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Title:	Vibrio cholerae porin OmpU induces LPS tolerance by attenuating TLR-mediated signaling
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Abstract:	Porins can act as pathogen-associated molecular patterns, can be recognized by the host immune system and modulate immune responses. Vibrio choleraeporin OmpU aids in bacterial survival in the human gut by increasing resistance against bile acids and anti-microbial peptides. V. choleraeOmpU is pro-inflammatory in nature. However, interestingly, it can also down-regulate LPS-mediated pro-inflammatory responses. In this study, we have explored how OmpU-pretreatment affects LPS-mediated responses. Our study indicates that OmpU-pretreatment followed by LPS-activation does not induce M2-polarization of macrophages/monocytes. Further, OmpU attenuates LPS-mediated TLR2/TLR6 signaling by decreasing the association of TLRs along with recruitment of MyD88 and IRAKs to the receptor complex. This results in decreased translocation of NFκB in the nucleus. Additionally, OmpU-pretreatment up-regulates expression of IRAK-M, a negative regulator of TLR signaling, in RAW 264.7 mouse macrophage cells upon LPS-stimulation. Suppressor cytokine IL-10 is partially involved in OmpU-induced down-regulation of LPS-mediated TNFα production in human PBMCs. Furthermore, OmpU-pretreatment also affects macrophage function, by enhancing phagocytosis in LPS-treated RAW 264.7 cells, and down-regulates LPS-induced cell surface expression of co-stimulatory molecules. Altogether, OmpU causes suppression of LPS-mediated responses by attenuating the LPS-mediated TLR signaling pathway.
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