Title:	Transmembrane oligomeric form of Vibrio cholerae cytolysin triggers TLR2/TLR6-dependent proinflammatory responses in monocytes and macrophages
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Abstract:	Vibrio cholerae cytolysin (VCC) kills target eukaryotic cells by forming transmembrane oligomeric β -barrel pores. Once irreversibly converted into the transmembrane oligomeric form, VCCacquires an unusual structural stability and loses its cytotoxic property. It is therefore possible that, on exertion of its cytotoxic activity, the oligomeric form of VCC retained in the disintegrated membrane fractions of the lysed cells would survive within the host cellular milieu for a long period, without causing any further cytotoxicity. Under such circumstances, VCC oligomers may potentially be recognized by the host immune cells. Based on such a hypothesis, in the present study we explored the interaction of the transmembrane oligomeric form of VCC with the monocytes and macrophages of the innate immune system. Our study shows that the VCC oligomers assembled in the liposome membranes elicit potent proinflammatory responses in monocytes and macrophages, via stimulation of the tolllike receptor (TLR)2/TLR6-dependent signalling cascades that involve myeloid differentiation factor 88 (MyD88)/interleukin-1-receptor-associated kinase (IRAK)1/tumour-necrosis-factor-receptor-associated factor (TRAF)6. VCC oligomer-mediated proinflammatory responses critically depend on the activation of the transcription factor nuclear factor-κ B. Proinflammatory responses induced by the VCC oligomers also require activation of the mitogen-activated protein kinase (MAPK) family member c-Jun N-terminal kinase, which presumably acts via stimulation of the transcription factor activator protein-1. Notably, the role of the MAPK p38 could not be documented in the proces
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