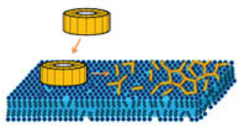
Communicative & Integrative Biology



ISSN: (Print) 1942-0889 (Online) Journal homepage: https://www.tandfonline.com/loi/kcib20

Membrane chaperoning by members of the PspA/IM30 protein family

Adrien Thurotte, Thomas Brüser, Thorsten Mascher & Dirk Schneider

To cite this article: Adrien Thurotte, Thomas Brüser, Thorsten Mascher & Dirk Schneider (2017) Membrane chaperoning by members of the PspA/IM30 protein family, Communicative & Integrative Biology, 10:1, e1264546, DOI: 10.1080/19420889.2016.1264546

To link to this article: https://doi.org/10.1080/19420889.2016.1264546

COMMUNICATIVE & INTEGRATIVE BIOLOGY
2017, VOL. 10, NO. 1, e1264546 (6 pages)
http://dx.doi.org/10.1080/19420889.2016.1264546

Taylor & Francis
Taylor & Francis Group

ARTICLE ADDENDUM

OPEN ACCESS

Membrane chaperoning by members of the PspA/IM30 protein family

Adrien Thurotte[a], Thomas Brüser[b], Thorsten Mascher[c], and Dirk Schneider[a]

[a]Institut für Pharmazie und Biochemie, Johannes Gutenberg-Universität Mainz, Mainz, Germany; [b]Institut für Mikrobiologie, Leibniz Universität Hannover, Hannover, Germany; [c]Institut für Mikrobiologie, Technische Universität Dresden, Dresden, Germany

ABSTRACT

PspA, IM30 (Vipp1) and LiaH, which all belong to the PspA/IM30 protein family, form high molecular weight oligomeric structures. For all proteins membrane binding and protection of the membrane structure and integrity has been shown or postulated. Here we discuss the possible membrane chaperoning activity of PspA, IM30 and LiaH and propose that larger oligomeric structures bind to stressed membrane regions, followed by oligomer disassembly and membrane stabilization by protein monomers or smaller/different oligomeric scaffolds.

ARTICLE HISTORY
Received 27 October 2016
Revised 18 November 2016
Accepted 18 November 2016

KEYWORDS
IM30; LiaH; membrane chaperone; membrane stress; oligomer; PspA; PspA/IM30 family; Vipp1; YjfJ

PspA, LiaH and IM30 are phylogenetically related proteins

The proteins PspA, IM30 (Vipp1), LiaH and YjfJ belong to the PspA/IM30 protein family. Structurally, all family members are predicted to be mainly α–helical,[1-4] and PspA, IM30 and LiaH form high-molecular weight homo-oligomeric ring structures. Solely YjfJ is reported to not form such complexes,[5] and, in fact, YjfJ is the most distant and least studied protein in this family. Therefore, it will not be further discussed here. The tendency of purified PspA, LiaH and IM30 to form ring structures or other higher order oligomers is extremely pronounced and monomers represent a minor fraction in solution.[6-9] The observed homo-oligomeric rings are strikingly similar: PspA has been calculated to form 36-mers, and LiaH also forms ring structures of that size range.[3,7] IM30 has the intrinsic propensity to form varying ring sizes containing (at least) 48–68 monomers.[1,2] Nevertheless, besides a common phylogenic origin and obvious structural similarities, PspA, IM30 and LiaH have different physiological functions.

PspA, IM30 and LiaH bind and stabilize stressed membranes

The phage shock protein (Psp) system represents a conserved stress response system of bacteria and archaea.[10] In enterobacteria, Psp response proteins are encoded by the *pspABCDE* operon and monocistronic *pspF* and *pspG* genes. Thus far, the enterobacterial Psp system is the best characterized representative involving a member of the PspA/IM30 family.

