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Title:	Vibrio cholerae porin OmpU mediates M1-polarization of macrophages/monocytes via TLR1/TLR2 activation
Authors:	Khan, Junaid (/jspui/browse?type=author&value=Khan%2C+Junaid) Sharma, P.K. (/jspui/browse?type=author&value=Sharma%2C+P.K.) Mukhopadhyaya, Arunika (/jspui/browse?type=author&value=Mukhopadhyaya%2C+Arunika)
Keywords:	M1/M2 polarization OmpU Porin Toll-like receptor
Issue Date:	2015
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Abstract:	<p>Polarization of the monocytes and macrophages toward the M1 and M2 states is important for hosts' defense against the pathogens. Moreover, it plays a crucial role to resolve the overwhelming inflammatory responses that can be harmful to the host. Polarization of macrophages/monocytes can be induced by pathogen-associated molecular patterns (PAMPs). PAMP-mediated monocyte/macrophage polarization is important during the infection, as pathogen can suppress host immune system by altering the polarization status of the macrophages/monocytes. OmpU, an outer membrane porin protein of <i>Vibrio cholerae</i>, possesses the ability to induce pro-inflammatory responses in monocytes/macrophages. It is also able to down-regulate the LPS-mediated activation of the monocytes/macrophages. Such observation leads us to believe that OmpU may induce a state that can be called as M1/M2-intermediate state. In the present study, we evaluated a set of M1 and M2 markers in RAW 264.7 murine macrophage cell line, and THP-1 human monocytic cell line, in response to the purified OmpU protein. We observed that OmpU, as a PAMP, induced M1-polarization by activating the Toll-like receptor (TLR) signaling pathway. OmpU induced formation of TLR1/TLR2-heterodimers. OmpU-mediated TLR-activation led to the MyD88 recruitment to the TLR1/TLR2 complex. MyD88, in turn, recruited IRAK1. Ultimately, OmpU-mediated signaling led to the activation and subsequent nuclear translocation of the NFkB p65 subunit. We also observed that blocking of the TLR1, TLR2, IRAK1, and NFkB affected OmpU-mediated production of M1-associated pro-inflammatory cytokines such as TNF<math>\alpha</math> and IL-6.</p>
URI:	<a href="https://www.sciencedirect.com/science/article/pii/S0171298515300061">https://www.sciencedirect.com/science/article/pii/S0171298515300061</a> ( <a href="https://www.sciencedirect.com/science/article/pii/S0171298515300061">https://www.sciencedirect.com/science/article/pii/S0171298515300061</a> ) <a href="http://hdl.handle.net/123456789/2737">http://hdl.handle.net/123456789/2737</a> ( <a href="http://hdl.handle.net/123456789/2737">http://hdl.handle.net/123456789/2737</a> )
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