# **SMART Teams**

**Exploring the**

**Molecular World**



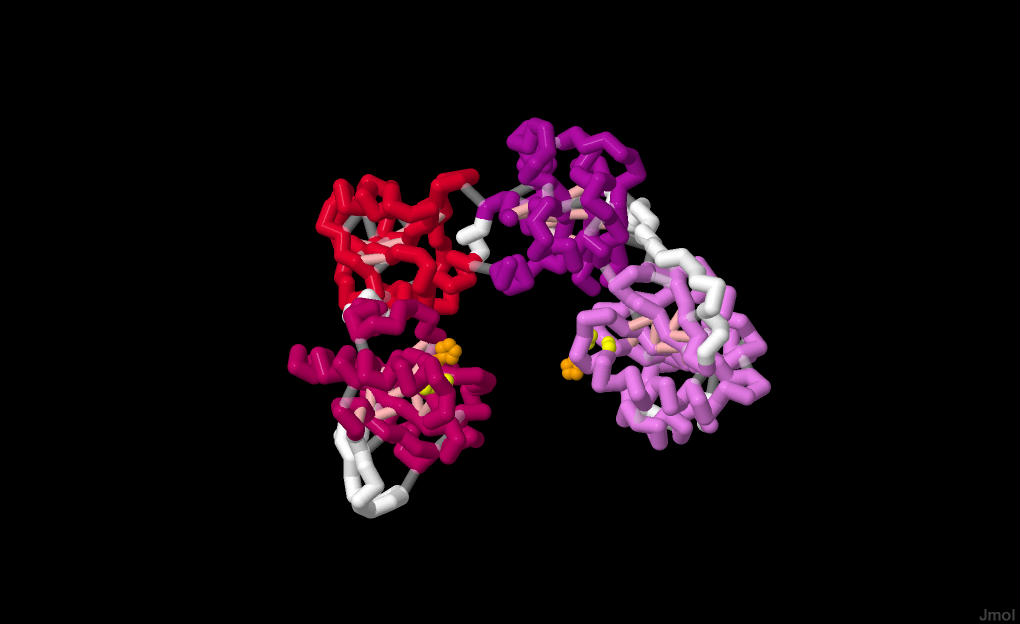
**DC. Everest SMART Team Project 2012-2013**

**Author: Alexis Abrego**

**Mentor: Dr. Shannon Colton**

**Advisor: Bill Heeren**

*reduced*



**Legend**

Domain 1: 23–130 [199,0,102] raspberry

Domain 2: 135–230 [230,0,46]cherry red

Domain 3: 235–344 [179,13,166] sugar plum

Domain 4: 368–471 - [238,130,238]frosted pink

backbone – white

Cys 53 & 56 & 397 & 400 - yellow



His55, 399- orange

ssbonds- [87,23,143]royal purple

hbonds [255,181,181]salmon pink

*oxidized*

In the life of as cell bonds are made and broken every single moment. Some bonds can be created and broken simply in the cytosol while other bonds require more controlled environments that only chaperonin and enzymes can provide. The protein Human Protein Disulfide Isomerase (HPDI) is a chaperonin and an enzyme that breaks the disulfide bonds in molecules.

This HPDI’s function of breaking disulfide bonds is conducted primarily by the cysteines in the two active sites (Cys 53 & 55, Cys 397 & 397). These molecules make the conformational change of breaking the ssbond by reducing it. This reaction is aided by His55 and 399 (orange) by adding to the redox potential value. In addition to conducting a redox reaction in the active site, the entire protein is also moderated by redox reaction. When it is in its oxidized form the protein is more spread out allowing the active site to be reached more easily. In an opposite fashion when the protein is reduced the active sites are more hidden and thus reaction speeds are greatly reduced. In the case of Ricin, the reducing of the disulfide bond between the A-chain and the B-chain is an essential piece to the tale of Ricin’s toxic purpose. For that disulfide bond which connects the two chains keeps the toxic A-chain from being able to neutralize ribosomes. The speed of the reaction also depends on how many HPDI proteins are in oxidized versus the reduced form.