

A phospholipid-methylating enzyme with membrane-deforming ability in *Agrobacterium tumefaciens*

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The contribution of membrane lipids to fundamental biological processes including cell differentiation, stress response or virulence is well established. *Agrobacterium tumefaciens* membranes are characterized by an unusual composition with the typical eukaryotic lipid phosphatidylcholine (PC) as a major component. Loss of phosphatidylcholine (PC) in *A. tumefaciens* is accompanied by a lack of tumor induction. Two different enzymes are responsible for PC formation. The integral membrane protein PC synthase (Pcs) uses CDP-DAG and choline to produce PC. The peripheral phospholipid *N*-methyltransferase (PmtA) utilizes phosphatidylethanolamine (PE) as substrate and the methyl donor S-adenosylmethionine for PC formation. Here, PmtA methylates the amino moiety of PE by three successive steps via the intermediates monomethyl-PE (MMPE) and dimethyl-PE (DMPE) to form PC(1).

We characterized the enzymatic properties and membrane interaction of PmtA *in vitro* and found that PmtA is a versatile enzyme not only involved in PC synthesis but also able to transform uniform spherical vesicles into either tubules or nanovesicles dependent on the lipid composition. Membrane deformation is independent of its methylation activity and requires the presence of the anionic lipid cardiolipin. In the absence of MMPE and DMPE vesicles are tubulated whereas membranes containing one of these lipids are transformed into smaller vesicles rather than to filaments. We identified and characterized two helical membrane-binding regions in PmtA with the N-terminal α A playing a central role in both membrane-binding and - deformation activity via two spatially separated regions. The biological function of PmtA-mediated membrane deformation remains to be established.

(1) Wessel M, Klüsener S, Gödeke J, Fritz C, Hacker S, Narberhaus F (2006). Virulence of *Agrobacterium tumefaciens* requires phosphatidylcholine in the bacterial membrane. *Mol Microbiol* . 62 :906-915