In the absence of stress, PspA inhibits the transcription factor PspF.[11] One model suggests that the Psp system is activated when PspA itself senses membrane defects or stress, resulting in liberation of PspF.[12] As a result, transcription of the *psp* genes is enhanced and the PspA level increases, which triggers the formation of membrane-protecting PspA assemblies.[13] In agreement with this hypothesis, PspA can stabilize stressed membranes *in vitro*.[14] Alternatively, the main sensory role is attributed to the membrane components PspB and PspC.[15-17] In this model, membrane stress results in recruitment of PspA to PspC, and a concomitant release of PspF activates the Psp response. Indeed, sensing of mistargeted secretins or membrane-destabilizing proteins requires PspB and PspC, which may function in conjunction with PspA.[18,19] In non-enterobacteria, PspBC may be replaced by other sensory systems. Recent analyses of the PspA-PspF interaction suggest that the PspAF complex does not necessarily need to dissociate to modulate *psp* gene expression.[20] In summary, the function of PspA appears to be dual: (i) membrane binding and membrane protection as well as (ii) upregulation of corresponding genes.

CONTACT Dirk Schneider Dirk.Schneider@uni-mainz.de Johannes Gutenberg-University Mainz, Department of Pharmacy and Biochemistry, Johann-Joachim-Becher-Weg 30, 55128 Mainz, Germany.

Addendum to: Heidrich J, Wulf V, Hennig R, Saur M, Markl J, Sönnichsen C, Schneider D. Organization into higher-ordered ring structures counteracts membrane binding of IM30, a protein associated with inner membranes in chloroplasts and cyanobacteria. J Biol Chem 2016; 291:14954-62; http://dx.doi.org/10.1074/jbc.M116.722686

IM30 (Inner Membrane-associated protein of 30 kDa), alternatively named Vipp1 (Vesicle-Inducing Protein in Plastids), is conserved in all organisms harboring thylakoid membranes (TMs), i.e. cyanobacteria (except several *Prochlorococcus marinus strains*), algae and higher plants.[21] It has been reported that IM30 can somehow functionally replace PspA in *Escherichia coli*.[22,23] Thus, while membrane binding and membrane protection appears to be conserved, IM30 must have gained extra functions. Although many IM30-specific functions have been proposed in the past (for a recent review, see ref. 21), a recent report suggests that IM30 can trigger membrane fusion upon membrane binding, a process crucial for TM biogenesis and maintenance.[24] A fusogenic activity would in fact explain many of the previously proposed IM30 activities.

The Lia system is highly conserved in *Bacillus* and *Listeria* species (low GC Gram-positive bacteria).[25] Its expression is controlled by the LiaFSR three-component system.[26] In the absence of stress conditions, the bifunctional histidine kinase LiaS is kept in its phosphatase state by the inhibitor protein LiaF, thereby repressing the activity of the transcriptional factor LiaR.[27] Under stress conditions, LiaF presumably releases LiaS, which switches its activity from phosphatase to kinase state. Once activated by phosphorylation, LiaR induces transcription of the *liaIH* operon.[26,27] LiaI is the membrane anchor of LiaH, which dynamically scans the membrane and recruits LiaH to the site of envelope damages.[28] It is postulated that oligomeric LiaI/LiaH complexes stabilize a perturbed envelope at the sites of membrane damage.[28]

Are members of the PspA/IM30 protein family membrane chaperones?

While all studied members of the PspA/IM30 family have acquired specific functions during the course of evolution, PspA[3,5,12,29,30] and IM30[1,2,4,6,12,31-33] have been shown to directly bind to membrane surfaces and thereby to alter the structure and organization of membranes eventually resulting in membrane stabilization. LiaH is suggested to have the same activity, but experimental verification is still missing.[28]

PspA and IM30 preferentially bind to negatively charged membranes or to surface curvature-stressed membranes.[6,12,24] Surface attachment of IM30 or PspA increases the packing density of individual lipids, i.e., the lipid bilayer becomes more ordered.[6,12] Increasing the lipid packing density might be a vital repair strategy if stress-induced defects occur in lipid bilayers. Membrane stress resulting in membrane reorganization and eventually in membrane defects might be induced by heat or osmotic stress, by membrane remodeling agents, such as alcohols, as well as by external forces. Importantly, the major lipid species of bacterial as well as TMs are non-bilayer forming lipids (such as PE (Phosphatidylethanolamine) in *E. coli* and MGDG (Monogalactosyldiacylglycerol) in cyanobacterial/chloroplasts' TMs). While MGDG is crucial for TM organization,[34] excess of MGDG severely decreases the membrane stability.[35] In fact, under prolonged stress affecting membrane integrity or fluidity, the MGDG content is adjusted.[36,37] Membrane binding of PspA or IM30 proteins might therefore simply spatiotemporally stabilize short-living protein-free (instable) lipid patches by insertion of the hydrophobic side-chains of a membrane-binding amphipathic helix between the lipid acyl-chains of the cytoplasmic lipid bilayer leaflet. Such membrane stabilizing mechanism is discussed for many membrane-active proteins or peptides that contain amphipathic helices, such as antimicrobial peptides[38-40] or BAR (Bin/Amphiphysin/Rvs)-domain proteins.[41,42] In case of negative curvature stress or other defects in lipid bilayer structures, binding of PspA or IM30 proteins to membrane surfaces might stabilize the bilayer structure by the discussed hydrophobic insertion mechanism.

