Allosteric control mechanism of the mercury-responsive transcriptional regulator MerR

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Bacteria participate in mercury transformation in natural and industrial environments. The bacterial *mer* operon in transposons or plasmids mediates mercury resistance by encoding specific genes that facilitate uptake of mercuric species, cleavage of Hg-C bonds, and reduction of Hg(II) to Hg(0). MerR responds specifically to nanomolar levels of Hg(II) by activating the transcription of the *mer* operon genes. Small angle x-ray scattering experiments and molecular dynamics were applied to study the allosteric control mechanism of MerR in the presence and absence of Hg(II). Binding of Hg(II) induces a significant reorientation of the MerR homodimer that causes two DNA-binding domains to open and rotate. A population-shift mechanism is proposed causing DNA-underwinding by MerR. This mechanism could explain why the Hg(II)-bound MerR has a lower affinity to DNA than the Hg(II)-free form. Here, we present the complete dynamics of transcription activation by MerR upon Hg(II)-binding.