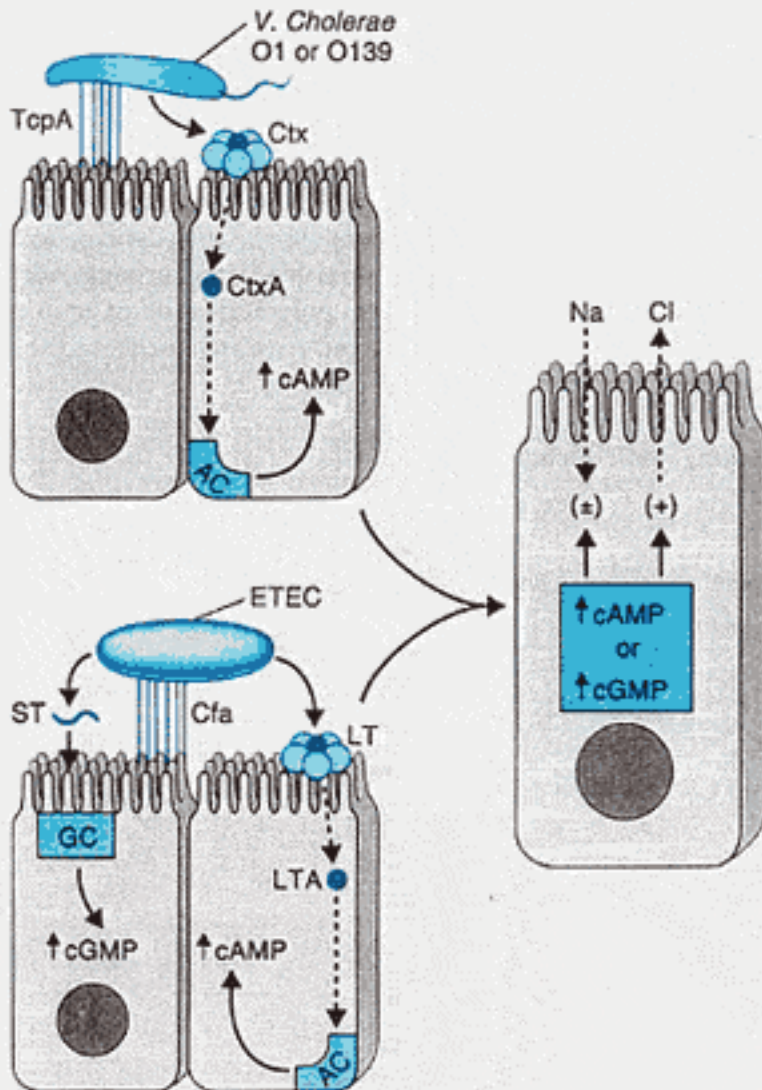


Figure 16.2. The pathogenesis of cholera and ETEC infections. The organisms colonize the mucosal surface via microbial adhesins, the toxin coregulated pilus (*TcpA*) of *V. cholerae* and the colonization factor antigen (*Cfa*) of enterotoxigenic *E. coli*. Cholera toxin (*Ctx*) or labile toxin (*LT*) binds to receptor, is taken up in vesicles, and is transported to the basolateral membrane to the adenylate cyclase (*AC*) complex. The toxins transfer ADP-ribose to the GTP-binding protein of *AC*, elevating cyclic-AMP. ETEC also produce a heat-stable (*ST*) toxin which binds to the membrane guanylate cyclase (*GC*) and increases cyclic GMP levels (*c-GMP*). Both *c-AMP* and *c-GMP* reduce Na^+ absorption in villus cells and increase Cl^- secretion in crypt cells, leading to watery diarrhea.