The ability of selected proteins to bind and to protect stressed membranes is important for the fitness and survival of microorganisms and plants, which are constantly exposed to different stresses, like cold, heat, reactive oxygen species (generated, among other, by an intense photosynthetic activity), acid or organic solvents.[43-46] Membrane binding and protection has already been described for oligomeric proteins belonging to the Small Heat Shock Protein (sHSP) family. sHSPs are low-molecular weight proteins (15–42kDa) that form dynamic 9- to 50-mers.[47] However, the sHSPs active unit is most certainly a dimer.[48] Hence, dimer formation requires dissociation of the oligomeric structure. Under heat stress conditions, the cyanobacterial sHSP HspA (also named Hsp17) delocalizes from the cytoplasm and binds to TMs to protect the membrane ultrastructure[49] by increasing the lipid order and reducing membrane fluidity.[50] Lo18, a sHSP from *Oenococcus oeni*, forms 16-mer spherical complexes.[51] Under ethanol-induced membrane stress, 16-mer complexes disassemble into dimers that bind to membranes and stabilize them.[52] Furthermore, expression of the *O. oeni* Lo18 in *Lactococcus lactis* improved acid stress tolerance of *L. lactis*,[53] and heterologous expression of HSP17 from *Caenorhabditis elegans* enabled *E. coli* to grow at temperatures that are normally lethal for the wild-type strain.[54]

It is tempting to postulate a similar role for members of the PspA/IM30 family, that is, that the oligomers dissociate on membrane surfaces upon membrane binding to stabilize defined membrane patches. In fact, for

PspA[11,20] and IM30[6,24] dissociation of their large ring assemblies is indicated. IM30 LMWO (lower molecular weight oligomers) bind with an about 5-fold higher affinity to PG liposomes than IM30 rings,[6] which argues for binding of monomers rather than high molecular mass oligomers to membrane surfaces and/or IM30 ring disassembly upon membrane binding.[24] Increased membrane affinity of LMWO (lower molecular weight oligomers) IM30 is discussed to originate from exposure of amphiphilic helices, which are otherwise involved in formation of coiled-coil-type interactions within IM30 rings.[21] Since LMWO (lower molecular weight oligomers) appear to not be stable in solution, formation of the ring structures might simply be vital for shielding the amphiphilic helices. Thereby, PspA or IM30 proteins remain soluble in the absence of stress conditions.

Binding of an IM30 ring to TMs requires a free lipid area of about 380 nm^2 (total surface of the ring considered as a disc with an assumed ring diameter of 22 nm).[2] In *Synechocystis* PCC 6803 TMs, the proportion of proteins (weight/weight) is 68%[55] and in spinach TMs, the proteins area occupancy has been estimated to be 75%.[56] Thus, binding of IM30 rings seems barely probable *in vivo* within the crowded environment of a TM, unless stressed membrane areas are protein depleted or an unknown mechanism creates such large protein-free lipid domains. In fact, crowding is not homogeneous in cyanobacterial and plant TMs that possess lateral heterogeneity with densely packed and more fluid areas.[57,58] However, while a single IM30 ring covers an area of ~380 nm^2, the center of a ring is open leaving a free area of ~80 nm^2.[2] Binding of smaller units (such as monomers) or flexibly rearranging scaffolds to damaged membranes could completely cover and protect damaged and/or stressed membranes by stabilizing the order of the lipid bilayer via filling the "gaps" between lipid head groups.

