

Abstract

The human disease cholera is caused by the gram negative bacteria *Vibrio cholerae* (*V. cholerae*). *V. cholerae* enters into the body through oral route and colonize in the intestine. Main symptoms of the disease are caused by an exotoxin named cholera toxin released by the pathogen upon entering into the host system. OmpU, a major porin protein of *V. cholerae* helps the bacteria for well survival in the gut and provides the resistance against the first line of host defense in terms of bile resistance and antibacterial peptide resistance. In our laboratory we observed that OmpU down-regulates pro-inflammatory cytokines like TNF- α and IL-6. My project was aimed at elucidating the role of OmpU in modulation of host chemokine responses such as, CCL2 and CCR2 which are important for monocyte and macrophage recruitment at the site of infection. We showed that in RAW 264.7 mouse macrophage cell line, OmpU down-regulates the chemokine CCL2 which is involved in monocyte recruitment from blood to the site of infection. Hence, it confirms the anti-inflammatory role of OmpU and suggests its involvement in pathogenesis by down-regulating the host responses.