Title:	Revisiting the membrane interaction mechanism of a membrane-damaging β-barrel pore-forming toxin Vibrio cholerae cytolysin
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Abstract:	Vibrio cholerae cytolysin (VCC) permeabilizes target cell membranes by forming transmembrane oligomeric β-barrel pores. VCC has been shown to associate with the target membranes via amphipathicity-driven spontaneous partitioning into the membrane environment. More specific interaction(s) of VCC with the membrane components have also been documented. In particular, specific binding of VCC with the membrane lipid components is believed to play a crucial role in determining the efficacy of the pore-formation process. However, the structural basis and the functional implications of the VCC interaction with the membrane lipids remain unclear. Here we show that the distinct loop sequences within the membrane-proximal region of VCC play critical roles to determine the functional interactions of the toxin with the membrane lipids. Alterations of the loop sequences via structure-guided mutagenesis allow amphipathicity-driven partitioning of VCC to the membrane lipid bilayer. Alterations of the loop sequences, however, block specific interactions of VCC with the membrane lipids and abort the oligomerization, membrane insertion, pore-formation and cytotoxic activity of the toxin. Present study identifies the structural signatures in VCC implicated for its functional interactions with the membrane lipid components, a process that presumably acts to drive the subsequent steps of the oligomeric β-barrel pore-formation and cytotoxic responses.
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