the base of the I helix. The distinct atomic shifts of invariant Asp 169 away from the active site may prevent interactions between its main chain amide and the pyrimidine of the 5-fluoro-2'-deoxyuridylate inhibitor and between the Asp 169 side chain and the quinazoline ring of the cofactor. These movements could explain the decreased sensitivity of the mutant enzyme to 5-fluoro-2'-deoxyuridylate inhibition.

**PS04.17.37 STRUCTURAL COMPARISON OF AMYLOIDOGENIC LIGHT CHAIN DIMER IN TWO CRYSTAL FORMS WITH NONAMYLOIDOGENIC COUNTERPARTS.** Norbert Schormann and Merrill D. Benson, Dept. of Medicine, Indiana University School of Medicine, Indianapolis, IN 46202; USA.

In the majority of cases, immunocyte-derived amyloid light chain (AL) (primary) amyloidosis is associated with a nonmalignant expansion of a single plasma cell clone. The monoclonal immunoglobulin (IG) light chain product of the plasma cell clone is the precursor of the amyloid fibril subunit protein.

To investigate structural factors in amyloid fibril formation an attempt has been made to compare the variable region of amyloid proteins with nonamyloid light chain proteins with the goal of developing a structural model for the polymerization or aggregation process of IG light chain proteins.

A cl protein from an individual (BRE) with amyloidosis was completely characterized at the primary structure level. The DNA sequence coding for the variable segment of the cl light chain (cl Vl) was amplified by PCR from the patient's bone marrow DNA using the determined amino acid sequence. The DNA construct was used to express the recombinant cl Vl protein in vitro. Protein BRE was purified to homogeneity and crystallized using ammonium sulfate as precipitant. Two distinct crystal forms were obtained (Crystal form I: monoclinic, space group P21, with a=82.37Å, b=77.75Å, c=82.23Å, β=119.97°; crystal form II: orthorhombic, space group C2221, with a=82.04Å, b=142.11Å, c=77.86Å).

An effort was made to verify the original Laue group assignment, since both space groups are lower symmetry subgroups of hexagonal space group P6122. The data sets were reindexed and processed using older R-Axis processing software and the newer BioteX program package. Reindexing trials and Laue checks of 'merge files' from different indexing solutions for crystal form I showed unambiguously that the crystal structure is correctly assigned as monoclinic. This structure was refined to 2.8Å. In the case of crystal form II, differences in R-syn and R-merge between indexing solutions were not as pronounced to discriminate against the hexagonal crystal system. In addition to refining the structure of BRE II in space group C222, to 1.8Å, we therefore included refinement in two possible hexagonal space groups (P61 and P6322, with a=b=82.05Å, c=77.87Å, γ=120°).

The structures of both crystal forms were compared to each other and to nonamyloidogenic light chain dimers with special emphasis on domain-domain interactions. Models for amyloid fibril formation in AL amyloidosis are discussed.

**PS04.17.38 STRUCTURE SOLUTION OF SOME C-REACTIVE PROTEINS.** Annette K. Shrive, David Holden, Allison Metcalfe, Dean A.A. Mylles, Margaret Hopkins, Ian D. Glover, David Hoolel, Anne C. Bloomer2 and Trevor J. Greenhough*, Dept. of Physics, and Biological Sciences, Keele University, Keele, Staffs, ST5 5BG, UK. MRC Laboratory of Molecular Biology, Hills Road, Cambridge CB2 2QH, UK. a and CCLRC Daresbury Laboratory, Warrington WA4 4AD, UK

C-reactive protein (CRP) is a member of the pentraxin family, a conserved, phylogenetically ancient super-family of oligo-