Summary and outlook

Functional characterization of PspA/IM30 proteins has been notoriously difficult. But combining the lessons learned from its best studied members – the PspA from enterobacteria, IM30 (Vipp1) from cyanobacteria and plants, and LiaH from Firmicutes– allowed extracting a first set of common features with respect to their oligomerization, membrane binding and putative membrane protecting function, as discussed here. We propose that membrane protection of members of the PspA/IM30 protein family is mediated by (1.) binding of oligomeric/multimeric assemblies or ring structures to stressed membrane areas followed (2.) by ring dissociation, which results in exposure of membrane-binding amphiphilic helices and finally in coverage of a stressed membrane area by monomers or flexible scaffolds (Fig. 1). Notably, membrane attachment of oligomeric proteins and subsequent local disassembly results in a high monomer concentration that is confined to a selected membrane area.

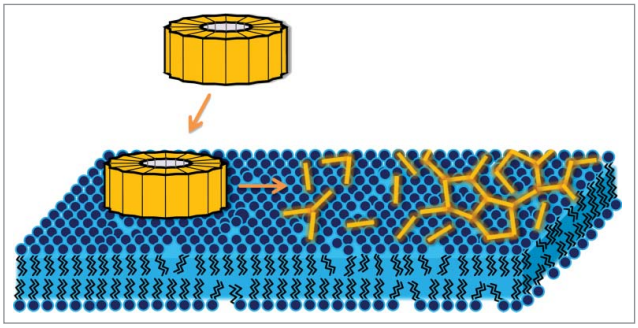


Figure 1. PspA/IM30 membrane binding and ring dissociation. Members of the PspA/IM30 family appear to have membrane protective activity. Likely, membrane protection is mediated by binding of oligomeric/multimeric (ring) assemblies to stressed membrane area followed by ring dissociation and coverage of a stressed membrane area by monomers or flexible scaffolds.

Clearly, besides the proposed membrane-protective activity of PspA/IM30 proteins, they have gained more specific functions in their special systems, and these activities are likely differently regulated. However, the here described insights can now serve as a starting point for more detailed functional and biochemical analyses that will ultimately unravel both general and organism-specific features of the ubiquitously distributed PspA/IM30 protein family.

Disclosure of potential conflicts of interest

No potential conflicts of interest were disclosed.

Funding

This work was supported by the University of Mainz; and the Ernst & Margarete Wagemann Foundation.

References

[1] Bultema JB, Fuhrmann E, Boekema EJ, Schneider D. Vipp1 and PspA: Related but not twins. Commun Integr Biol 2010; 3(2):162-5; PMID:20585511; http://dx.doi.org/10.4161/cib.3.2.10529

[2] Fuhrmann E, Bultema JB, Kahmann U, Rupprecht E, Boekema EJ, Schneider D. The vesicle-inducing protein 1 from *Synechocystis* sp. PCC 6803 organizes into diverse higher-ordered ring structures. Mol Biol Cell 2009; 20 (21):4620-8; PMID:19776353; http://dx.doi.org/10.1091/mbc.E09-04-0319

[3] Hankamer BD, Elderkin SL, Buck M, Nield J. Organization of the AAA(+) adaptor protein PspA is

an oligomeric ring. J Biol Chem 2004; 279(10):8862-6; PMID:14688274; http://dx.doi.org/10.1074/jbc.M307889200

[4] Otters S, Braun P, Hubner J, Wanner G, Vothknecht UC, Chigri F. The first alpha-helical domain of the vesicle-inducing protein in plastids 1 promotes oligomerization and lipid binding. Planta 2013; 237(2):529-40; PMID:23053543; http://dx.doi.org/10.1007/s00425-012-1772-1

[5] Jovanovic G, Mehta P, McDonald C, Davidson AC, Uzdavinys P, Ying L, Buck M. The N-terminal amphipathic helices determine regulatory and effector functions of phage shock protein A (PspA) in *Escherichia coli*. J Mol Biol 2013; 426:1498-511; PMID:24361331; http://dx.doi.org/10.1016/j.jmb.2013.12.016

[6] Heidrich J, Wulf V, Hennig R, Saur M, Markl J, Sonnichsen C, Schneider D. Organization into higher-ordered ring structures counteracts membrane binding of IM30, a protein associated with inner membranes in chloroplasts and cyanobacteria. J Biol Chem 2016; 291:14954-62; PMID:27226585; http://dx.doi.org/10.1074/jbc.M116.722686

