TABLE 4. SUMMARY: ACTIVITIES OF EXTRACELLULAR BACTERIAL  TOXINS

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| NAME OF TOXIN | BACTERIA INVOLVED | ACTIVITY |
| Anthrax toxin (EF) | Bacillus anthracis | An adenylate cyclase enzyme that increases levels in intracellular cyclic AMP in phagocytes and formation of ion-permeable pores in cell membrane. Leads to edema and decreased phagocytic responses |
| Adenylate cyclase toxin (pertussis AC) | Bordetella pertussis | Acts locally to increase levels of cyclic AMP in phagocytes and formation of ion-permeable pores in cell membranes |
| Alpha toxin | Staphylococcus aureus | Protein subunits assemble into an oligomeric structure that forms an ion channel (pore) in the cell plasma membrane |
| Cholera enterotoxin (Ctx) | Vibrio cholerae | ADP ribosylation of G proteins stimulates adenlyate cyclase and increases cAMP in cells of the GI tract, causing secretion of water and electrolytes leading to diarrhea |
| E. coli LT toxin | Escherichia coli | Similar to cholera toxin |
| E. coli ST toxins | Escherichia coli | Binding of the heat-stable enterotoxins (ST) to a guanylate cyclase receptor results in an increase in cyclic GMP (cGMP) that adversely effects electrolyte flux. Promotes secretion of water and electrolytes from intestinal epithelium leading to diarrhea. |
| Shiga toxin | Shigella dysenteriae E. coli O157:H7 | Enzymatically cleaves eucaryotic 28S rRNA resulting in inhibition of protein synthesis in susceptible cells. Results in diarrhea, hemorrhagic colitis (HC) and hemolytic uremic syndrome (HUS) |
| Perfringens enterotoxin | Clostridium perfringens | Stimulates adenylate cyclase leading to increased cAMP in epithelial cells. Result is diarrhea |
| ToxinA/ToxinB | Clostridium difficile | Modifies Rho, a subfamily of small GTP-binding proteins that are regulators of the actin cytoskeleton. Deamidation of the glutamine residue at position 63 of Rho to a glutamic acid produces a dominant active Rho protein unable to hydrolyze bound GTP. Pathological result is cell necrosis and bloody diarrhea associated with colitis |
| Botulinum toxin | Clostridium botulinum | Zn++ dependent protease that inhibits neurotransmission at neuromuscular synapses resulting in flaccid paralysis |
| Tetanus toxin | Clostridium tetani | Zn++ dependent protease that Inhibits neurotransmission at inhibitory synapses resulting in spastic paralysis |
| Diphtheria toxin (Dtx) | Corynebacterium diphtheriae | ADP ribosylation of elongation factor 2 leads to inhibition of protein synthesis in target cells |
| Exotoxin A | Pseudomonas aeruginosa | Inhibits protein synthesis; similar to diphtheria toxin |
| Anthrax toxin (LF) | Bacillus anthracis | Lethal Factor (LF) is a Zn++dependent protease that induces cytokine release and is cytotoxic to cells by an unknown mechanism |
| Pertussis toxin (Ptx) | Bordetella pertussis | ADP ribosylation of G proteins blocks inhibition of adenylate cyclase in susceptible cells |
| Exfoliatin toxin\* | Staphylococcus aureus | Cleavage within epidermal cells (intraepidermal separation); also acts as a superantigen |
| Staphylococcus enterotoxins\* | Staphylococcus aureus | Superantigen causes massive activation of the immune system, including lymphocytes and macrophages; exact role in in emesis not not known |
| Toxic shock syndrome toxin (TSST-1)\* | Staphylococcus aureus | Superantigen acts on the vascular system causing inflammation, fever and shock |
| Erythrogenic toxin [streptococcal pyrogenic exotoxin (SPE)]\* | Streptococcus pyogenes | Super antigen same as TSST - inflammation, fever and shock; can cause localized erythematous reactions (scarlet fever) |

\* The pyrogenic exotoxins produced by Staphylococcus aureus andStreptococcus pyogenes have been designated as superantigens. They represent a family of molecules with the ability to elicit massive activation of the immune system. These proteins share the ability to stimulate T cell proliferation by interaction with Class II MHC molecules on APCs and specific V beta chains of the T-cell receptor. The important feature of this interaction is the resultant production of IL-1, TNF, and other lymphokines which appear to be the principal mediators of disease processes associated with these toxins.