Title:	Vibrio cholerae OmpU Mediates CD36-Dependent Reactive Oxygen Species Generation Triggering an Additional Pathway of MAPK Activation in Macrophages
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Abstract:	OmpU, one of the porins of Gram-negative bacteria Vibrio cholerae, induces TLR1/2–MyD88–NF-κB–dependent proinflammatory cytokine production by monocytes and macrophages of human and mouse origin. In this study, we report that in both the cell types, OmpU-induced proinflammatory responses involve activation of MAPKs (p38 and JNK). Interestingly, we observed that in OmpU-treated macrophages, p38 activation is TLR2 dependent, but JNK activation happens through a separate pathway involving reactive oxygen species (ROS) generation by NADPH oxidase complex and mitochondrial ROS. Further, we observed that OmpU-mediated mitochondrial ROS generation probably depends on OmpU translocation to mitochondria and NADPH oxidase–mediated ROS production is due to activation of scavenger receptor CD36. For the first time, to our knowledge, we are reporting that a Gram-negative bacterial protein can activate CD36 as a pattern recognition receptor. Additionally, we found that in OmpU-treated monocytes, both JNK and p38 activation is linked to the TLR2 activation only. Therefore, the ability of macrophages to employ multiple receptors such as TLR2 and CD36 to recognize a single ligand, as in this case OmpU, probably explains the very basic nature of macrophages being more proinflammatory than monocytes.
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