[7] Wolf D, Kalamorz F, Wecke T, Juszczak A, Mader U, Homuth G, Jordan S, Kirstein J, Hoppert M, Voigt B. and others. In-depth profiling of the LiaR response of *Bacillus subtilis*. J Bacteriol 2010; 192(18):4680-93; PMID:20639339; http://dx.doi.org/10.1128/JB.00543-10

[8] Kobayashi R, Suzuki T, Yoshida M. *Escherichia coli* phage-shock protein A (PspA) binds to membrane phospholipids and repairs proton leakage of the damaged membranes. Mol Microbiol 2007; 66(1):100-9; PMID:17725563; http://dx.doi.org/10.1111/j.1365-2958.2007.05893.x

[9] Standar K, Mehner D, Osadnik H, Berthelmann F, Hause G, Lünsdorf H, Bruser T. PspA can form large scaffolds in *Escherichia coli*. FEBS Lett 2008; 582(25-26):3585-9; PMID:18789328; http://dx.doi.org/10.1016/j.febslet.2008.09.002

[10] Vothknecht UC, Otters S, Hennig R, Schneider D. Vipp1: a very important protein in plastids?! J Exp Bot 2012; 63(4):1699-712; PMID:22131161; http://dx.doi.org/10.1093/jxb/err357

[11] Joly N, Burrows PC, Engl C, Jovanovic G, Buck M. A lower-order oligomer form of phage shock protein A (PspA) stably associates with the hexameric AAA(+) transcription activator protein PspF for negative regulation. J Mol Biol 2009; 394(4):764-75; PMID:19804784; http://dx.doi.org/10.1016/j.jmb.2009.09.055

[12] McDonald C, Jovanovic G, Ces O, Buck M. Membrane stored curvature elastic stress modulates recruitment of maintenance proteins PspA and Vipp1. MBio 2015; 6(5); http://dx.doi.org/10.1128/mBio.01188-15

[13] Mehta P, Jovanovic G, Ying L, Buck M. Is the cellular and molecular machinery docile in the stationary phase of *Escherichia coli*? Biochem Soc Trans 2015; 43(2):168-71; PMID:25849912; http://dx.doi.org/10.1042/BST20140267

[14] Kobayashi K, Kondo M, Fukuda H, Nishimura M, Ohta H. Galactolipid synthesis in chloroplast inner envelope is essential for proper thylakoid biogenesis, photosynthesis, and embryogenesis. Proc Natl Acad Sci U S A 2007; 104(43):17216-21; PMID:17940034; http://dx.doi.org/10.1073/pnas.0704680104

[15] Yamaguchi S, Darwin AJ. Recent findings about the *Yersinia enterocolitica* phage shock protein response. J Microbiol 2012; 50(1):1-7; PMID:22367931; http://dx.doi.org/10.1007/s12275-012-1578-7

[16] Yamaguchi S, Reid DA, Rothenberg E, Darwin AJ. Changes in Psp protein binding partners, localization and behaviour upon activation of the *Yersinia enterocolitica* phage shock protein response. Mol Microbiol 2013; 87(3):656-71; PMID:23290031; http://dx.doi.org/10.1111/mmi.12122

[17] Flores-Kim J, Darwin AJ. Activity of a bacterial cell envelope stress response is controlled by the interaction of a protein binding domain with different partners. J Biol Chem 2015; 290(18):11417-30; PMID:25802329; http://dx.doi.org/10.1074/jbc.M114.614107

[18] Jovanovic G, Engl C, Mayhew AJ, Burrows PC, Buck M. Properties of the phage-shock-protein (Psp) regulatory complex that govern signal transduction and induction of the Psp response in *Escherichia coli*. Microbiology 2010; 156(Pt 10):2920-32; PMID:20595257; http://dx.doi.org/10.1099/mic.0.040055-0

[19] Mehner D, Osadnik H, Lünsdorf H, Brüser T. The Tat system for membrane translocation of folded proteins recruits the membrane-stabilizing Psp machinery in *Escherichia coli*. J Biol Chem 2012; 287(33):27834-42; PMID:22689583; http://dx.doi.org/10.1074/jbc.M112.374983

