

## E0059

**Crystal structures of wild-type and mutant (E261Q) of long-chain acyl-CoA dehydrogenase with and without substrate respectively.** B. Narayanan, H. K. Lee<sup>1</sup>, A. W. Strauss<sup>2</sup>, R. Paschke and J.-J. P. Kim, Dept. of Biochemistry, Medical College of Wisconsin, Milwaukee, WI, USA, <sup>1</sup>Visiting Professor from Chungju National Univ., Chungju, Korea, <sup>2</sup>Dept. of Pediatrics, Vanderbilt Univ., Nashville, Tennessee, USA.

Long chain acyl-CoA dehydrogenase (LCAD) is one of the nine known members of the acyl-CoA dehydrogenase flavoprotein family that are involved in fatty acid catabolism and some amino acid metabolism and catalyze  $\alpha,\beta$ -dehydrogenation of the CoA-thioester substrate. Wild-type and mutant human LCAD were expressed in *E.Coli*, purified to homogeneity, and crystallized by vapor diffusion technique.

Diffraction data to 2.4Å were collected from crystals of both wild-type and mutant LCAD. The wild-type LCAD crystal belongs to space group P1 (a=86.2, b=94.8, c=119.0Å;  $\alpha=89.2^\circ$ ,  $\beta=75.0^\circ$ ,  $\gamma=88.4^\circ$ ) and contained two tetramers per ASU; while the mutant LCAD crystallizes in the space group P2<sub>1</sub>2<sub>1</sub>2<sub>1</sub> (a=101.1, b=102.0, and c=173.6Å) and has one tetramer per ASU. The structures were solved by molecular replacement using the human MCAD structure as the probe.

The preliminary structures reveal that Glu261 functions as the catalytic base that abstracts the proton of the substrate. The active site geometry reveals enough room for palmitoyl-CoA(C16-CoA) to bind in its active site.