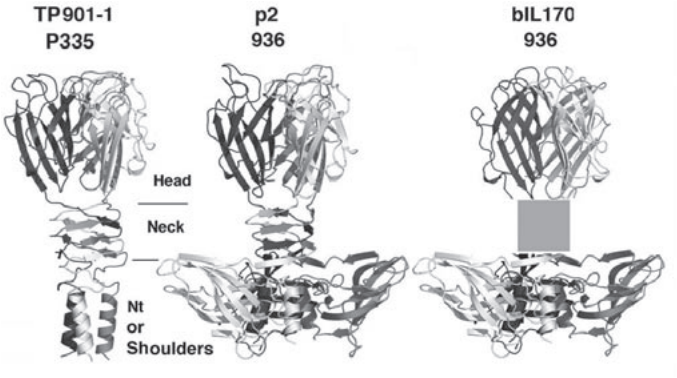
and those of adenoviruses or reoviruses, which invade mammalian cells, suggests that these viruses, despite evolutionary distant targets and the different chemical nature of their genomes (DNA vs RNA), might have a common ancestral gene. Results on RBPs of other lactococcal phages (bIL170 and TP901-1) will also be presented, which further illustrate the modular nature of these proteins in the viral world, and their insertion in the viral baseplate.



Keywords: receptor binding protein, bacteriophage, *Lactococcus lactis*

P04.13.308

Acta Cryst. (2008). A**64**, C327

Structure of the SARS coronavirus nucleocapsid protein RNA-binding dimerization domain

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Coronavirus nucleocapsid proteins are basic proteins that encapsulate viral genomic RNA to form part of the virus structure. The nucleocapsid protein of SARS-CoV is highly antigenic and associated with several host-cell interactions. Our previous studies using nuclear magnetic resonance revealed the domain organization of the SARS-CoV nucleocapsid protein. RNA has been shown to bind to the N-terminal domain (NTD), although recently the C-terminal half of the protein has also been implicated in RNA binding. Here, we report that the C-terminal domain (CTD), spanning residues 248－365 (NP248-365), had stronger nucleic-acid-binding activity than the NTD. To determine the molecular basis of this activity, we have also solved the crystal structure of the NP248-365 region. Residues 248－280 form a positively charged groove similar to that found in the infectious bronchitis virus (IBV) nucleocapsid protein. Furthermore, the positively charged surface area is larger in the SARS-CoV construct than in the IBV. Interactions between residues 248-280 and the rest of the molecule also stabilize the formation of an octamer in the asymmetric unit. Packing of the octamers in the crystal forms two parallel, basic helical grooves, which may be oligonucleotide attachment sites, and suggests a mechanism for helical RNA packaging in the virus.

Keywords: severe acute respiratory syndrome coronavirus, nucleocapsid, viral packaging

P04.13.309

Acta Cryst. (2008). A**64**, C327

Crystallization and structure determination of recombinant hepatitis E virus-like particle

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Hepatitis E virus (HEV) causes water-borne epidemics with a substantial mortality rate in pregnant women. It accounts for the major part of enterically transmitted hepatitis infection worldwide. The major structural protein is derived from open reading frame 2 of the viral genome, which forms the virus-like particles (VLPs) as expressed in insect cells. Single crystals of HEV-VLPs obtained by the hanging-drop vapor-diffusion methods at 293 K diffracted X-rays to 3.8 Å resolution, and the atomic structure of the HEV-VLP has been determined by phase extension from a low-resolution electron microscopy structure. The structure of the capsid protein of HEV has three domains, which exhibit similarities to the caliciviruses, agents of acute nonbacterial gastroenteritis.

Keywords: virus structure, virus coat proteins, viral structure and function

P04.13.310

Acta Cryst. (2008). A**64**, C327−328

The N-terminal induced-fit loops of capsid protein of Rice dwarf virus stabilize capsid of the virus

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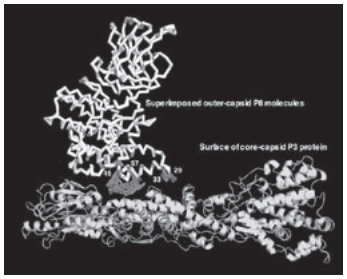
Induced-fit loops were observed in the N-terminal region of the P8 outer-capsid protein of Rice dwarf virus. In thirteen independent P8 molecules, each of the loops had a different conformation. These induced-fit loops appeared to stabilize and strengthen the binding between the respective P8 outer-capsid proteins and the P3 core-capsid protein, with the heterologous bindings, in the double-layered capsid, having disparate symmetry. Among five kinds of P8 trimer, all the P8 proteins in the P-trimers that were located at the five-fold axes exhibited some disorder in the N-terminal region. This observation, together with the total binding energy between the P-trimer and vertical P3 and horizontal P8 was lowest among the trimers, suggests that it would be this trimer that would be released when one of the outer-capsid trimers is shed from the core particle to mediate the biological activity of the virus. In figure, the thirteen



independent P8 molecules are superimposed on one of the P3 core-capsid protein. Cα trace of the P8 molecule is shown in a stick model. Start and end of the two induced-fit loops, shown in gray stick, are indicated in terms of residue number.

