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2D modelization of A(C1 + C3) and B(C2 + C3) subunits of rat prostate steroid binding protein

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A strong homology has been recently detected between the primary sequences of polypeptide components C1, C2 or rat prostate steroid binding protein (PSBP) and that of the uteroglobin monomer U[1]. Using the results of the predicted secondary structures[2] of the C1, C2, C3 components of the PSBP and the U chain, and also the experimental 3D model of uteroglobin dimer (U + U) refined at high resolution[3], we have constructed the 3D structures of the two similar subunits of PSBP, domain A(C1 + C3) and domain B(C2 + C3). For this model building, the general Manosk software[4] working on a PS300 Evans & Sutherland colour graphics system was used. The main secondary structures of the uteroglobin dimer has been kept for PSBP A and B units, deletions and insertions occuring principally within loops between α helices. The 3–69′ and 69–3′ disulphide bridges of uteroglobin are kept unmodified in the PSBP. Moreover, the replacement of Val44 (and Val44′), near the 2-fold axis of uteroglobin dimer, by Cys45 for C1, Cys49 for C2 and Cys48 for C3 gives rise to a new disulphide bridge. This feature brings a very strong support to the putative 3D structural homology between uteroglobin and the subunits A and B of PSBP. From a comparison with the first proposal primary sequence homology[2], a new alignment with U of the end parts of the C1, C2 components was proposed, considering the 3D models of the A and B subunits of the PSBP. However, for limited areas, some uncertainty still arises concerning the actual structure of the PSBP. The use of a macromolecular energy minimization package (Ceram[5]) has completed the model building of the A and B subunits, and gave the conformational energy evaluation for each step of the energy refinement. The resulting geometry of the two subunits of the PSBP, are proposed here. An energy refinement on the C3 component of the PSBP was also made by Robson-Platt program. From these models, we propose a possible way of association between the two units A and B.

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State-of-the-art of computing hardware for molecular modelling: a benchmark and its results

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The performances of a number of computers ranging from microprocessor based workstations to so-called 'Crayettes', have been evaluated in a real-life test using the Brugel package for macromolecular mechanics and dynamics.

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Simulation of the 3D structure of pAR5 mutant ATCase using computer graphics and energy minimization

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The pAR5 mutant aspartate transcarbamylase of *E.coli* differs from the 'wild' type enzyme by eight amino-acids at the C-terminal end of the regulatory chains that are replaced by a new sequence of 6 amino-acids. The altered form lacks cooperative interactions between catalytic sites and is insensitive to the feedback inhibitor CTP. It is sensitive to ATP and retains catalytic functions[1]. Assuming the structure of the pAR5 protein to be close to that of the crystalline T form established by the group of Lipscomb[2], we have modelled the conformational rearrangement due to the mutation. We have found that it affects a region that is important for both homotropic and heterotropic interactions, using the Manosk V1.1 software[3] for modelling and graphics. Two amino-acids are deleted and six others substituted at the C-terminal of the regulatory chain. The energy minimization facility of Ceram[4] is then applied to both the native and mutant structures of the allosteric domain. Only minimal modifications are observed after 200 cycles of energy minimization. Contacts with neighbouring catalytic chains can then be analysed in the mutant.

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