[20] Osadnik H, Schopfel M, Heidrich E, Mehner D, Lilie H, Parthier C, Risselada HJ, Grubmüller H, Stubbs MT, Brüser T. The PspF-binding domain PspA and the PspA.F complex - New insights into the coiled-coil dependent regulation of AAA+ proteins. Mol Microbiol 2015; 98:743-59; PMID:26235546; http://dx.doi.org/10.1111/mmi.13154

[21] Heidrich J, Thurotte A, Schneider D. Specific interaction of IM30/Vipp1 with cyanobacterial and chloroplast membranes results in membrane remodeling and eventually in membrane fusion. Biochim Biophys Acta 2016; In press; PMID:27693914; http://dx.doi.org/10.1016/j.bbamem.2016.09.025

[22] DeLisa MP, Lee P, Palmer T, Georgiou G. Phage shock protein PspA of *Escherichia coli* relieves saturation of protein export via the Tat pathway. J Bacteriol 2004; 186(2):366-73; PMID:14702305; http://dx.doi.org/10.1128/JB.186.2.366-373.2004

[23] Zhang L, Kato Y, Otters S, Vothknecht UC, Sakamoto W. Essential role of VIPP1 in chloroplast envelope maintenance in Arabidopsis. Plant Cell 2012; 24(9):3695-707; PMID:23001039; http://dx.doi.org/10.1105/tpc.112.103606

[24] Hennig R, Heidrich J, Saur M, Schmuser L, Roeters SJ, Hellmann N, Woutersen S, Bonn M, Weidner T, Markl J, et al. IM30 triggers membrane fusion in cyanobacteria and chloroplasts. Nat Commun 2015; 6:7018; PMID:25952141; http://dx.doi.org/10.1038/ncomms8018

[25] Jordan S, Hutchings MI, Mascher T. Cell envelope stress response in Gram-positive bacteria. FEMS Microbiol Rev 2008; 32(1):107-46; PMID:18173394; http://dx.doi.org/10.1111/j.1574-6976.2007.00091.x

[26] Jordan S, Junker A, Helmann JD, Mascher T. Regulation of LiaRS-dependent gene expression in *Bacillus subtilis*: identification of inhibitor proteins, regulator binding sites, and target genes of a conserved cell envelope stress-sensing two-component system. J Bacteriol 2006; 188

(14):5153-66; PMID:16816187; http://dx.doi.org/10.1128/JB.00310-06

[27] Schrecke K, Jordan S, Mascher T. Stoichiometry and perturbation studies of the LiaFSR system of *Bacillus subtilis*. Mol Microbiol 2013; 87(4):769-88; PMID:23279150; http://dx.doi.org/10.1111/mmi.12130

[28] Dominguez-Escobar J, Wolf D, Fritz G, Hofler C, Wedlich-Soldner R, Mascher T. Subcellular localization, interactions and dynamics of the phage-shock protein-like Lia response in *Bacillus subtilis*. Mol Microbiol 2014; 92 (4):716-32; PMID:24666271; http://dx.doi.org/10.1111/mmi.12586

[29] Brissette JL, Weiner L, Ripmaster TL, Model P. Characterization and sequence of the *Escherichia coli* stress-induced psp operon. J Mol Biol 1991; 220(1):35-48; PMID:1712397; http://dx.doi.org/10.1016/0022-2836(91)90379-K

[30] Lloyd LJ, Jones SE, Jovanovic G, Gyaneshwar P, Rolfe MD, Thompson A, Hinton JC, Buck M. Identification of a new member of the phage shock protein response in *Escherichia coli*, the phage shock protein G (PspG). J Biol Chem 2004; 279(53):55707-14; PMID:15485810; http://dx.doi.org/10.1074/jbc.M408994200

[31] Aseeva E, Ossenbuhl F, Eichacker LA, Wanner G, Soll J, Vothknecht UC. Complex formation of Vipp1 depends on its alpha-helical PspA-like domain. J Biol Chem 2004; 279(34):35535-41; PMID:15210715; http://dx.doi.org/10.1074/jbc.M401750200