Keywords: virus coat proteins, virus assembly, viral structure and function

P04.13.311

Acta Cryst. (2008). A**64**, C328

The structure of baculovirus intracellular polyhedrin crystals reveals homoplasy of viral polyhedra

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Because insect viruses often remain in soil or leaves for prolonged periods before finding suitable hosts, they have evolved unique strategies to preserve their infectivity in such conditions. The most striking of these survival strategies are polyhedra, crystals of the viral polyhedrin protein which form a tough matrix protecting virus particles. Virus particles embedded in polyhedra can remain infectious for decades in the soil but, once ingested by new larvae, polyhedra readily dissolve in the alkaline environment of mid-guts initiating a new infectious cycle. Recently, the first atomic structure of polyhedra revealed the architecture of such infectious crystals produced by the silkworm cypovirus, a RNA virus belonging to the Reoviridae family. To understand how this strategy evolved in the viral world, we have engaged in the structural analysis of polyhedra produced by baculoviruses, DNA viruses completely unrelated to cypoviruses. I will present the 2.3Å structure of baculovirus polyhedra determined by X-ray crystallography from crystals 5-10 micrometers in diameter purified from infected cells. These crystals belong to the *I*23 space group with cell edge parameters of 103Å, just like cypovirus polyhedra. They are also made of polyhedrin trimers and extremely dense and robust except in alkaline conditions. Despite these functional and structural similarities, baculovirus and cypovirus polyhedrin proteins are unrelated and the way they pack in polyhedra is strikingly different. This evolutionary convergence to very similar crystalline architectures from different building blocks is reminiscent of the wide use of the icosahedral symmetry in virus particles.

Keywords: virus assembly, virus evolution, microcrystals

P04.13.312

Acta Cryst. (2008). A**64**, C328

Insight into viral inhibition of apoptosis - Structures of myxoma virus M11L and vaccinia virus F1L

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Programmed cell death (apoptosis) is a critically important mechanism that enables multicellular organisms to eliminate damaged, infected or unwanted cells. The Bcl-2 family of proteins, which contains both pro- and antiapoptotic members, plays a central role in regulating apoptosis. The two proapoptotic members Bax and Bak are activated in response to apoptotic stimuli and play a pivotal role by triggering the release of pro-death factors by a series of unknown conformational events that result in mitochondrial membrane permeabilization (MMP). In healthy cells, Bax and Bak are held in check by antiapoptotic family members such as Bcl-2, Bcl-xL and Mcl-1. Apoptotic stimuli result in the release of proapoptotic BH-3 only proteins that neutralize antiapoptotic Bcl-2, thus freeing Bak and Bax to cause MMP. Apoptosis is recognised as a key innate immunity defence mechanism, and viruses have developed different strategies to ensure their survival in the face of host immune responses. Viral Bcl-2 homologs are deployed by a number of viruses to prevent cells from apoptosis during infection. Myxoma virus (MV) and vaccinia virus (VV), which both belong to the poxviruses, encode numerous anti-apoptotic proteins, but lack obvious Bcl-2 homologues. The MV protein M11L and the VV protein F1L have been identified as major virulence factors that locate to the outer mitochondrial membrane, lack sequence similarity to any other protein and have been shown to inhibit apoptosis. We have determined the crystal structures of free M11L and M11L in complex with a Bak 26-mer peptide as well as the crystal structure of F1L, and investigated their antiapoptotic properties. Our analysis provides new insight into the mechanism by which MV and VV subvert host apoptosis to ensure virus survival.

Keywords: apoptosis, protein homology, viral protein

P04.13.313

Acta Cryst. (2008). A**64**, C328-329

Structure of influenza H5N1 nucleoprotein and its interaction with RNA

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Influenza is a contagious respiratory illness causing annual epidemics. The threat of a pandemic outbreak of influenza virus H5N1 has become a major concern worldwide. The nucleoprotein (NP) plays both structural and functional roles in influenza viruses and represents an attractive drug target. Here we report the 0.33nm crystal structure of H5N1 NP, which is composed of a head domain, a body domain and a tail loop. Our structure resolves the important linker residues (residues 397-401, 429-437) that connect the tail loop with the remainder of the molecule and a flexible, basic loop (residues 73-91) located in an arginine-rich groove surrounding Arg150. Using surface plasmon resonance, this basic loop and arginine-rich groove, but mostly a protruding element containing Arg174 and Arg175, were found to be important in RNA binding. A possible mechanism by which NP associates with RNA is as follow. First, the flexibility of the basic loop (residues 73-91) may allow it to sample the environment and capture RNA. The captured RNA could