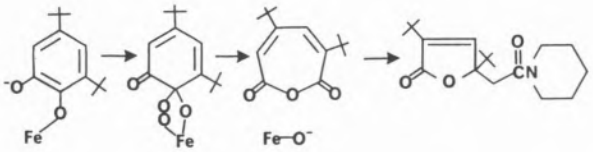
becomes monodentate during the reaction, as suggested by the structure of the $[Fe(NTA)DBC]^{2-}$ complex which shows an unsymmetrically chelated DBC ligand (Fe-O(DBC), 1.89 and 1.98 A). The cleavage reaction is initiated by the breaking of the longer Fe-O(DBC) bond. The time required for the reaction (4 days) probably reflects the energy necessary to break this bond. The activated complex then reacts with O_2 to form a peroxide intermediate. It is proposed that the ferric center coordinates the peroxide and facilitates its decomposition to the desired cleavage product as illustrated below:



REFERENCES

[1] L. QUE JR., *Adv. Inorg. Biochem.*, **5**, 167-199 (1983).
[2] J.W. PYRZ, A.L. ROE, L.J. STERN, L. QUE JR., *J. Am. Chem. Soc.*, **107**, 614-620 (1985).
[3] L. QUE JR., J.D. LIPSCOMB, E. MÜNCK, J.M. WOOD, *Biochim. Biophys. Acta*, **485**, 60-74 (1977).
[4] R.H. HEISTAND II, A.L. ROE, L. QUE JR., *Inorg. Chem.*, **21**, 676-681 (1982).
[5] M.R. ROGERS, L. QUE JR., unpublished results.
[6] R.B. LAUFFER, R.H. HEISTAND II, L. QUE JR., *Inorg. Chem.*, **22**, 50-55 (1983).
[7] L.S. WHITE, P.V. NILSSON, L.H. PIGNOLET, L. QUE JR., *J. Am. Chem. Soc.*, **106**, 8312-8313 (1984).
[8] R.B. LAUFFER, R.H. HEISTAND II, L. QUE JR., *J. Am. Chem. Soc.*, **103**, 3947-3949 (1981).
[9] M.G. WELLER, U. WESER, *J. Am. Chem. Soc.*, **104**, 3752-3754 (1982).

MS5.10 — FR

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STRUCTURE, RECOGNITION AND TRANSPORT OF FERRIC ENTEROBACTIN IN *E. COLI*

The transport and uptake of iron by microbes, a process which is essential for their growth, is mediated by low-molecular-weight complexing agents called siderophores [1,2]. A siderophore produced by *E. coli*, enterochelin [3] (here called enterobactin [4]), is the most powerful iron complexing agent known and has been among the most thoroughly studied of the siderophores [5]. Ferric enterobactin transport in *E. coli* has been studied with respect to the specificity of the outer membrane protein receptor and the mechanism of enterobactin-mediated transport of ferric ion across the outer membrane. Transport kinetic and inhibition studies were performed with ferric enterobactin and synthetic structural analogs (Fig. 1) to map the parts of the molecule important for receptor binding. The ferric complex of the synthetic structural analog of enterobactin, 1,3,5-N,N',N''-tris(2,3-dihydroxybenzoyl)-triaminomethylbenzene (MECAM) is transported with the same maximum velocity as ferric enterobactin. A double label transport assay with $^{59}Fe[^{3}H]$-MECAM showed that the ligand and the metal are transported across the outer membrane when a large excess of extracellular complex was added to the cell suspension. At least 60% of internalized ^{59}Fe enterobactin exchanged with extracellular ^{55}Fe enterobactin (Fig. 2). Internalized $^{59}Fe[^{3}H]$MECAM was released from the cell as

