Title:	Study of modulation of immune function of dendritic cells by Vibrio cholerae outer membrane OmpU/ Vinica Dhar
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Abstract:	OmpU is one of the major outer membrane porin proteins of human pathogen Vibrio cholerae. It aids in the survival of the bacterium in the gut during pathogenesis. Previous reports from our lab have highlighted the host immunomodulatory role of OmpU in innate immune cells (monocytes and macrophages). In the present study, we have shown that OmpU can modulate dendritic cell (DC) responses as well. DCs are the foremost antigen-presenting cells which decide the fate of adaptive immune responses. In this study, we have shown that OmpU induces pro-inflammatory responses in DCs via TLR2 pathway and also via activation of NLRP3 inflammasome. Towards probing the signalling pathway, we have observed that OmpU induces generation of mitochondrial ROS, which triggers NLRP3 inflammasome activation. Further probing revealed that calcium signalling is one of the contributors of OmpU-induced mitochondrial ROS generation in DCs. We have also observed that OmpU gets translocated to the mitochondria of DCs and OmpU translocation to the mitochondria also contributes to the mitochondrial ROS generation. Furthermore, we observed that, in addition to the mitochondrial ROS, OmpU induces cytoplasmic ROS that also contribute to the pro-inflammatory responses in DCs. OmpU induces NADPH oxidase (NOX) activation for production of cytoplasmic ROS. The NOX-mediated ROS depends on TLR2-mediated signalling; however, along with TLR2, scavenger receptor CD36 plays indispensable role in cytoplasmic ROS generation in DCs. Further probing of downstream signalling suggests that MAPK JNK is involved in the activation of NOX complex by OmpU in DCs.
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