the conserved regions suggests that the folding of CHM and GDI may be similar. Site-directed mutagenesis of a region defined by two of the SCRs critical role for this region in the binding of Rab proteins. The crystal structure of GDI has been determined using MIRAS method and refined to crystallographic R-value of 19.8% and R-free 27.5% for a total of 450 residues of the molecule in the asymmetric unit.

**MS04.07b.03** THE STRUCTURE OF EF-Tu:EF-Ts COMPLEX SUGGESTS A MECHANISM FOR NUCLEOTIDE RELEASE. Takemasa Kawashima, Carmen Berthet-Colominas, Michael Wulf, Stephen Cussack, and Reuben Leberman, EMBL Grenoble Outstation B.P. 156, 38042 Grenoble Cedex, France. *European Synchrotron Radiation Facility, B.P. 220, 38043 Grenoble Cedex, France.

The crystal structure of the bacterial elongation factor complex EF-Tu:EF-Ts from Escherichia coli has been determined at 2.5 Å resolution. It reveals a typical guanine nucleotide binding protein in interaction with its guanine nucleotide release factor. The complex is a tetramer where two EF-Ts form a tight dimer and each EF-Tu binds essentially to one EF-Ts, with barely any contact between EF-Tu, such that the complex is best described by the formula EF-Tu(2):EF-Ts(2). Comparison of EF-Tu-GDP and EF-Tu-GTP shows that the conformational change between the two structures mainly alters the magnesium binding site, by disrupting the coordination of the ion. This suggests a molecular mechanism by which EF-Ts releases the GDP bound to EF-Tu where the affinity of EF-Tu for GDP is lowered by the loss of the magnesium ion.


**MS04.07b.04** GAMMA-FIBRINOGEN: CRYSTAL STRUCTURE OF A 30 KDA C-TERMINUS FRAGMENT AT 2.1 Å RESOLUTION. V.C. Yee, K.P. Pratt, H.C. Cote, L. Le Trong, D.W. Chang, R.E. Stenkamp, and D.C. Teller, Departments of Biochemistry and Biological Structure, University of Washington, Seattle, WA, 98195.

The crystal structure of a 30 kDa carboxy terminal fragment of the fibrinogen gamma chain has been determined using MIR phases to 2.5 Å resolution, and refined against diffraction data to 2.1 Å resolution. Fibrinogen is the central structural protein in the blood coagulation process. Cleavage of fibrinogen by the serine protease thrombin yields fibrin, which spontaneously polymerizes to form a clot matrix. Subsequent covalent crosslinking by factor XIIIa, a transglutaminase, produces a mechanically and proteolytically stable blood clot.

Fibrinogen is a disulfide-crosslinked dimer of heterotrimers, and contains two copies each of the alpha, beta, and gamma chains. A recombinant 30 kDa C-termminus fragment of the fibrinogen gamma chain containing the principle factor XIIIa crosslinking site, the calcium-binding site, the platelet receptor recognition domain, part of the polymerization surface, and one of the sites which activating t-PA activation of plasminogen, has been the focus of our crystallographic studies. This fragment crystallizes in space group P212121 with 4 molecules in the asymmetric unit. The crystal structure was solved to 2.43 Å resolution. The crystal asymmetric unit contains two half-tetramers, each with 222 symmetry. Each subunit essentially consists of two Greek keys and one Greek key-like motif, arranged in a threefold symmetric fashion. The -chain forms part of the Greek key-like motif and plays an important role in subunit association. Unlike the case of villin membrane outer layer protein-1, the threefold symmetry is not reflected in the sequence. Furthermore, the three Greek keys in the former are connected by disulfide bridges while in Jacalin the threefold symmetric structure is stabilized entirely by non-covalent interactions. The structure of Jacalin presents the first observation of a -prism fold in a lectin. Two of the loops at one end of the prism constitute the carbohydrate binding site which shows novel features including the involvement of a N-terminal amino group generated by post-translational modification.

**MS04.07b.05** STRUCTURAL BASIS OF CALCIUM INDUCED E-CADHERIN RIGIDIFICATION AND DIMERIZATION. Bhushan Nagar, Michael Overduin, Mitsuhiko Ikura, James M. Rini. "Departments of Molecular and Medical Genetics and Biochemistry, University of Toronto, Toronto, Ontario, MSS IA8, Canada. "Division of Molecular and Structural Biology, Ontario Cancer Institute and Department of Medical Biophysics, University of Toronto, 610 University Avenue, Toronto, Ontario, M5G 2M9, Canada.

The cadherins mediate cell adhesion and play a fundamental role in normal development. They typically consist of five tandemly repeated extracellular domains, a single membrane spanning segment, and a cytoplasmic region. The N-terminal extracellular domains mediate cell-cell contact while the cytoplasmic region interacts with the cytoskeleton through the catenins. Cadherins are defined by their calcium dependence: removal of calcium abolishes adhesive activity and renders cadherins vulnerable to proteases. The two N-terminal domains of epithelial cadherin (E-cadherin) have been expressed in E. coli and crystallized from 11mg/ml solutions by the hanging-drop vapour-diffusion method in the presence of CaCl2. The well solution consisted of 1.2M Ammonium sulfate, 30 mM CaCl2, 100 mM Tris buffer pH 9.0. Plate-like crystal clusters of dimensions 0.6 x 0.4 x 0.2 mm grow within 2 days in space group C2 (a=122.0, b=80.5, c=73.2 Å, ß=114.5°. The structure was solved (to 2.0 Å resolution) using a combination of multilength anomalous diffraction (MAD) phasing and real space averaging techniques. The structure reveals a two-fold symmetric dimer, each molecule of which binds a contiguous array of three bridged calcium ions. Each molecule of the dimer is composed of two 7-stranded 8-barrels connected by a 10 residue linker. Not only do the bound calcium ions linearize and rigidify the molecule, but they promote dimerization. The E-cadherin dual domain structure defines the role played by calcium in the cadherin mediated formation and maintenance of solid tissues.