IRON ACQUISITION FROM FERRITIN NANOCAGE

Rabindra Kumar Behera

Department of Chemistry, National Institute of Technology, Rourkela - 769008, Odisha, India

Email: beherarabi@nitrkl.ac.in

Ferritins are self-assembled, symmetrical, hollow, intracellular iron storage proteins, which can store thousands of iron atoms in the form of ferrihydrite ($Fe_2O_3.xH_2O$) mineral inside its nanocavity [1, 2]. Iron is vital to all organisms, including pathogens for their virulence. When needed, ferritin iron is released in a controlled way for various cellular activities. However, the low solubility of Fe(III) at neutral pH and stable ferritin protein cage prevents easy iron exit [2]. In contrast, high solubility of Fe(II), selective ferritin pore modification and limited dissolved oxygen concentration, favors the iron release process. Therefore, in order to facilitate iron release, ferritin pores were modified by rational protein engineering and reductive approach was employed using a combination of physiological reducing agent (NADH) and suitable electron transfer (ET) mediators. Our study reveals that the reductive mobilization of iron from ferritin is dependent on relative rate of NADH oxidation, dissolved oxygen consumption and mineral core reduction, which again depend on the mid-point potential of mediators and their ability to shuttle the electron from NADH to ferritin core. Moreover, altering electrostatics around symmetric ferritin pores found to accelerate the kinetics of Fe(II) acquisition [3].

References:

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