^{1}H, ^{13}C and ^{15}N resonance assignments for the symmetric homodimer ArsD

Jun Ye[1], Yanan He[1], Jack Skalicky[2], Barry P. Rosen[1] and Timothy L. Stemmler[1]

[1] Department of Biochemistry and Molecular Biology, Wayne State University, and [2] Department of Biochemistry, University of Utah

The *arsRDABC* operon of plasmid R773 confers resistance to arsenite and antimonite in *Escherichia coli.* The *arsD* gene encodes a 13 KDa protein that has a dual function as an As(III) chaperone (see poster by Y. F. Lin) and as a *trans*-acting repressor that regulates *arsRDABC* transcription. One physiological role of ArsD is to control expression of ArsB. ArsB is integral membrane protein that functions as an arsenite efflux protein, and high level expression of ArsB can be toxic to the cell. ArsD is a homodimer of 118-residue monomers that binds to the operator/promoter site of the *ars* operon. It responds to environmental trivalent metalloids As(III) and Sb(III) by dissociating from the DNA, allowing transcription of the *ars* genes. ArsD is a novel protein, with only six known homologues, and is not related to any known regulatory proteins. Its structure may yield insight into its mechanism of DNA binding and metalloid responsiveness.

We have undertaken the NMR characterization of ArsD to provide the basis for understanding the structural aspects of how the protein functions. Here we report the atomic assignments for the symmetric ArsD homodimer. These assignments provide the first step in characterizing the proteins solution structure and dynamic properties. In addition, these data serve as controls that will allow us to identify amino acid specific chemical environment changes in ArsD that are coupled with the binding of arsenite, antimonite and DNA. Therefore, the work outlined in this report provides the basis for future experiments already in progress, directed at understanding the mechanism by which ArsD helps regulates ArsB expression and hence controls cellular As toxicity in *E. coli*.