s8a.m10.p1 **Structure of the complex between Rac1 and its inhibitor partner RhoGDI at 2.7 Å resolution.** S. Grizot[a], J. Fauré[b], F. Fieschi[a], P. Vignais[b], M.-C. Dagher[b] and E. Pebay-Peyroula[a]. [a]*Institut de Biologie Structurale J.P. Ebel CEA/CNRS/UJF 41, rue Jules Horowitz, 38027 Grenoble Cédex 01, France.* [b]*Laboratoire BBSI, CEA/CNRS/UJF, UMR 5092, Département de Biologie Moléculaire et Structurale, CEA-Grenoble, 17 rue des Martyrs, 38054 Grenoble Cédex 09, France.*
Keywords: G protein, GTP/GDP.

The small G protein Rac is involved in a variety of essential cellular processes. This molecular switch can cycle between a GTP-bound "on" state and a GDP-bound "off" state. This cycle is tightly regulated by proteins that can activate the GDP-GTP exchange reaction or stimulate the hydrolysis of the GTP. The Rho-related GTP-binding proteins present an additional level of regulation : the RhoGDI protein. The complex Rac/RhoGDI prevents the dissociation of the GDP from the small G protein and maintains Rac in the cytosol by shielding its isoprenylated C-terminus. (1)

The proteins were obtained by coinfecting Sf9 cells with recombinant baculoviruses. After purification of the heterodimer, crystals of the complex were grown in hanging drops by mixing equal volumes of protein solution (8-10 mg ml^{-1}) and reservoir solution (30% PEG 4000, 100 mM NaCitrate pH 5.6, 5 mM $MgCl_2$ and 200 mM Ammonium Acetate). Crystals belong to the orthorhombic space group $P2_12_12$ with unit cell dimensions a = 154.7 Å, b = 88.7 Å, c = 62.6 Å and contain two complexes in the asymmetric unit. A data set was collected at 2.7 Å resolution on beam line ID14-EH4 (ESRF, Grenoble). The structure was solved by molecular replacement using the program AmoRe. The position of Rac was unambiguously found but not RhoGDI. RhoGDI was then located by molecular replacement with a similar complex structure recently published (2). The current model was refined to an R factor of 25.5% and an R*free* of 28%.

The stucture reveals the essential regions for the complexation. The N-terminus (amino acids 30 to 60) of RhoGDI interacts with the switch regions of Rac whereas the geranylgeranyl arm of Rac plunges into a hydrophobic pocket in the immunoglobulin-like domain of RhoGDI.

[1] Fauré J. et al. (1999) *Eur. J. Biochem.* 262, 879-889.
[2] Longenecker K. et al. (1999) *Acta Cryst*. D55, 1503-1515.

s8a.m10.p2 **A Redox-Dependent Interaction Between Two Electron-Transfer Partners Intervening In Photosynthesis**. R. Morales, G. Kachalova, M-H. Charon, M. Frey *IBS/LCCP 41 Rue Jules Horowitz, 38027 Grenoble France ; frey@lccp.ibs.fr*
Keywords: photosynthesis, protein-protein interactions, redox-linked changes.

Ferredoxin:$NADP^+$:reductase (FNR) plays a major role in the conversion of light energy into chemical energy during photosynthesis by catalyzing the reduction of $NADP^+$ into NADPH. The reducing power of NADPH is essential for carbon assimilation. For the reaction, FNR uses two high energy electrons photoinduced by the photosystem I and conveyed, one at a time by a ferredoxin (Fd). Each electron transfer from Fd to FNR requires the formation of a transient complex between the reduced Fd and a preformed FNR/$NADP^+$ complex[1]

Determination of the X-ray structures of the cyanobacterium *Anabaena* ferredoxin in its reduced (1.18 Å) and oxidized (1.3 Å) state[2] has shown that Fd undergoes a redox-linked conformational change in the vicinity of its [2Fe-2S] cluster : the peptide bond linking Cys46 and Ser47 points its carbonyl oxygen away from one of the cluster sulfur atoms (S2) in the reduced molecule and toward it in the oxidized one. In any redox state this 46-47 peptide is in close contact with the Phe65 aromatic ring which is itself exposed to the solvent.

In parallel, the interaction between the oxidized FNR and Fd, from the same cyanobacterium, was studied by crystallography, at 2.4 Å resolution, leading to *the first three-dimensional picture of a Fd/FNR biologically relevant complex*. This complex shows that Fd and FNR can specifically interact with each other in accordance with a wealth of biological evidences[1]. In this crystallographic complex the, above mentioned, Fd Phe65 aromatic ring is buried and in close contact with FNR Leu76, Leu78 and Val136 at the core of the molecular interaction. This suggests that the role of the redox-linked 46-47 peptide "flip" which is induced by the transfer of one electron from Fd to FNR, is to displace the Fd Phe65 aromatic side-chain, thus triggering the dissociation of the ferredoxin from the transient complex.

[1] Arakaki *et al.* "Plant-type ferredoxin-$NADP^+$ reductases: a basal structural framework and a multiplicity of functions", Faseb J. (1997) 11, 133-40.
[2] Morales *et al.* "Refined X-ray Structures of the Oxidized, at 1.3 Å, and Reduced, at 1.17 Å, [2Fe-2S] Ferredoxin from the Cyanobacterium *Anabaena* PCC7119 Show Redox-Linked Conformational Changes.", Biochemistry (1999) 38: 15764-15773.