M^{-1} and possess antibacterial properties.

The multifunctionality of HBP makes it highly relevant for structural investigations. The structural information contributing to the elucidation of HBP's multifunctionality and the role of the heavy glycosylation.

1. Flodgaard H. et al., Eur. J. Biochem, 197, 535-547 (1991).
2. Østergaard E. and Flodgaard H., J. Leukocyte Biol., 51, 316-323 (1992).
3. Østergaard E. et al., APMIS, 100, 1073-1080 (1992).

PS04.15.19 CRYSTALLIZATION AND PRELIMINARY X-RAY STUDY OF THE VISCUM ALBUM ML1 TOXIN IN ITS NATIVE STATE. N.V.Konareva, A.A.Mikhailov, Institute of Crystallography, Moscow, Russia A.G.Tonevitskii, I.I.Agapov, D.E.Temjakov SRIIMGS, Moscow, Russia A.N.Popov, H.D.Bartunik GBF/MPG, DESY, Hamburg, Germany

The Viscum album ML1 toxin belongs to a group of phytotoxins, such as abrin, ricin, or modeccin, which inhibit the protein synthesis of eukaryotic cells. The toxins of this group are glycoproteins with molecular weights of about 60 kDa. They consist of two subunits, which have their own functions. One of the subunits is lectin with two sites of galactose binding, and the other subunit is a highly specific N-glycosidase - an enzyme, which modifies the 28S RNA-60S ribosomal subunits. The catalitic A (active) and the binding B (binding) subunits are linked into a dimer by a disulfide bond. A handing-drop version of vapor diffusion was used for crystal growth. A precipitating countersolution consisted of ammonium sulfate (in a concentration of 30-35% to a saturated at 20 C solution) and 0.14 M NaCl in a 0.1 M acetate or 0.1M phosphate buffer in a pH range from 4 to 5. The crystallization solution contained equel volumes of a solution of protein (10-13 mg/ml) in one of the above-mentioned buffers and a countersolution. To reduce the number of crystal nuclei, dioxane was added in an amount of 0.01 to 0.02 the volume of the crystallization solution. The X-ray difraction data were collected using the BW6 synchrotron station with a DORIS storage ring (DESY,GBF/MPG, Hamburg, Germany). Intensities were registered using a MARresearch Imaging Plate Scanner. Crystal data are: sp.gr.P6122 (P6522); a=b=110.4 Å, c=309.6 Å. Intensities of 20,683 unique reflections with I>s were measured in the resolution region from20 to 3 Å resolution. The completeness of the data set is 89% and R(I)merge=5.5%.

PS04.15.20 STRUCTURAL BASIS OF HIV-1 PROTEASE INHIBITION BY A MONOCLONAL ANTIBODY. Julien Lescar[1], Renata Stouracova[1,2], MarieMadeleine Riottot[1], Véronique Chitarra[1], Jiri Brynda[1,2], Juraj Sedlacek[2], Graham A. Bentley[1], [1]Unité d'Immunologie Structurale, Institut Pasteur, Paris, [2]Department of Gene Manipulation, Institute of Molecular Genetics, Academy of Sciences of the Czech Republic, Prague

The crystal structure of the Fab fragment of the anti-HIV-1 protease monoclonal antibody, F11.2.32, has been solved both in the unligated state and as two different complexes with cross-reacting peptide fragments from the viral enzyme corresponding to residues 36-46 and 36-57. This antibody inhibits HIV-1 protease activity with an inhibition constant in the micromolar range. Diffraction measurements made at cryogenic temperatures gave Bragg intensities to 3.0 Å resolution for the free Fab, 2.1 Å resolution for the Fab complexed to peptide 36-46 and 2.6 Å resolution for the Fab complexed to peptide 36-57. Initial models for the three crystal structures were obtained by molecular replacement and refined by simulated anealing.

The structures reveal the peptide to be bound at the centre of the antigen-binding site between residues 38 to 42, which adopt a type II β turn conformation. There is only partial correspondence between the structure of this region of the peptide and that of the native antigen.

PS04.15.21 CRYSTAL STRUCTURE OF OUTER SURFACE PROTEIN A (OspA) OF *BORRELIA BURGDORFERI* COMPLEXED WITH A MURINE MONOCLONAL Fab. Hong Li[1], John Dunn[1], Benjamin J.Luft[2], & Catherine L. Lawson[1], [1]Biology Department, Brookhaven National Laboratory, Upton, N.Y. 11973, [2]Division of Infectious Diseases, School of Medicine, State University of Stony Brook, Stony Brook, N.Y. 11794-8153

OspA is an abundant outer surface protein of *Borrelia burgdorferi* that is currently being tested in several forms as a prophylactic vaccine against Lyme disease. The crystal structure of recombinant OspA (with amino-terminal lipidated cysteine replaced by non-lipidated alanine) was solved in a complex with the Fab fragment of an agglutinating monoclonal antibody. The structure of OspA will be useful for mapping sequence variability and for defining antigenic epitopes to aid in vaccine design.

OspA consists almost entirely of antiparallel ß-strands with short turns or small loops. Nine strands (5-13) form a central ß-sheet with right-handed twist. Strands 1 to 4 pack against the central sheet in an orthogonal sandwich motif. Strands 14 to 17 and 18 to 21 form two sheets that pack against the sole a-helix terminating at lysine 273, forming a $\beta/\beta/\alpha$ barrel. A homology search of the protein structure data base (DALI) suggests that OspA does not share structure homology with any known proteins. The Fab184.1 binds to loops in the region of the N-terminal sandwich and may stabilize an otherwise flexible N-terminal region. Sequence variability maps to two C-terminal loops on one edge of the central sheet, plus one loop of the C-terminal barrel.

The structure was solved by a combination of multiple isomorphous replacement and molecular replacement methods. The refined model consists of residues 22 to 273 of OspA (28 kD), all residues of Fab184.1 (50 kD), and 324 waters (current R-factor is 22.9 % and R free is 29.4 at 1.95 Å).

PS04.15.22 CRYSTAL STRUCTURE OF A CDR3 MUTANT OF A T CELL ANTIGEN RECEPTOR Va DOMAIN. Hongmin Li[1], Marina I. Lebedeva[1], E. Sally Ward[2], and Roy A. Mariuzza[1]. [1]Center for Advanced Research in Biotechnology, University of Maryland Biotechnology Institute, 9600 Gudelsky Drive, Rockville, MD 20850; [2]University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75235-8576

The crystal structure of an engineered T cell receptor Va domain containing a grafted CDR3 was determined at 2.3Å resolution by molecular replacement using the wild type Va domain (1) as a search model. The final R factor is 0.156 with R.M.S. deviations from standard bond lengths and bond angles of 0.010Å and 1.936°, respectively. Like the wild type Va, the mutant Va crystallized as a homodimer very similar to antibody V_LV_H dimers However, the relative orientation of the two chains in the mutant Va homodimer differs from that in the wild type by a rotation of 14°. The buried surface area in the dimer interface of the mutant is 24.1Å^2 less than that in the wild type. While the residues forming the interface are essentially the same in the two structures, there are only five pairs of interface hydrogen bonds in the case of mutant compared with 17 for the wild type. This implies that the mutant Va homodimer is stabilized mainly by hydrophobic interactions. The difference in relative orientation of the Va domains in the two dimers will be discussed in the context of the variability in domain orientation reported for V_LV_L and V_LV_H dimers.

1. Fields et al, Science 270, 1821-1824 (1995).