[32] Fuhrmann E, Gathmann S, Rupprecht E, Golecki J, Schneider D. Thylakoid membrane reduction affects the photosystem stoichiometry in the cyanobacterium *Synechocystis* sp. PCC 6803. Plant Physiol 2009; 149(2):735-44; http://dx.doi.org/10.1104/pp.108.132373

[33] Bryan SJ, Burroughs NJ, Shevela D, Yu J, Rupprecht E, Liu LN, Mastroianni G, Xue Q, Llorente-Garcia I, Leake MC, et al. Localisation and interactions of the Vipp1 protein in cyanobacteria. Mol Microbiol 2014; 94(5):1179-1195; PMID:25308470; http://dx.doi.org/10.1111/mmi.12826

[34] Deme B, Cataye C, Block MA, Marechal E, Jouhet J. Contribution of galactoglycerolipids to the 3-dimensional architecture of thylakoids. Faseb Journal 2014; 28:3373-83; PMID:24736411; http://dx.doi.org/10.1096/fj.13-247395

[35] Hincha DK, Oliver AE, Crowe JH. Lipid composition determines the effects of arbutin on the stability of membranes. Biophys J 1999; 77(4):2024-34; PMID:10512822; http://dx.doi.org/10.1016/S0006-3495(99)77043-8

[36] Shimojima M, Ohta H. Critical regulation of galactolipid synthesis controls membrane differentiation and remodeling in distinct plant organs and following environmental changes. Progress in Lipid Research 2011; 50(3):258-66; PMID:21414359; http://dx.doi.org/10.1016/j.plipres.2011.03.001

[37] Moellering ER, Benning C. Galactoglycerolipid metabolism under stress: a time for remodeling. Trends Plant Sci 2011; 16(2):98-107; PMID:21145779; http://dx.doi.org/10.1016/j.tplants.2010.11.004

[38] Shai Y. Mechanism of the binding, insertion and destabilization of phospholipid bilayer membranes by alpha-helical antimicrobial and cell non-selective membrane-lytic peptides. Biochim Biophys Acta 1999; 1462(1–2):55-70; PMID:10590302; http://dx.doi.org/10.1016/S0005-2736(99)00200-X

[39] Ablan FD, Spaller BL, Abdo KI, Almeida PF. Charge Distribution Fine-Tunes the Translocation of alpha-Helical Amphipathic Peptides across Membranes. Biophys J 2016; 111(8):1738-49; PMID:27760360; http://dx.doi.org/10.1016/j.bpj.2016.08.047

[40] Strandberg E, Ulrich AS. AMPs and OMPs: Is the folding and bilayer insertion of beta-stranded outer membrane proteins governed by the same biophysical principles as for alpha-helical antimicrobial peptides? Biochim Biophys Acta 2015; 1848(9):1944-54; PMID:25726906; http://dx.doi.org/10.1016/j.bbamem.2015.02.019

[41] Drin G, Antonny B. Amphipathic helices and membrane curvature. FEBS Lett 2010; 584(9):1840-7; PMID:19837069; http://dx.doi.org/10.1016/j.febslet.2009.10.022

[42] Madsen KL, Bhatia VK, Gether U, Stamou D. BAR domains, amphipathic helices and membrane-anchored proteins use the same mechanism to sense membrane curvature. FEBS Lett 2010; 584(9):1848-55; PMID:20122931; http://dx.doi.org/10.1016/j.febslet.2010.01.053

[43] Torok Z, Crul T, Maresca B, Schutz GJ, Viana F, Dindia L, Piotto S, Brameshuber M, Balogh G, Peter M, et al. Plasma membranes as heat stress sensors: from lipid-controlled molecular switches to therapeutic applications. Biochim Biophys Acta 2014; 1838(6):1594-618; PMID:24374314; http://dx.doi.org/10.1016/j.bbamem.2013.12.015

[44] Kultz D. Molecular and evolutionary basis of the cellular stress response. Annu Rev Physiol 2005; 67:225-57; PMID:15709958; http://dx.doi.org/10.1146/annurev.physiol.67.040403.103635

[45] Latifi A, Ruiz M, Zhang CC. Oxidative stress in cyanobacteria. FEMS Microbiol Rev 2009; 33(2):258-78; PMID:18834454; http://dx.doi.org/10.1111/j.1574-6976.2008.00134.x

