

P032 Molecular basis of a CRP-FNR transcriptional regulator:
how CprK controls halo-respiration

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Halogen containing compounds including chlorinated ethenes and chlorophenols are amongst the most abundant pollutants in the soil, sediments and groundwater, caused primarily by past and present industrial and agricultural activities. Over a decade ago, evidence emerged that certain bacteria are able to conserve energy via the reductive dehalogenation of halo-organic compounds in a respiration-type metabolism. The transcriptional regulator CprK induces expression of halo-respiratory genes upon binding of o-chlorophenol ligands and is reversibly inactivated by oxygen through disulphide bond formation. We report crystal structures of CprK in the ligand-free, ligand-bound and DNA-bound states, making it the first member of the widespread CRP-FNR protein superfamily for which a complete structural description of both the redox-dependent and allosteric molecular rearrangements is available. Comparison of individual structures, in conjunction with kinetic and thermodynamic ligand binding studies, indicate that binding of the halogenated phenolate ligand occurs with extreme positive cooperativity. Comparison of the CprK structures with other members of the CRP-FNR superfamily reveal that, despite resemblance in overall structure, distinct allosteric mechanisms are employed to control their specific DNA binding capacity.