DOWNLOAD PDF IRON BINDING PROTEINS

Chapter 1 : Transferrin - Wikipedia

Iron-binding proteins are carrier proteins and metalloproteins which play many important roles in various organic processes, including metabolism, the immune response, and delivery pathways for small molecules such as oxygen (when bound to iron in heme).

Next Section Abstract Cyanobacteria account for a significant percentage of aquatic primary productivity even in areas where the concentrations of essential micronutrients are extremely low. To better understand the mechanism of iron selectivity and transport, the structure of the solute binding domain of an ATP binding cassette iron transporter, FutA1, was determined in the presence and absence of iron. There are extensive interactions between these ligating residues and the rest of the protein such that the conformations of the side chains remain relatively unchanged as the iron is released by the opening of the metal binding cleft. This is in stark contrast to the zinc-binding protein, ZnuA, where the domains of the metal-binding protein remain relatively fixed, whereas the ligating residues rotate out of the binding pocket upon metal release. This motion may require relatively little energy since total contact area between the domains is the same whether the protein is in the open or closed conformation. Consistent with the pH dependence of iron binding, the main trigger for iron release is likely the histidine in the iron-binding site. Finally, neither FutA1 nor FutA2 binds iron as a siderophore complex or in the presence of anions, and both preferentially bind ferrous over ferric ions. Previous Section Next Section Bioavailable iron is a limiting nutrient for primary production in large areas of the oceans. This concentration of free iron in aquatic environments is dynamic and varies greatly depending upon the local environment. Microbes play a large role in the cycling of iron between the ferric and ferrous forms and generally reduce ferric iron under anaerobic conditions by using it as a final electron acceptor. Conversely, microbes can oxidize ferrous iron under aerobic conditions when other compounds such as nitrate are the final electron acceptors. Organisms can import either form of iron. A number of bacteria, algae, and Cyanobacteria increase the bioavailability of ferric iron through the secretion of organic molecules, such as siderophores, into the extracellular environment. In some cases these siderophores may actually facilitate a photochemical reduction of the bound ferric ion 2. Alternatively, ferric iron can be locally reduced to the more soluble ferrous form and imported directly. This latter can be accomplished by the organisms itself as is the case with some algal species that use ferric chelate reductase to reduce the iron before import or via a symbiotic organism such as bacteria associated with the roots of freshwater and marine macrophytes that reduce ferric iron in the rhizosphere 3. Both Gram-negative and Gram-positive bacteria have evolved ATP binding cassette-type transport systems for the high affinity uptake of transition metal ions such as iron, manganese, and zinc, particularly at low extracellular levels of these metals 4 - 6. The ATP binding cassette-type binding proteins from a number of bacteria have been grouped into clusters on the basis of their sequence homologies and the metal ligand identities 7. In Synechocystis , the futA1 slr and futA2 slr genes encode periplasmic-binding proteins that have been proposed to be part of a high affinity iron transport system. Because high concentrations of citrate inhibited iron uptake, it was concluded that the iron was not being transported as a ferric citrate complex. These results suggested that there was at least some redundancy in the functions of FutA1 and FutA2. Recently, it was shown that FutA2 binds iron with approximately the same relative affinity as FutA1 9. However, when futA2 is deleted, the mutants accumulate normal levels of iron-containing ferredoxin in the periplasm but fail to accumulate copper in plastocyanin. This result combined with the fact that Synechocystis expresses FutA2 to high levels led to the conclusion that it might not be directly involved in iron transport but, instead, sequesters iron in the periplasm and acts as a metallochaperone. As so, they suggested that it might assist in iron import as well as prevent these ions from competing with other metal binding periplasmic proteins such as those which bind copper. Here we report the high resolution structure of FutA1 in the presence and absence of bound iron. FutA1 has a C-clamp structure that is very similar to that of Campylobacter jejuni 10 and binds iron via four tyrosine and one histidine residues in the absence of anions or siderophores. There are extensive interactions between the chelating residues and the rest of the protein such that these residues remain in a relatively fixed conformation upon the

