Title:	Functional Mapping of the Lectin Activity Site on the β-Prism Domain of Vibrio cholerae Cytolysin IMPLICATIONS FOR THE MEMBRANE PORE-FORMATION MECHANISM OF THE TOXIN*
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Keywords:	β-Trefoil Vibrio cholerae cytolysin Eukaryotic cells Cytolysin
Issue Date:	2013
Publisher:	The American Society for Biochemistry and Molecular Biology, Inc.
Citation:	Journal of Biological Chemistry,288(3), pp.1665-1673.
Abstract:	Background: Vibrio cholerae cytolysin is a pore-forming toxin with lectin-like activity. Results:β-Prism domain mediates the lectin activity, which determines efficient membrane binding and modulates membrane oligomerization of the toxin. Conclusion:β-Prism domain-mediated lectin activity acts to play critical regulatory role in the mode of action of the toxin. Significance: The study elucidates novel implications of the β-Prism domain-mediated lectin activity. Vibrio cholerae cytolysin (VCC) is a prominent member in the family of β-barrel pore-forming toxins. It induces lysis of target eukaryotic cells by forming transmembrane oligomeric-barrel channels. VCC also exhibits prominent lectin-like activity in interacting with β1-galactosyl-terminated glycoconjugates. Apart from the cytolysin domain, VCC harbors two lectin-like domains: the-Trefoil and the-Prism domains; however, precise contribution of these domains in the lectin property of VCC is not known. Also, role(s) of these lectin-like domains in the mode of action of VCC remain obscure. In the present study, we show that the β-Prism domain of VCC acts as the structural scaffold to determine the lectin activity of the protein toward β1-galactosyl-terminated glycoconjugates. Toward exploring the physiological implication of the β-Prism domain, we demonstrate that the presence of the-Prism domain-mediated lectin activity is crucial for an efficient interaction of the toxin toward the target cells. Our results also suggest that such lectin activity may act to regulate the oligomerization ability of the membrane-boundVCCtoxin. Based on the data presented here, and also consistent with the existing structural information, we propose a novel mechanism of regulation imposed by the β-Prism domain's lectin activity, implicated in the process of membrane pore formation by VCC.
URI:	https://www.jbc.org/content/288/3/1665 (https://www.jbc.org/content/288/3/1665) http://hdl.handle.net/123456789/3013 (http://hdl.handle.net/123456789/3013)
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