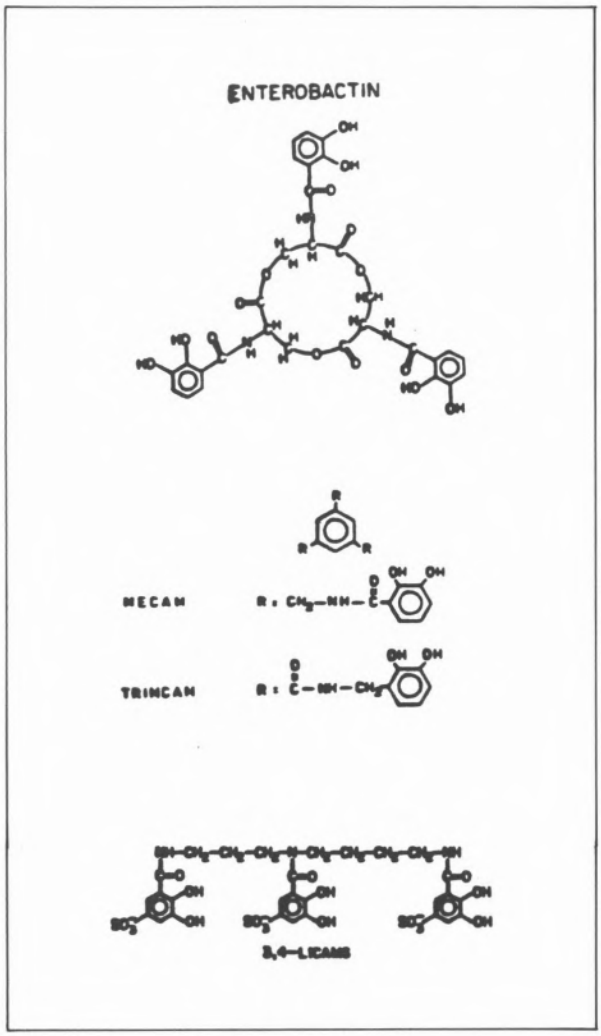

Fig. 1

The structure of enterobactin and several synthetic enterobactin analogs: MECAM[1,3,5-N,N',N''-tris(2,3-dihydroxybenzoyl)triaminomethylbenzene]; TRIMCAM[1,3,5-tris(2,3-dihydroxybenzoylcarbamido)benzene]; and LICAMS[1,5,10-N,N',N''-tris(5-sulfo-2,3-dihydroxybenzoyl)triazadecane]

the intact complex when either unlabeled Fe MECAM or Fe enterobactin was added extracellularly. The results suggest a mechanism of active transport of unmodified coordination complex across the outer membrane with possible accumulation in the periplasm. Energy-dependent binding of ^{67}Ga enterobactin was observed, but the rate was substantially lower than the rate of ^{59}Fe enterobactin transport. The results establish important correlations between the coordination chemistry of the metal and the mechanism of receptor-mediated uptake.

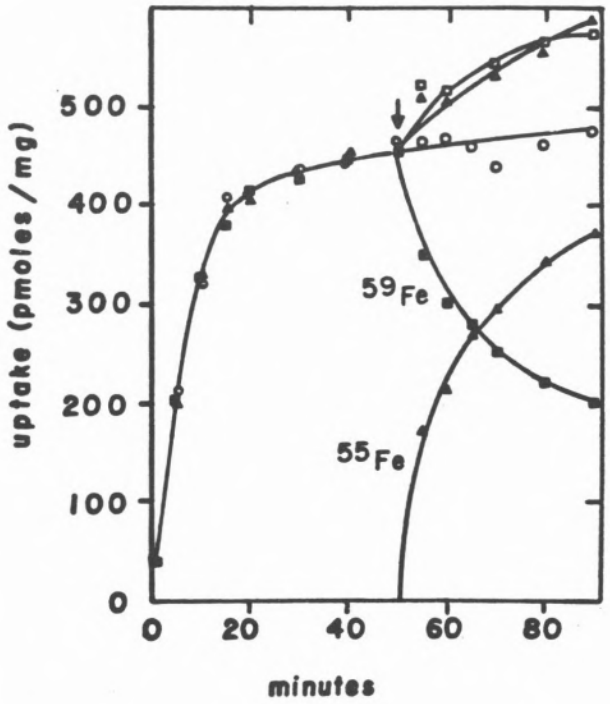

Fig. 2

Exchange of external and cellular ferric enterobactin. The cell concentration was 1.22 mg/mL and the pH was 7.4. In all experiments the initial concentration of ^{59}Fe enterobactin was 2μM; ○, ^{59}Fe enterobactin uptake with no additions; Δ, (control) ^{59}Fe enterobactin uptake with the addition (at 51 min, arrow) of the same substrate at 30 μM concentrations; ■, ^{59}Fe enterobactin uptake with addition (at 51 min, arrow) of ^{55}Fe enterobactin at 30 μM concentrations; ▲, ^{55}Fe enterobactin accumulation in the same experiment; □, numerical sum of ^{55}Fe (▲) and ^{59}Fe(■) in the same experiment

REFERENCES

[1] K.N. Raymond, G. Müller, B.F. Matzanke, *Topics in Current Chemistry*, **123**, 49-102 (1984).

