Abstract

Cyclic adenosine monophosphate (cAMP) is an universal second messenger that regulates a large number of molecular mechanisms inside the eukaryotic cell. The level of synthesized cAMP is tightly regulated by endogenous adenylatecyclase (AC), and therefore this enzyme is often a target for various bacterial toxins. To manipulate intracellular cAMP levels in a target cell, bacteria have developed two different strategies for their toxins. *Bordetella pertussis* adenylate cyclase toxin (CyaA*), Bacillus anthracis* edema factor (EF) and *Pseudomonas aeruginosa* exotoxinY have in their structure an enzymatic AC domain which is activated by an intracellular cofactor and has several times higher activity than the eukaryotic AC enzyme itself. Other toxins, such as *Bordetella pertussis* pertussis toxin (PT), *Vibrio cholerae* cholera toxin (CT), and *Escherichia coli* heat labile toxin use ADP-ribosylation reaction of AC-coupled heterotrimeric G proteins to increase its activity and uncontrolled cAMP production. This work presents a literature search with accent on the molecular mechanism of interaction of these toxins with the target cell.

Keywords: bacterial pathogens, virulence factors, intracellular cAMP elevation, bacterial toxins, adenylatecyclase (adenylylcyclase), *Bordetella pertussis, Vibrio cholerae, Pseudomonas aeruginosa, Escherichia coli*