spacer- and RPEL-actins, which bind weakly in solution. Cytoplasmic MRTF-A localization in resting fibroblasts requires spacer-actin binding. The bipartite importin {alpha-beta} binding site is buried in the pentameric assembly, explaining how elevated G-actin concentrations prevent MRTF-A nuclear import. The structures show how actin loads onto the RPEL domain, and reveal a molecular mechanism by which actin can control the activity of one of its ligands.

[1] S. Mouilleron, S. Guettler, C.A. Langer, R. Treisman, N.Q. McDonald, *EMBO J* **2008,** *27,* 3198.

Keywords: actin, structure, MRTF-A

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The Molybdenum Storage Protein – a special kind of metallo-protein

Juliane Poppe,[a] Ron Huelskoetter,[a,b] Bjoern Kowalewski,[b] Klaus Schneider[b] and Ulrich Ermler,[a] [a]*Dept. of Molecular Membrane Biology, Max-Planck-Institute of Biophysics, Frankfurt/Main (Germany).* [b]*Faculty of Chemistry, University of Bielefeld, Bielefeld (Germany).* E-mail: Juliane.Poppe@mpibp-frankfurt.mpg.de

The diazotrophic soil bacterium *Azotobacter vinelandii* utilizes a FeMo-cofactor containing nitrogenase to accomplish nitrogen fixation. As nitrogenase is required in large amounts, the cell extracts a lot of molybdenum (Mo) from the environment and stores it in a special Molybdenum Storage Protein (MoSto)[1] as polyoxometalate clusters[2]. MoSto crystallizes in a needle-like shape. Best crystals diffract to 1.6Å resolution. An X-ray crystal analysis of the Mo-loaded MoSto revealed different types of Mo-oxide based clusters some being covalently bound while others are not. These clusters are synthesized in an ATP-dependent process whose mechanism is not yet known. *In-vitro* experiments showed that it is possible to entirely deplete MoSto from its metal clusters and subsequently reload it again. The depletion is a pH-driven triphasic process which can be varied with temperature and time of incubation [1]. MoSto can load up to 120 atoms per protein molecule. Further research is necessary to determine the mechanism how the clusters are built from monomolybdates and how their selective release from MoSto is organized.

[1] J. Schemberg *et al*., *ChemBioChem* **2008**, *9,* 595-602. [2] D. Fenske *et al*., *ChemBioChem* **2005**, *6,* 405-413

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Features critical for membrane binding revealed by DivIVA crystal structure

Maria A. Oliva,[a,b] Sven Halbedel,[c] Stefan Freund,[a] Thomas Leonardl,[a,d] Dimitri Veprintsev,[a] Leendert Hamoen,[c] Jan Löwe,[a] [a]*Structural Studies Division, Medical Research Council-Laboratory of Molecular Biology, Cambridge (UK).* [b]*Departamento Físico-Química, Centro de Investigaciones Biológicas, CSIC-Madrid (Spain).* [c]*Center for Bacterial Cell Biology, Institute for Cell and Molecular Biosciences. Newcastle University (UK).* [d]*Laboratory of Molecular Biology, NIDDK. National Institutes of Health, 9000 Rockville Pike, Bethesda (USA).* E-mail: marian@cib.csic.es

DivIVA is a highly conserved protein in Gram-positive bacteria that localizes at the poles and division sites, presumably through direct sensing of membrane curvature. The correct localization of the protein is essential for its biological function, which involve both the correct placement of the septum at midcell and also the segregation of the DNA into the pre-spore during sporulation. DivIVA deletion causes filamentous growth in *Bacillus subtilis*, whereas overexpression causes hyphal branching in *Streptomyces coelicolor*. The mechanism of subcellular DivIVA targeting and function remain unknown. We present data that demonstrates that DivIVA binds directly to bacteria membrane and explains how this protein shows a multifunctional behavior.

We prove that DivIVA is a two independent domains protein linked by a flexible tag and both domains together are essential to fulfill protein localization and function. The crystal structure determination shows that both domains share a parallel coiled-coil folding. Surprisingly the N-terminal domain also includes crossed and intertwined loops between the two coiled-coil molecules, which is completely new and is involved in protein targeting in combination with the presence of a key hydrophobic residue surrounded by positively charged residues. To clarify N-terminal domain function in DivIVA membrane targeting we have used an *in vivo* and *in vitro* approach, which shows Phe17 and its chemical context in DivIVA structure, is essential for membrane binding. We propose that the hydrophobic residues insert into the membrane and that the positively charged residues bind to the membrane surface. Also low-resolution crystal structure of the C-terminal domain displays a curved tetramer made from two parallel coiled-coils, which is the lowest oligomerization state of DivIVA showed by AUC. The N-terminal and C-terminal parts have been merged into a model of the full length, 30nm long DivIVA protein. Our experiments suggest that full-length DivIVA is an elongated tetramer with membrane targeting residues at both ends, which increase the protein ability to bind to membranes and other proteins.

Keywords: bacterial cell division, peripheral membrane protein, membrane curvature

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Structural basis for complement Factor I control and its disease associated sequence polymorphisms

Pietro Roversi,[a] Steven Johnson,[a] Joseph J.E. Caesar,[a] Florence McLean,[a] Kirstin J. Leath,[a] Stefanos A. Tsiftsoglou,[b] B. Paul Morgan,[c] Claire L. Harris,[c] Robert B. Sim,[b] and Susan M. Lea,[a] [a]*Sir William Dunn School of Pathology, University of Oxford, South Parks Road, Oxford OX1 3RE, England, (UK);* [b]*Department of Biochemistry, University of Oxford, South Parks Road, Oxford OX1 3QU, England, (UK);* [c]*Department of Infection, Immunity and Biochemistry, School of Medicine, Cardiff University, Heath Park, Cardiff CF14 4XN,Wales, (UK).* E-mail: Pietro.roversi@path.ox.ac.uk

The complement system is a key component of innate immune defence, coordinating inflammation with innate and adaptive responses, both in infection and in homeostasis. Complement regulation is critical for prevention and control of disease.

We have determined the crystal structure of the complement regulatory enzyme human Factor I (fI). Factor I is seen to be in a proteolytically inactive form, demonstrating that it circulates in a zymogen-like state, despite being fully processed to the mature sequence.

Mapping of functional data from mutants of fI onto the structure suggests that this inactive form is maintained by the non-catalytic heavy chain allosterically modulating activity of the light chain.

Once the ternary complex of fI, a cofactor and a substrate is formed,