[2] J.B. Neilands, *Ann. Rev. Nutr.*, **1**, 27-46 (1981).

[3] I.G. O'Brien, F. Gibson, *Biochim. Biophys. Acta*, **215**, 393 (1970).

[4] J.R. Pollack, J.B. Neilands, *Biochem. Biophys. Res. Comm.*, **38**, 989 (1970).

[5] G. Müller, Y. Isowa, K.N. Raymond, *J. Biol. Chem.*, (1984), submitted for publication.

MS5.11 — FR

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ACTIVATION OF HYDROPEROXIDES BY $Fe^{II}(MeCN)_4(ClO_4)_2$ AND $Fe^{III}Cl_3$ IN ACETONITRILE; MODEL SYSTEMS FOR THE ACTIVE SITES OF *PEROXIDASES*, *CATALASE*, AND *MONOXYGENASES*

Addition of $Fe^{II}(MeCN)_4(ClO_4)_2$ to solutions of hydrogen peroxide in dry acetonitrile catalyzes the rapid disproportionation of H_2O_2 via initial formation of a $Fe^{II}(H_2O_2)^{2+}$ adduct, which, in turn, oxidizes a second H_2O_2 to yield dioxygen. The intermediate of the latter step dioxygenates diphenylisobenzofuran, 9,10-diphenylanthracene, and rubrene, which are traps for singlet-state dioxygen. This intermediate also dioxygenates electron-rich unsaturated carbon-carbon bonds

$[Ph_2C=CPh_2 \longrightarrow 2Ph_2C(O)$,
$PhC \equiv CPh \longrightarrow PhC(O)C(O)Ph$,
$cis\text{-}PhCH=CHPh \rightarrow 2PhCH(O)]$.

In the presence of organic substrates such as 1,4-cyclohexadiene, 1,2-diphenylhydrazine, catechols, and thiols, the Fe(II)-H_2O_2/MeCN system yields dehydrogenated products (PhH, PhN=NPh, quinones, and RSSR) with conversion efficiencies that range from 100% to 17%. Although the Fe(II) catalyst does not promote the disproportionation of Me_3COOH or *m*-ClPhC(O)OOH, these hydroperoxides are activated for the dehydrogenation of organic substrates. With substrates such as alcohols, aldehydes, methyl styrene, thioethers, sulfoxides, and phosphines, the $Fe^{II}(H_2O_2)^{2+}$ adduct promotes their monoxygenation to aldehydes, carboxylic acids, epoxide, sulfoxides, sulfones, and phosphine oxides, respectively.

$$Fe(II) + H_2O_2 \longrightarrow Fe^{II}(H_2O_2)^{2+} \xrightarrow{RH} Fe(II) + ROH + H_2O$$

The reaction efficiencies for the group of substrates with the Fe(II) adducts that are formed by H_2O_2, Me_3COOH, and *m*-ClPhC(O)OOH have been evaluated. Also, the reaction rates for the substrate-$[Fe^{II}(H_2O_2)^{2+}]$ dehydrogenations and monoxygenations relative to that for Ph_2SO have been determined, as have the substituent effects for the monoxygenation of 4-X-$PhCH_2OH$ and 4-X-PhCH(O). The $Fe^{II}(H_2O_2)^{2+}$ adduct is an efficient catalyst for the autoxygenation of PhCH(O) to PhC(O)OOH. In all of these processes the iron(II) catalyst remains in its reduced state.

Solutions of $Fe^{III}Cl_3$ in dry acetonitrile also catalyze the rapid disproportionation of H_2O_2 to O_2 and H_2O, but the catalyst remains in the Fe(III) state. In the presence of triphenylphosphine, dimethyl sulfoxide, and olefins the $Fe^{III}Cl_3$-H_2O_2/MeCN system yields monoxygenated substrates (Ph_3PO, Me_2SO_2, and epoxides). The epoxidation of olefins is especially favored by the $Fe^{III}Cl_3$-H_2O_2 adduct.

Both of these catalyst systems [$Fe^{II}(MeCN)_4(ClO_4)_2$ and $Fe^{III}Cl_3$] in dry acetonitrile activate hydroperoxides for the dehydrogenation and monoxygenation of organic substrates, and do not promote radical processes (Fenton chemistry). Their ability to facilitate these reactions via the oxene chemistry of ferryl (FeO^{2+}) and perferryl ($FeCl_3O$) make them useful reaction mimics for the active sites of *peroxidases*, *catalase*, and *monoxygenases*.