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

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

Fasseli Coulibaly[1], Elaine Chiu[1], Keiko Ikeda[2], Sascha Gutmann[3], Peter W Haebel[4], Clemens Schulze-Briese[3], Hajime Mori[5], Peter Metcalf[1]

[1]University of Auckland, School of Biological Sciences, Thomas Building, 2A Symonds Street, Auckland, Auckland, 1001, New Zealand, [2]Protein Crystal Corporation, Osaka, Japan, [3]Swiss Light Source at Paul Scherrer Institute, Villigen, Switzerland, [4]Altana Pharma AG, Konstanz, Germany, [5]Kyoto Institute of Technology, Kyoto, Japan, E-mail：f.coulibaly@auckland.ac.nz

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

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

Marc Kvansakul[1], M F van Delft[1], H Yang[1], E F Lee[1], W D Fairlie[1], P E Czabotar[1], J M Gulbis[1], S F Fischer[1], M A Perugini[2], D C Huang[1], P M Colman[1]

[1]The Walter and Eliza Hall Institute of Medical Research, STRUCTURAL BIOLOGY, 1G ROYAL PARADE, Parkville, Victoria, 3050, Australia, [2]Department of Biochemistry and Molecular Biology, The University of Melbourne, Parkville, Victoria 3010, Australia, E-mail：kvansakul@wehi.edu.au

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

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Structure of influenza H5N1 nucleoprotein and its interaction with RNA

Pang-Chui Shaw[1], Andy Ka-Leung Ng[1], Hongmin Zhang[2], Jiahuai Wang[2], Kemin Tan[3], Shannon Wing-Ngor Au[1]

[1]Chinese University of Hong Kong, Department of Biochemistry, The Chinee University of Hong Kong, Shatin, Hong Kong, NT, China, [2]Department of Medical Oncology, Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA, USA, [3]Midwest Center for Structural Genomics and Structural Biology Center, Argonne National Laboratory, Argonne, IL, USA, [4]Department of Microbiology, The Chinese University of Hong Kong, Shatin, Hong Kong, NT, China, E-mail：pcshaw@cuhk.edu.hk

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