Microsymposia

Two main factors are responsible for the electronic transport in icosahedral Al-Pd-Re. The first is the dimensionless intergranular conductance $g$. Samples with $g \geq 1$ exhibit metallic properties. Samples with $g \leq 1$ show an insulating behavior. Depending on the structural state of the material and the temperature, both factors may be important. The notion of granular system means $g \leq g_c$, where $g_c$ is the dimensionless intragranular conductance. The homogeneously disordered case corresponds to $g = g_c$. The second factor is the electrostatic (Coulomb) energy $E_C > \delta$, which accounts for the change in the energy of a grain when an electron is added or removed. It plays an important role in the insulating regime when electrons are localized in the grains. At low coupling $g \ll 1$ an electron has to overcome an electrostatic barrier into a neighboring granule. Depending on the structural state and temperature the both factors may be important for the specific behavior of the electrical conductivity. The character of electrostatic disorder defines either the Efros–Shklovskii or the Mott type of the VRH regime primarily with elastic co-tunneling at $T \leq (\delta E_c)^{1/2}$. For icosahedral Al-Pd-Re this temperature is less than 10 K. For strong intergranular coupling, the metallic regime with a power-law temperature variation of the electrical conductivity $\sigma$ takes place. At low temperatures the temperature-dependent part of $\sigma$ is dominated by quantum-interference corrections.

The metallurgical reason for the specific behavior of polygrain icosahedral Al-Pd-Re most likely relates to the fact that the icosahedral phase is the primary crystallization phase only for alloys with rhenium content lower that 5 at.%. For icosahedral phase with 8.5 at.% rhenium the primary crystallization phase is Al$_5$Re that crystallizes in the shape of needles leading to porous ingots with numerous voids.


Keywords: electronic, icosahedral, quasicrystal

---

### MS.63.5


**Variable-Range-Hopping Conductivity in Quasicrystals**

Yu. Kh. Vekilov, M. A. Chernikov, Department of Theoretical Physics and Quantum Technologies, National University of Science and Technology “MISIS”, Moscow, (Russia). E-mail: yuri.vekilov@gmail.com

Among icosahedral phases, including single crystals of icosahedral Al-Pd-Re, the variable-range-hopping (VRH) regime is only revealed in polycrystal samples of icosahedral Al-Pd-Re with the resistivity ratio $R = \rho(4.2\text{K})/\rho(300\text{K}) \geq 13$. Experiments show that this regime relates to the presence of defects, voids and inclusions of secondary phases [1, 2]. Here we analyze the low temperature electronic transport in polycrystal samples of icosahedral Al-Pd-Re using an analogy with granular electronic conductors. Our preliminary results are given in Ref. 3.

The density of electronic states at the Fermi energy $N(E_F)$ in icosahedral quasicrystals is nonzero. We therefore consider a model in which metallic quasicrystalline particles of a size ranging from a few nanometers to hundreds of nanometers are embedded into an insulating matrix. The electronic levels are discrete due to electron confinement within a single “grain”. The mean level spacing $\delta$ is inversely proportional to the volume $V$ of the grain $\delta \approx 1/(V[N(E_F)V])$. For icosahedral Al-Pd-Re $\delta$ is an order of magnitude higher that for a typical metal.

---

### MS.64.1


**Structural basis for the sequential assembly of photosynthetic multiienzyme complex**

Hiroyoshi Matsumura,¹ Akihiro Kai,¹ Takuyuki Maeda, Masahiro Tamoi,² Mika Hirose,² Natsuko Kizu,² Akira Wadano,¹ Tsuyoshi Inoue,¹ Shigeru Shigeoka,² *Graduate School of Engineering, Osaka University, Osaka (Japan). Faculty of Agriculture, Kinki University, Nara (Japan). Kagoromo International University, Osaka (Japan).* E-mail: matsumura@chem.eng.osaka-u.ac.jp

Protein–protein interactions are likely to be generally important in coordinated regulation of metabolic pathways. In photosynthetic CO$_2$ assimilation pathway (the Calvin cycle), such regulation is partially achieved by an intrinsically disordered protein, CP12, which acts as a linker in the sequential assembly of Calvin cycle enzymes glyceraldehyde-3-phosphate dehydrogenase (GAPDH) and phosphoribulokinase (PRK). Both enzymes activities are inhibited when embedded within the GAPDH/CP12/PRK complex. The reversible association/disociation of the complex are mediated by light/dark transitions, which are responsible for the changes in redox potential and the concentrations of metabolites such as NAD(H) and NADP(H). However, the molecular mechanisms for the sequential multiprotein assembly still remain elusive.

