The membrane lipid bilayer acts as a natural barrier surrounding the cells that helps in protecting the cells from the outside environment. In addition to this, a membrane lipid bilayer plays a crucial role in compartmentalization of the cells, as well as in regulation of the exchange of materials in and out of a living cell. In doing so, the membrane lipid bilayer serves as the natural permeability barrier for every living cell. It is essential for the cells to communicate with the outer environment, and regulate the transport of ions, solvents, and small molecules across the membranes. Transport and signaling through the lipid bilayer are ubiquitous processes, and these are essential for the cells to react to the changes in the environment (1-3). The cells employ many specialized membranes proteins that act as the transporters or channels for the passage of ions, solvent and small molecules across the membrane lipid bilayer. Major class of the integral membrane proteins generate aqueous channels, through which molecules are transported. For example, the membrane protein channels found in the outer membrane of the Gram-negative bacteria, known as porins, act as the molecular sieves allowing the flow of small molecules with a particular size limit across the bacterial membranes (1,2,4,5). Therefore, biomembranes are equipped with a selective permeability barrier function that is very much essential for sustaining the living cells. However, the cytolytic pore-forming protein toxins, found in the vast array of organisms starting from bacteria to humans, destroy the selective permeability barrier function of the cell membranes by making pores on the membrane lipid bilayer of the target cells (1-6). The present thesis work deals with the structure-function relationship of Vibrio cholerae cytolysin (VCC), a potent membrane-damaging pore-forming protein toxin secreted by the Gram-negative bacteria V. cholerae, the causative agent of the severe diarrheal disease cholera.