Title:	Vibrio cholerae porin ompu induces caspase-independent programmed cell death upon translocation to the host cell mitochondria
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Abstract:	Porins, a major class of outer membrane proteins in Gramnegative bacteria, primarily act as transport channels. OmpU is one of the major porins of human pathogen, Vibrio cholerae. In the present study, we show that V. cholerae OmpU has the ability to induce target cell death. Although OmpU-mediated cell death shows some characteristics of apoptosis, such as flipping of phosphatidylserine in the membrane as well as cell size shrinkage and increased cell granularity, it does not show the caspase-3 activation and DNA laddering pattern typical of apoptotic cells. Increased release of lactate dehydrogenase in OmpU-treated cells indicates that the OmpU-mediated cell death also has characteristics of necrosis. Further, we show that the mechanism of OmpU-mediated cell death involves major mitochondrial changes in the target cells. We observe that OmpUtreatment leads to the disruption of mitochondrialmembrane potential, resulting in the release of cytochrome c and apoptosis-inducing factor (AIF). AIF translocates to the host cell nucleus, implying that it has a crucial role in OmpU-mediated cell death. Finally, we observe that OmpU translocates to the target cell mitochondria, where it directly initiates mitochondrial changes leading to mitochondrial membrane permeability transition and AIF release. Partial blocking of AIF release by cyclosporine A in OmpU-treated cells further suggests that OmpU may be inducing the opening of the mitochondrial permeability transition pore. All of these results lead us to the conclusion that OmpU induces cell death in target cells in a programmed manner in which mitochondria play a central role. © 2015 by The American Society for Biochemistry and Molecular Biology, Inc.
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