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The Vault particle, with an astounding molecular weight of 13 MDa, is the largest Ribonucleoprotein described. Although its function is still unclear, it has been related to cell signalling, proliferation and immune responses. The Vault complex shows an overall barrel-shaped structure organized in two identical moieties, each consisting of thirty-nine copies of the major vault protein MVP. Earlier data indicated that Vault particles can dissociate at acidic pH.

Recently, we solved the crystal structure of the Vault particle at 8 Å resolution, together with the 2.1 Å structure of the seven N-terminal domains (R1-R7) of MVP. The structure reveals the interactions that govern Vault association and provides an explanation for a reversible dissociation induced by low pH. [1]. This information complemented the 3.5Å X-ray structure of the vault particle, published at the beginning of 2009 by other authors [2], showing the MVP monomers folded into twelve distinct domains: nine repeat domains, a shoulder domain, a cap-helix and a cap ring. The comparison between the structures of R1-R7 and the equivalent region in the 3.5Å structure of the entire particle showed important discrepancies in the tracing of domains R1 and R2. Furthermore, in the structure reported by Tanaka et al., all the atomic positions and temperature factors of the 39 copies of MVP found in the crystal asymmetric unit were included explicitly in the refinement (PDB ids. 2ZU0, 2ZV4 and 2ZV5, with an averaged temperature factor of 121.4 Å2). In this report, refinement of the vault structure was performed using the DEN protocols as included in the Refine low-resolution package of CNS [3]. During refinement the 39-fold non-crystallographic symmetry was always strictly maintained with constrains applied to a single MVP monomer, which was generated as a hybrid model containing the R1R7 structure at the N-terminus (residues form Glu4 to Val380) and the coordinates corresponding to monomer A (PDB id: 2ZV4) at the C-terminus (residues from P381 to G814). The deposited structure factors of the 3.5 Å vault structure (PDB id: 2ZU0) were used as experimental data. Refinement was combined with averaging and solvent flattening with DM and the quality of the resulting maps was also enhanced by applying a negative Bsharp value in a resolution dependent weighting scheme (map sharpening).

This refinement suggests important movements in the N-terminus of the MVP structure during the closing of the two Vault halves and provides an improved view of the C-terminal region.

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Preparation and Characterization of the complex [Co^{III}(TpivPP)(4,4'-bipy)(Cl)]

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Cobalt porphyrin species are specially used as models for hemoproteines and actually investigations concerning these species includes many areas such as organic reaction catalysis [1], [2] biosensors [3] and materials presenting non-linear optical activities.The (4,4'-bib yridine)(chlorido)(α,α,α,α-tetrakis(*o*-pivalamidophenyl)porphyrinato) cobalt(III) has been synthesized and characterized by UV-vis, IR and proton NMR spectroscopy which indicate, inter alia, that the cobalt ion presents the oxidation state III. The X-ray molecular structure shows that the Co(III) ion is hexa-coordinated by the four nitrogen atoms of the pyrrole rings of the TpivPP anion, the chlorido axial ligand on the pocket side of the porphyrin and the nitrogen atom of 4,4'-bipyridine ligand. The average cobalt-pyrrole nitrogen atoms is Co-N_p = 1.983 (2) Å, the Co—Cl and Co—N(bpy) bond lengths are respectively 2.230 (1)Å and 2.030 (3)Å. The compound [Co^{III}(TpivPP)(Cl)(4,4'-bipy)] crystallizes in the monoclinic space group C2/c with unit cell dimensions *a*=18,906(5) Å, *b*=19,112(6) Å, *c*=18,264(8) Å, *β*=92,112°. The structure was refined to *R*=4,61%, WR_2=13,78% and S=1,079 .

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Interaction of Alzheimer β-amyloid and metal complexes with lipid bilayers

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Alzheimer's disease is an age-related disorder characterized by progressive cognitive decline and neurodegeneration. One of the key features of this disease is the presence of amyloid plaques associated with neuritic degeneration [1]. The amyloid plaques are composed predominantly of 40-42-residue peptides, the amyloid β-peptide (βA), being the βA(1-42) the most toxic. Soluble βA peptides interact with cell membranes and have been proposed to affect membrane integrity leading to apoptosis. The molecular mechanisms of these interactions are still unclear. In the context of these observations we investigated the molecular mechanisms that might explain the abnormal accumulation of aluminum in the brains of patients with Alzheimer's disease.

There are reports indicating that Al(III) interacts with cell membranes inducing structural and functional perturbations. They might be due to: a) direct interaction of Al(III) with proteins forming ion channels, receptors and enzymes, b) induction of structural alterations in the membrane lipid bilayer, or c) action on the lipid-protein interfaces. To elucidate among these alternatives, and given the structural complexity of native cell membranes, we utilized molecular models consisting of bilayers of dimyristoylphosphatidylcholine (DMPC) and dimyristoyl-phosphatidylethanolamine (DMPE), representative of phospholipid classes located in the outer and inner monolayers of many cell membranes, respectively.

The capacity of βA and of its complexes with Al, Zn, Cu and Fe to perturb the multibilayer structures of DMPC and DMPE was evaluated by X-ray diffraction. In the context of these studies we report that βA, in our experimental conditions, did not alter any of the bilayer structures while the addition of the βA-Al complex deeply disordered that of DMPC membrane [2]. Our findings imply that Al, compared to the other βA-metal complexes tested could have a specifically relevant effect in enhancing βA toxicity. It is important to stress that, in our experimental conditions, it is not Al alone which causes such alterations but the