[46] Torres S, Pandey A, Castro GR. Organic solvent adaptation of Gram positive bacteria: applications and biotechnological potentials. Biotechnol Adv 2011; 29(4):442-52; PMID:21504787; http://dx.doi.org/10.1016/j.biotechadv.2011.04.002

[47] Haslbeck M, Franzmann T, Weinfurtner D, Buchner J. Some like it hot: the structure and function of small heat-shock proteins. Nat Struct Mol Biol 2005; 12(10):842-6; PMID:16205709; http://dx.doi.org/10.1038/nsmb993

[48] Haslbeck M, Buchner J. Chaperone function of sHsps. Prog Mol Subcell Biol 2002; 28:37-59; PMID:11908065; http://dx.doi.org/10.1007/978-3-642-56348-5_3

[49] Nitta K, Suzuki N, Honma D, Kaneko Y, Nakamoto H. Ultrastructural stability under high temperature or intensive light stress conferred by a small heat shock protein in cyanobacteria. FEBS Lett 2005; 579(5):1235-42; PMID:15710419; http://dx.doi.org/10.1016/j.febslet.2004.12.095

[50] Torok Z, Goloubinoff P, Horvath I, Tsvetkova NM, Glatz A, Balogh G, Varvasovszki V, Los DA, Vierling E, Crowe JH, et al. *Synechocystis* HSP17 is an amphitropic protein that stabilizes heat-stressed membranes and binds denatured proteins for subsequent chaperone-mediated refolding. Proc Natl Acad Sci U S A 2001; 98(6):3098-103; PMID:11248038; http://dx.doi.org/10.1073/pnas.051619498

[51] Maitre M, Weidmann S, Rieu A, Fenel D, Schoehn G, Ebel C, Coves J, Guzzo J. The oligomer plasticity of the small heat-shock protein Lo18 from *Oenococcus oeni* influences its role in both membrane stabilization and protein protection. Biochem J 2012; 444(1):97-104; PMID:22360742; http://dx.doi.org/10.1042/BJ20120066

[52] Maitre M, Weidmann S, Dubois-Brissonnet F, David V, Coves J, Guzzo J. Adaptation of the wine bacterium *Oenococcus oeni* to ethanol stress: role of the small heat shock protein Lo18 in membrane integrity. Appl Environ Microbiol 2014; 80(10):2973-80; PMID:24584255; http://dx.doi.org/10.1128/AEM.04178-13

[53] Weidmann S, Maitre M, Laurent J, Coucheney F, Rieu A, Guzzo J. Production of the small heat shock protein Lo18 from *Oenococcus oeni* in *Lactococcus lactis* improves its stress tolerance. Int J Food Microbiol 2016; In press; PMID:27318622; http://dx.doi.org/10.1016/j.ijfoodmicro.2016.06.005

[54] Ezemaduka AN, Yu J, Shi X, Zhang K, Yin CC, Fu X, Chang Z. A small heat shock protein enables *Escherichia coli* to grow at a lethal temperature of 50°C conceivably by maintaining cell envelope integrity. J Bacteriol 2014; 196(11):2004-11; PMID:24659772; http://dx.doi.org/10.1128/JB.01473-14

[55] Szalontai B, Nishiyama Y, Gombos Z, Murata N. Membrane dynamics as seen by fourier transform infrared spectroscopy in a cyanobacterium, *Synechocystis* PCC 6803. The effects of lipid unsaturation and the protein-to-lipid ratio. Biochim Biophys Acta 2000; 1509(1–2):409-19; PMID:11118550; http://dx.doi.org/10.1016/S0005-2736(00)00323-0

[56] Kirchhoff H, Mukherjee U, Galla HJ. Molecular architecture of the thylakoid membrane: lipid diffusion space for plastoquinone. Biochemistry 2002; 41(15):4872-82; PMID:11939782; http://dx.doi.org/10.1021/bi011650y

[57] Kana R. Mobility of photosynthetic proteins. Photosynthesis Research 2013; 116(2–3):465-79; PMID:23955784; http://dx.doi.org/10.1007/s11120-013-9898-y

[58] Pribil M, Labs M, Leister D. Structure and dynamics of thylakoids in land plants. J Exp Bot 2014; 65(8):1955-72; PMID:24622954; http://dx.doi.org/10.1093/jxb/eru090