Here we report the crystal structures of GAPDH-CP12 complex and PRK from *Synechococcus elongatus* (Fig. 1). The structure of GAPDH-CP12 complex represents that the C-terminal region of CP12
Microsymposia

binds to a groove of tetrameric GAPDH, while N-terminal region of CP12 is largely unstructured. We also observed that the dimeric PRK adopts distinct conformations generated by a rigid body movement of the protomer, suggesting that PRK exists in equilibrium between distinct conformations in solution. Together with mutagenesis analysis, these data demonstrated that CP12 is partially folded on the binding of the first target GAPDH, and the folded part of CP12 within GAPDH-CP12 complex plays a crucial role in selective interaction with a specific conformer from possible conformational states of the second target PRK to complete the GAPDH-CP12-PRK complex. These studies give further understanding how the complex formation down-regulates GAPDH and PRK to synchronize to the light transition, and also sheds light on the question of how the GAPDH-CP12-PRK complex is formed.

Fig. 1 Crystal structures of GAPDH-CP12(left) and PRK (right).

Keywords: assembly, photosynthesis, enzyme

MS.64.2

Structural basis for specific aminoacyl-tRNA synthesis
Shigeyuki Yokoyama, RIKEN Systems and Structural Biology Center, Yokohama, Graduate School of Science, The University of Tokyo, Tokyo (Japan). E-mail: yokoyama@biochem.s.u-tokyo.ac.jp

In protein biosynthesis, each codon is translated into its specific amino acid by aminoacyl-tRNA. For most of the amino acids used in translation, their cognate aminoacyl-tRNA synthetases (aaRSs) synthesize aminoacyl-adenylate from the amino acid and ATP, and then transfer the aminoacyl moiety to the 3-terminal adenosine of the cognate tRNA, depending on strict recognition of both amino acid and tRNA. In most cases, the anticodon of tRNA serves as the major determinant for the recognition by the corresponding aaRS. However, several aaRSs recognize other part of tRNA for specific aminoacylation. Alanyl-tRNA synthetase (AlaRS) and histidyl-tRNA synthetase (HisRS) recognize the G3:U70 wobble base pair and the –1 guanosine, respectively. We have determined crystal structures of AlaRS and HisRS in complex with their cognate alanine and histidine tRNAs, respectively, which reveal the unique mechanisms of tRNA recognition. On the other hand, for some amino acids, the aminoacyl-tRNA is not synthesized in the canonical, direct manner, but indirectly synthesized by conversion of the aminoacyl moiety of another aminoacyl-tRNA. For example, glutaminyl-tRNA is synthesized from glutamyl-tRNA by “transamidosome” in most of microbes. The selenocysteine, the twenty-first amino acid in protein synthesis, is synthesized from the serylated form of its specific tRNA. We will present structural basis of specific tRNA recognition in these indirect aminoacyl-tRNA synthesis mechanisms, on the basis of our crystal structures of the machineries in the tRNA-bound form.

Keywords: tRNA, enzyme, complex

MS.64.3

First view of insulin bound to its primary binding site on the insulin receptor
Michael Lawrence,1,* John Menting,2* Geoffrey Kong,2* Mai Margetts,2 Colin Ward,1 *The Walter and Eliza Hall Institute of Medical Research and 2The Department of Medical Biology, University of Melbourne, Parkville, Victoria, (Australia). Email: lawrence@wehi.edu.au

While the three-dimensional structure of insulin was an early triumph of protein crystallography [1], to date there has been no three-dimensional structural information regarding the manner in which the insulin hormone binds to the insulin receptor (IR). Therapeutic targeting of IR is of key importance in the treatment of both Type 1 and Type 2 diabetes and structural information regarding the mode of insulin binding to IR is of particular relevance to the design of novel insulins with enhanced therapeutic profiles [2]. IR is also closely related the Type 1 insulin-like growth factor receptor (IGF-1R), which is under intensive investigation as an anti-cancer target [3]. The mode of ligand binding to these receptors is likely highly similar, but, again, little is known about the mode of IGFs binding to IGF-1R.

IR is a (α ββγ) disulfide-linked homodimer [4]. Each receptor monomer consists of a disulphide-linked α-chain and β-chain, and disulfides cross-link the monomers at two sites within the respective α-chains. Cross-linking, biochemical and mutagenesis studies have revealed that the initial hormone-receptor interaction is insulin binding to the so-called “Site 1” on IR. Site 1 consists of elements of first leucine-rich repeat domain (L1) of one receptor monomer and the C-terminal segment (α CT) of the α-chain of the second monomer [5]. Following this event, insulin then forms a crosslink to “Site 2”, which lies at the junction of the first and second fibronectin domains of the second monomer. The resulting complex is of picomolar affinity and effects signal transduction.

After twenty years of effort, we have now obtained a crystal structure of insulin in complex with Site 1, with the IR elements being contributed by a domain-minimized dimeric receptor construct. Crystal formation was achieved by attaching a pair of Fab molecules to the receptor / hormone assembly. The structure reveals that the B-helix of insulin lies parallel to the α CT helix on the surface of the central β-sheet of L1. The α CT helix is repositioned with respect to its location in the apo-IR structure and our structure indicates that it effects the displacement of the extended C-terminal segment of the insulin B-chain away from its location next to the B-helix [6]. Our structure indicates further that elements of the hexamer-forming surface of insulin would be directed towards Site 2 within an intact IR dimer, suggesting a mechanism for signal transduction.

Keywords: insulin, insulin receptor, protein-protein interactions