release of iron and the opening of the iron-binding site via domain movement. From analysis of the changes in the contact surface area, this rocking motion may require relatively small changes in the total energy of the protein and, therefore, may have minimal impact on the release or binding of iron. This observation is consistent with the homology of FutA1 to the C. An amino acid sequence alignment between FutA1 and FutA2 indicates that the iron-binding site of FutA2 is also composed of four tyrosines and one histidine. Therefore, it is not surprising that we found that purified FutA2 binds ferrous rather than ferric iron. Consistent with this finding is the fact that the iron binding configuration of these proteins is conserved among bacterial strains that favor low oxygen environments where ferrous iron is more prominent. The pETa Novagen vector was previously modified such that the thrombin cleavage site was replaced with a tobacco etch virus TEV cleavage site. Thirty minutes after lowering the temperature, protein expression was induced by the addition of 0. The cells were allowed to grow for an additional 16 h before harvesting by centrifugation. Cells were lysed on ice by four s cycles of sonication separated by 3 min of cooling. Before crystallization, a final concentration of 1 mm ammonium ferric sulfate Sigma-Aldrich and 1 mm citrate were added to FutA1, which enhanced the deep red-brown color of the purified protein. Ferric iron was used in these early experiments because literature had suggested that FutA1 was a ferric-binding protein 8. It should be noted, however, that only high concentrations of freshly prepared iron solutions enhanced the color of the protein. Colorless apoFutA1 crystals were obtained at room temperature via the hanging drop method of vapor diffusion against 2. Red-colored crystals of Fut1 complexed with iron were obtained in a similar manner as the apo crystals, with a substitution of mm HEPPS, pH 8. The iron-loaded protein crystals were flash-frozen for synchrotron data collection. Briefly, crystals were harvested from the hanging drop experiments and soaked for several hours in synthetic mother liquor composed of mm HEPPS, pH 8. The crystals were then flash-cooled by submersion in liquid nitrogen. X-ray data processing statistics are presented in Table 1.

Chapter 2 : The Structure of the Iron-binding Protein, FutA1, from Synechocystis

Lactoferrin is an iron-binding protein closely related to the serum iron transport protein, transferrin, and is part of the larger transferrin protein family. Lactoferrin is found in mucosal secretions (tears, saliva, vaginal fluids, urine, nasal and bronchial secretions, bile, GI fluids) and notably in milk and colostrum.

The serum iron test measure the amount of iron in your blood. The total iron-binding capacity TIBC test looks at how well the iron moves through your body. Iron is an important mineral that your body needs to stay healthy. Your body uses iron to make hemoglobin, the protein in your red blood cells that carries oxygen throughout your body. This condition is called iron deficiency anemia. Iron in your body is carried, or bound, mainly to a protein made by your liver called transferrin. The TIBC test is based on certain proteins, including transferrin, found in the blood. Your transferrin levels are almost always measured along with iron and TIBC. Why do I need these tests? You may need these tests if your healthcare provider thinks your iron level is too low or too high. Not having enough iron in your diet is the most common cause of anemia and the most common type of diet deficiency in the U. Your healthcare provider may do this test to look at your diet, nutrition, liver, or other conditions that cause iron to be low, such as increased blood loss or pregnancy. Symptoms of iron deficiency include: Being tired and feeling weak Getting frequent infections Feeling cold all the time Having swelling in the tongue Struggling to keep up at school or work In children, having delayed mental development Symptoms of too much iron can include: Feeling tired and weak Belly pain What other tests might I have along with these tests? Iron, TIBC, and transferrin blood tests are almost always done together. Other blood tests that may also be done include measuring your hemoglobin; your percent of red blood cells, or hematocrit; and all the cells in your blood, called a complete blood count. What do my test results mean? Test results may vary depending on your age, gender, health history, the method used for the test, and other things. Your test results may not mean you have a problem. Ask your healthcare provider what your test results mean for you. Normal results of iron testing may be different for men, women, and children. Normal results for iron are: Some common conditions that may cause the amount of iron in your blood to be too low include:

Chapter 3 : Antimicrobial properties of iron-binding proteins.

