# Sample Abstracts from Past Qualification Models

Two abstracts from past qualification models have been provided to help guide the team in the abstract writing process. The first abstract highlights the major components for inclusion in an abstract. An additional example is provided as well. We encourage your SMART Team to practice in identifying these components on the second abstract.

**********

**Marquette University High School SMART Team**

P. Ahn, K. Arnhold, S. Bartos, B. Burkle, M. Chang, M. Coury, N. Cowan, R. Craze, N. Dittrich, R. Foster, J. French, T. Gamblin, J. Gorski, M. Hameed, R. Johnson, S. Khan, E. Lewis, J. Otten, M. Rivera, A. Sabatino, T. Sargent, J. Schimmels, A. Smith, R. Stegeman, D. Strom, J. Strom, B. Tsuji, C. Visaya, J. Yamat, and N. Yorke.

Teachers: Mr. Klestinski & Mr. Kaiser

**Commented [BJ1]:** School, student & teacher names

## Modeling Chlorotoxin

**Commented [BJ2]:** Title should be more informative. I would have titled it: "Chlorotoxin: A Novel Approach to Fighting Cancer"

**PDB:** 1CHL

**Primary Citation:** Lippens, G., Najib, J., Wodak S., Tartar a. (1994). NMR Sequential assignments and Solution Structure of Chlorotoxin, a Small Scorpion Toxin That Blocks Chloride Channels. *Biochemistry*, 34: 13-21.

**Commented [BJ3]:** PDB file and corresponding primary citation paper info.

**Format:** Alpha carbon backbone

**RP:** Zcorp with plaster

**Commented [BJ4]:** Default Model specifications. Please include this.

**Description:**

The American Brain Tumor Association reports that 700,000 people in the U.S. are living with a brain or central nervous system tumor. According to Dr. Jim Olson, chlorotoxin, a chloride channel blocker extracted from the venom of the Deathstalker scorpion (Leiurus quinquestriatus), can be injected into cancer patients to target brain tumors. Chlorotoxin selectively interacts with the protein matrix metalloproteinase 2 (MMP-2), as well as Cl- channels, which are upregulated in many cancers. Chlorotoxin, when bound with a fluorescent dye, can make it easier for doctors to identify and remove cancerous cells without harming healthy tissue. The Marquette High School SMART (Students Modeling A Research Topic) Team has designed a model of chlorotoxin using 3D printing technology to investigate structure-function relationships. Chlorotoxin, a small protein with only 36 amino acids, has a three-stranded beta sheet, an alpha helix, and four disulfide bridges that aid in the stability of the protein. Three lysines,15, 23, and 27, are present that bind to Cy5.5, a near-infrared fluorescence-emitting dye. In order to better expedite drug development, the CTX/Cy5.5 molecule must be mono-labeled. Alanine or arginine mutations were introduced to lysines 15 and 23 ensuring Cy5.5 binds to lysine 27 only. Research has shown chlorotoxin's ability to target other forms of cancer such as melanoma, small cell lung carcinoma, and neuroblastoma. Future cancer research should focus on other ways of utilizing chlorotoxin's cancer-targeting properties. Perhaps chlorotoxin could be used not just to "paint tumors" but to eradicate them completely.

**Commented [BJ5]:** The "Hook" or "Big Picture" statement to grab the audience.

**Commented [BJ6]:** Protein name and origin

**Commented [BJ7]:** A snippet on function. I'd recommend expanding on this.

**Commented [BJ8]:** The impact; why we care about this research!

**Commented [BJ9]:** SMART Team Modeling Statement… put this AFTER your structure discussion.

**Commented [BJ10]:** Structural features discussion

**Commented [BJ11]:** 244 word count. Within appropriate range (200-250)

**Commented [BJ12]:** Impact, future research and concluding statement that ties back in to the opening "hook".

## Sample Abstract from Past Qualification Models


### 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: Mr. Joe Kinscher


Commented [BJ13]: Take a look at this abstract. Did they include everything? Do you suggest any changes? Discuss!

### Arylsulfatase A

**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 Interation 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.