Bordetella pertussis is an important human pathogen that causes an infection disease called whooping cough. This gram-negative bacterium produces an adenylate cyclase toxin (CyaA) that recognizes an integrin receptor CD11b/CD18 present on the surface of myeloid phagocytes and delivers an adenylate cyclase (AC) domain into the cell cytosol. This thesis deals with the endocytic machinery of CyaA and its potential use as a specific marker for endocytosis of the CD11b/CD18 receptor molecule. Detoxified mutant of CyaA, CyaA-AC^-, that has the capacity to promote calcium influx as well the potassium efflux, was shown to trigger activation of the integrin receptor CD11b/CD18 followed with endocytic uptake by clathrin-dependent pathway. On the other side, the inactive mutant CyaA-KP-AC^- that is unable to provoke integrin activation was endocytosed by clathrin-independent pathway. These results suggest that the various endocytic pathways of the CD11b/CD18 are determined by different conformational states of the receptor molecule.