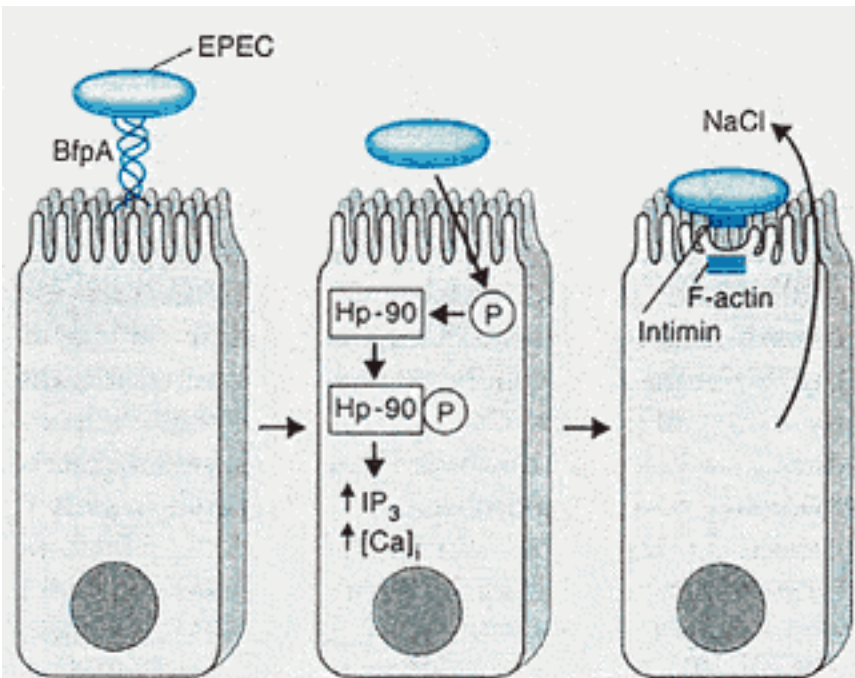
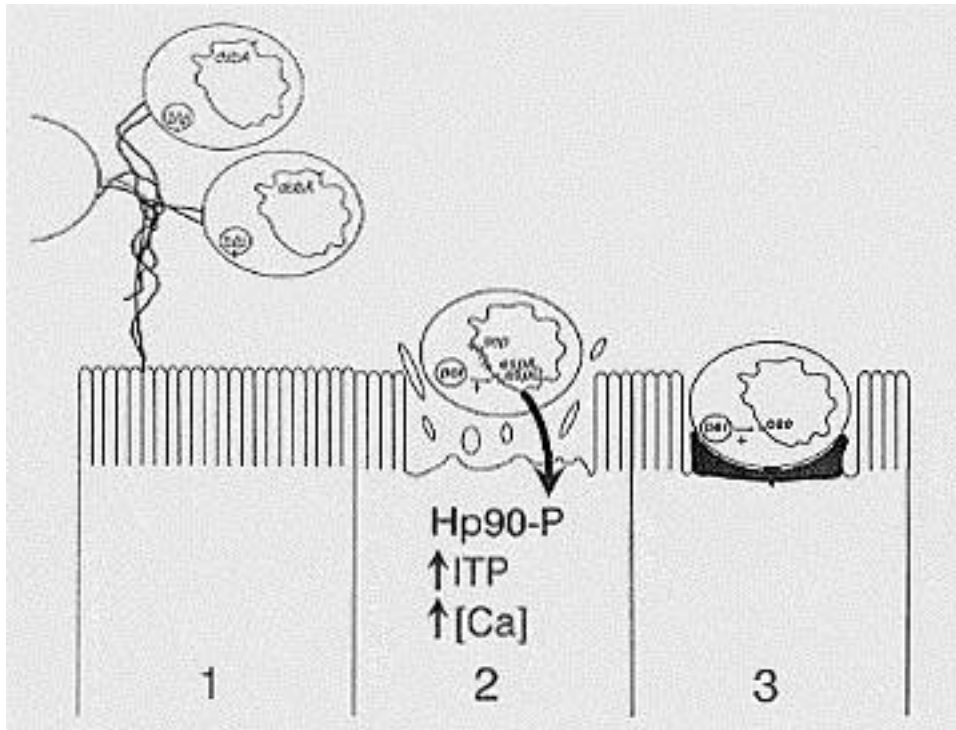
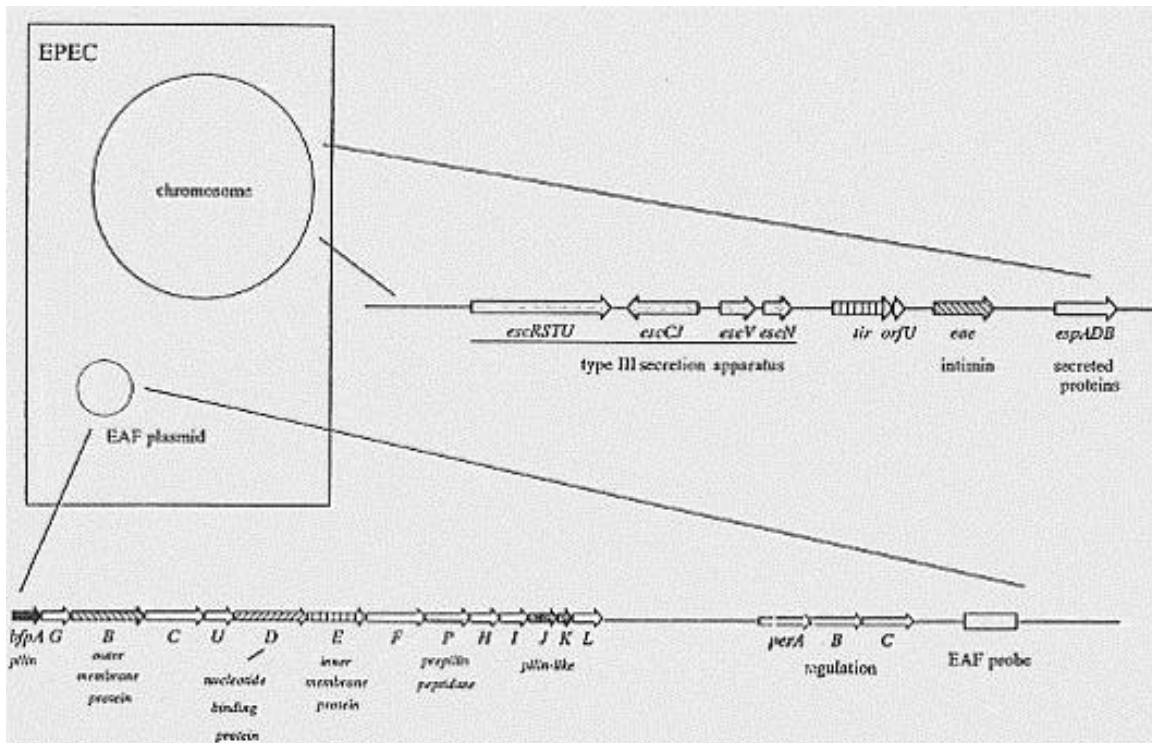


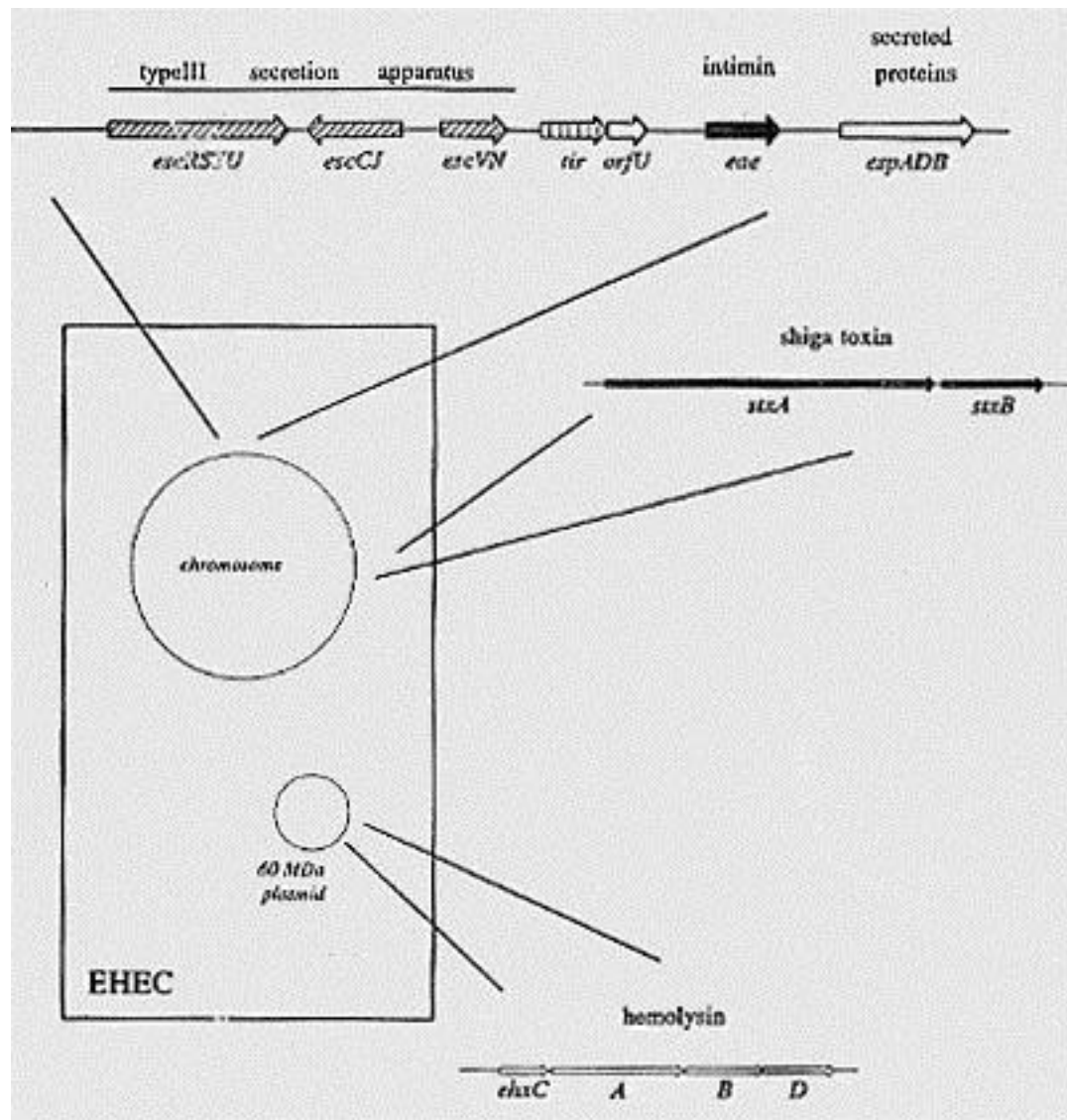
Figure 16.3. The pathogenesis of EPEC. First, the organism attaches to the small bowel epithelial cell via a bundle-forming pilus (*BfpA*). This binding sets in motion signal-transducing events involving phosphorylation of a major epithelial cell protein, Hp-90; activation of phospholipase C; increases in inositol triphosphate (IP_3) and calcium ($[Ca]_i$); and damage to the microvilli. In a third stage, intimin mediates intimate adherence, and a 39 kDa protein causes polymerization of actin and other host cytoskeletal proteins and rearrangements of the cytoskeletal structure. Together these form the characteristic EPEC pedestal with the intimately adherent organism (the attaching and effacing lesion).



33e



33f



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