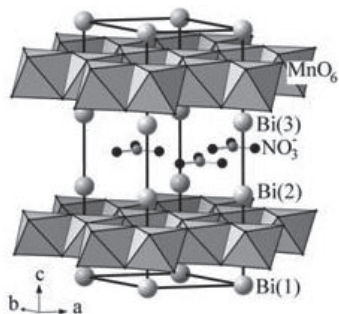


**P02.15.45***Acta Cryst.* (2008). A64, C214**Multitechnique solution of a new structural type  
Bi<sub>3</sub>Mn<sub>4</sub>O<sub>11</sub>.22(NO<sub>3</sub>)<sub>0.93</sub>**Olga Smirnova<sup>1</sup>, Azuma Masaki<sup>2</sup>, Nobuhiro Kumada<sup>1</sup>, Y Kusano<sup>3</sup>, Takahiro Takei<sup>1</sup>, Yoshinori Yonesaki<sup>1</sup>, Nobukazu Kinomura<sup>1</sup><sup>1</sup>University of Yamanashi, Centre for Crystal Science and Technology, 7 Miyamae, Kofu, Yamanashi, 400-8511, Japan, <sup>2</sup>Institute for Chemical Research, Kyoto University, <sup>3</sup>Kurashiki University of Science and Arts, E-mail: smirnova@yamanashi.ac.jp

The black fine powder of the new compound was obtained by hydrothermal reaction; indexing of the electronic diffraction patterns provided the hexagonal cell. A combination of direct method of structure solution from powder (EXPO2004) and direct-space method (FOX) using only synchrotron X-ray data allowed to locate heavy atoms in the structure, but provided very diffuse information about the light atoms. Further structure solution and Rietveld refinement (FullProf) using neutron data led to the non-centrosymmetric unit cell described in a space group *P3* ( $a = 4.9679(3)$  Å,  $c = 13.161(1)$  Å) with one NO<sub>3</sub><sup>-</sup> group per cell, significant distortion of Mn octahedra, and also random oxygen vacancies around Bi(1) atoms (Figure). Bi(1) has either five ligands or appears in a distorted octahedra; while Bi(2) and Bi(3) atoms have umbrella-like environment built of three oxygen ligands. The absence of the inversion centre was confirmed by the second harmonic generation measurement; the presence of NO<sub>3</sub><sup>-</sup> groups in the structure was confirmed by FT-IR measurements and TG Mass spectroscopy.



Keywords: electronic diffraction, X-ray diffraction, neutron diffraction

**P02.15.46***Acta Cryst.* (2008). A64, C214**The interaction between human rhinovirus 3C protease and stem loop D studied by solution scattering**John Y H Chow<sup>1</sup>, Jolyon K Claridge<sup>2</sup>, Cy Jeffries<sup>1</sup>, Stephen J Headley<sup>2</sup>, Giselle A Soares<sup>2</sup>, Martin Schwalbe<sup>2</sup>, Steven M Pascal<sup>2</sup>, Jill Trehwella<sup>1</sup><sup>1</sup>University of Sydney, School of Molecular and Microbial Biosciences, Room 676, School of Molecular and Microbial Biosciences, Building G08, University of Sydney, New South Wales, 2006, Australia, <sup>2</sup>Institute of Fundamental Sciences, Massey University, Palmerston North, New Zealand, E-mail: j.chow@mmb.usyd.edu.au

We present the results of recent small angle X-ray scattering and neutron contrast variation experiments that are part of a large effort to determine the structure of the 3C protease (3Cpro) and stem loop D (SLD) complex from human rhinovirus 14 (HRV-14). Human rhinoviruses are members of the picornaviridae family of small RNA viruses. Like all RNA viruses, picornaviridae achieve gene expression by inducing the host cell to produce a single polypeptide that is post-translationally cleaved to produce active viral proteins. In HRV-14 the role of cleaving viral proteins from the host-expressed polypeptide is performed by 3Cpro, which also acts as a

crucial component of the HRV-14 ribonucleoprotein complex that initiates transcription of the viral genome during replication. The activity of this complex is dependent on the binding of 3Cpro to the cloverleaf structure known as SLD near the start of the 5' region of the genome. While the structure of the 3Cpro-SLD complex is unknown, individual structures of 3Cpro and SLD have been solved by X-ray diffraction and NMR. We have studied a 1:1 3Cpro-SLD complex using small-angle scattering and determined the ensemble average structure of the complex in solution. Comparisons between the structure of the complex and existing high resolution structures of the individual components will provide insights into conformational changes during the formation of the complex and assist in structure-based drug design.

Keywords: neutron contrast variation, SAXS, RNA-protein complexes

**P02.15.47***Acta Cryst.* (2008). A64, C214**Inhibition of histidine kinase A in *Bacillus subtilis*: A neutron contrast variation study**David A Jacques<sup>1</sup>, David B Langley<sup>1</sup>, Andrew E Whitten<sup>2</sup>, Cy M Jeffries<sup>1</sup>, Jill Trehwella<sup>1</sup><sup>1</sup>University of Sydney, School of Molecular and Microbial Biosciences, Building G08 Butlin Avenue, University of Sydney, NSW, 2006, Australia, <sup>2</sup>Australian Nuclear Science and Technology Organisation, Lucas Heights, NSW, Australia, E-mail: djac4793@mail.usyd.edu.au

We have used small-angle scattering with neutron contrast variation to investigate the interaction between Histidine Kinase A (KinA) and two of its inhibitors: Sda and KipI. In *Bacillus subtilis*, KinA is responsible for initiating a phosphorelay that culminates in the expression of genes controlling spore formation. The organism expresses Sda if it fails to replicate its chromosome, thereby preventing spore formation. Two Sda molecules then bind KinA and inhibit the autophosphorylation reaction. We have shown that Sda binds the dimerisation domain of KinA, which undergoes a conformational change resulting in a compaction of the KinA structure. While KipI is a much larger protein than Sda, and is expressed under different conditions, two KipI molecules also interact with the dimerisation domain of KinA resulting in the same compaction. Our molecular models for the inhibitor complexes (based on the crystal structures of homologous proteins) coupled with bioinformatic analyses of these systems show that the compaction is due to a collapse of the catalytic domains towards the dimerisation domains, which prevents the catalytic domains from appropriately accessing the target histidine. Our results also indicate that the dimerisation domain (a four-helix bundle) operates as a conduit for transmitting an inhibitory signal down the length of the molecule, an observation that has implications for the histidine kinases that are widely used in bacterial signal transduction.

Keywords: neutron contrast variation, small-angle scattering, signal transduction

**P02.15.48***Acta Cryst.* (2008). A64, C214–215**The KipI-KipA complex and histidine kinase regulation in *Bacillus subtilis***David B Langley<sup>1</sup>, David A Jacques<sup>1</sup>, Andrew Whitten<sup>2</sup>, Jill Trehwella<sup>1</sup>