SMART Teams 2017-2018 MSOE UNIVERSITY

Qualification Phase

Task #8

Finalized Abstract and Model Summary Sheet Submission

Due: Friday, November 3rd

1. **Finalize your Abstract:**
   You have written and edited an abstract detailing the molecular story of the protein Glutathione-S-Trasnferase. At this time, you should have received ample feedback from your Teammates, Teacher Advisor and Judy in order to ensure the abstract is as well-written as it possibly can be to help you tell your molecular story. (See Task 9 – Communicating You Science)

2. **Use your finalized abstract and your Jmol file to create an official "Model Summary Sheet":**
   This will serve as a display for your model. It combines the abstract, an image of your model design, and all the design specifications for the features displayed on your protein. **Please use the Template Model Summary provided on the CBM SMART Teams Task Page.** A sample from a previous year is included for your reference on the next page.

The model description summary sheet should include the following information:

Page One:
- ✓ 17-18 SMART Teams Letter Header on the top
- ✓ Include all the basics: School, Authors, Teacher(s), Abstract Title, PDB File, Primary Citation.
- ✓ **Format.** Alpha carbon backbone
- ✓ **RP.** Zcorp with plaster
- ✓ Your finalized abstract text
- ✓ **Jmol Illustration**. Include a picture of your finalized Jmol design (with a white background).

Page Two:
- ✓ **Specific Model Information:** Using your finalized design sheet, list all the displayed features of your model, and their specific Jmol color. Describe why displayed domains, sidechains, ligands, or any other special features are of importance in your molecular story.
- ✓ **Reference to the CBM SMART Teams Website.** Include the URL for the SMART Team website on your model description sheet: http://cbm.msoe.edu/smartTeams/smartTeamsLocal.php

Due: Friday, November 3rd

First, second and third place WILL be awarded for Task #8!

Qualification Model Summary

PAST SAMPLE:

Valders High School SMART Team

Vanessa Bratz, Alyssa Christianson, Sanne De Bruijin, Elizabeth Evans, Meagan Green, Paige Howard, Supriya Leitner, Kristin Schneider, Mariah Ulness, Jacqueline Wenzel

Teacher: Joe Kinscher

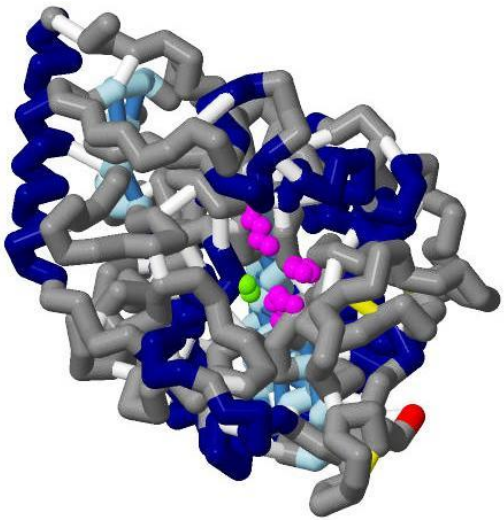
Modeling Arylsulfatase A (ASA)

PDB: 1e1z

Primary Citation: von Bulow, Rixa, Schmidt, Bernhard, Dierks, Thomas, von Figura, Kurt, Uson Isabel (2001). Crystal Structure of an Enzyme-Substrate Complex Provides Insight into the Interaction between Human Arylsulfatase A and its Substrates During Catalysis. J. Mol. Biol. 305, 269-277.

Format: Alpha carbon backbone

RP: Zcorp with plaster



Description:
Metachromatic leukodystrophy (MLD) affects 1 in every 40,000 to 160,000 individuals worldwide. This lethal condition is caused by an inherited autosomal recessive disorder characterized by the accumulation of sulfatide fats in myelin producing cells. Individuals diagnosed with MLD have one or more mutations in their ARSA gene. This gene provides instructions for producing the enzyme arylsulfatase A (ASA) which is located in cellular waste removing lysosomes. There, ASA aides in breaking down sulfatide fats. However, the mutation in the ARSA gene causes an inability to break down these fats. This causes a buildup of sulfatide fats in myelin producing cells, which have the purpose of insulating, protecting, and propagating nerve impulses. Nerve cells, covered in myelin, make up white matter. The accumulation of sulfatides in myelin producing cells causes progressive destruction of these cells, and thus white matter throughout the central and peripheral nervous system is destroyed. White matter damage leads to the progressive deterioration of intellectual functions and motor skills. Individuals with this disorder eventually lose awareness of their surroundings and become unresponsive. The Valders SMART (Students Modeling A Research Topic) Team modeled ASA using 3D printing technology to study structure-function relationships. Ser69 is responsible for forming a covalent bond with the sulfatide fat. Also in the active site, His229, Lys302, Lys123, and Ser150 form hydrogen bonds with the sulfatide holding it for hydrolysis. A Ser69 mutation in the ARSA gene diminishes the ability of ASA to hydrolyze sulfatide fats, resulting in MLD.

Specific Model Information:

Amino acid side chains involved in catalysis:

- Ser69 covalently bonds with the sulfatide and is displayed in ball and stick and colored chartreuse

The following amino acid side chains form hydrogen bonds with the ligand and hold it in the active site during hydrolysis. Side chains are displayed in ball and stick and colored magenta.

- His229
- Lys 302
- Lys 123
- Ser 150

Highlighted protein structures:

- Alpha helix structures are colored dark blue
- Beta sheet structures are colored light blue
- Hydrogen bonds are colored steel blue
- Disulfide bonds are colored yellow
- N-terminus is colored blue
- C-terminus is colored red

Supporting struts are white