Anion-independent Iron Binding by *Campylobacter jejuni* Ferric Binding Protein

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The bacterium *Campylobacter jejuni* is a leading cause of human gastroenteritis. Under iron limited conditions, *C. jejuni* expresses a ferric binding protein (cFbpA) that in many pathogenic bacteria functions to acquire iron as part of their virulence repertoire. The cFbpA crystal grew in space group $P2_1$ with 2 molecules in the asymmetric unit. The biological unit is the monomer.

The overall structure of cFbpA [1] consists of 2 globular domains linked by 2 β-strands. The cFbpA crystal structure reveals unprecedented iron coordination in a distorted octahedral geometry by only 5 protein ligands. The histidine and 1 tyrosine are from the N-terminal domain, whereas the 3 remaining tyrosine ligands are from the C-terminal domain. Surprisingly, a synergistic anion is not observed in the cFbpA iron binding site suggesting a novel role for this protein in iron uptake. In the well-characterized ferric transport proteins, the respective synergistic anions are important for iron binding and release. The 4 Tyr ligands are 1.9 to 2.1 Å from the iron and have B-factors similar to that of the iron (13 $Å^2$). In contrast, His14 forms a weaker interaction with iron (~2.3 Å) and the imidazole ring average B-factor is elevated (20.6 $Å^2$). His14 may mediate iron release similar to carbonate in transferrin.

[1] Tom-Yew S.A.L., Cui D. T., Bekker E.G., Murphy M.E.P., *J. Biol. Chem.*, 2004.

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