The iron is thus bound in a polynuclear non-ionic iron-hydroxide, which accounts for their low toxicity. These iron ions interact strongly with endogenous iron-binding proteins in the body.

Under normal circumstances, about one-third of transferrin iron-binding pockets are filled. Consequently, with the exception of iron overload where all the transferrin binding sites are occupied, non-transferrin-bound iron in the circulation is virtually nonexistent. Distribution of plasma and tissue iron can be traced using 59Fe as a radioactive tag. The subject receives autologous transferrin loaded with radioactive iron that then can be monitored. Blood samples can be analyzed at timed intervals to determine the rate of loss of the radioactive label. Such ferrokinetic studies indicate that the normal half-life of iron in the circulation is about 75 minutes Huff et al. The absolute amount of iron released from transferrin per unit time is the plasma iron turnover PIT. Such radioactive tracer studies indicate that at least eighty percent of the iron bound to circulating transferrin is delivered to the bone marrow and incorporated into newly formed erythrocytes Jandl and Katz, ; Finch et al. Other major sites of iron delivery include the liver, which is a primary depot for stored iron, and the spleen. Hepatic iron is found in both reticuloendothelial cells and hepatocytes. Reticuloendothelial cells acquire iron primarily by phagocytosis and breakdown of aging red cells These cells extract the iron from heme and return it to the circulation bound to transferrin. Hepatocytes take up iron by at least two different pathways. The first involves receptor-mediated endocytosis of transferrin. In addition, hepatocytes can take up ionic iron by a process independent of transferrin Inman and Wesling-Resnick, Ferrokinetics and the Bone Marrow Given the preeminent role of the bone marrow in the clearance of labeled iron from the circulation, ferrokinetics provide a window on erythropoietic activity. Conditions that augment erythrocyte production increase the PIT. For example, hemolytic anemias such as hereditary spherocytosis and sickle cell disease induce rapid delivery of transferrin-bound iron to the marrow. In contrast, disorders that reduce red cell production prolong the PIT. This picture is seen, for example, with anemia due to Diamond Blackfan anemia. When erythrocytes are produced and released into the circulation in a normal fashion, the process of erythropoiesis is termed "effective". In patients with certain hemolytic anemias, however, the nascent red cells are so abnormal they are destroyed before leaving the marrow cavity. In this circumstance, the erythropoiesis is "ineffective", meaning simply that the erythropoietic precursors have failed to accomplish their primary task: The ferrokinetic profiles such cases show rapid removal of iron from transferrin with a delayed entry of label into the pool of circulating red cell hemoglobin. Cellular Iron Uptake Although transferrin was characterized fifty years ago Laurell and Ingelman, , its receptor eluded investigators until the early s. In a quest to better understand the behavior of neoplastic cells, investigators prepared monoclonal antibodies against tumor cells. The target of these monoclonal antibodies later was found to be the cell surface transferrin receptor glycoprotein Sutherland et al. A broad body of literature now supports the concept that the iron-transferrin complex is internalized by receptor-mediated endocytosis. The general structure of the transferrin receptor is shown in Figure 2. This disulfide-linked homodimer has subunits containing amino acids each Kuhn et al. Four glycosylation sites three N-linked and one O-linked line the protein Hayes et al. Glycosylation-defective mutants have fewer disulfide bridges, bind transferrin less efficiently and are expressed less prominently on the surface expression than are normal receptors Williams and Enns, a ; Williams and Enns, b. Schematic representation of the transferrin receptor Figure 2. The molecule is a transmembrane homodimer linked by disulfide bonds. An acyl group attached to the cytoplasmic tail of the molecule anchors the assembly to the plasma membrane. The transmembrane domain, between amino acids 62 and 89, functions as an internal signal peptide, as none exits at the N-terminal end Zerial et al. A molecule of fatty acid usually palmitate covalently links each subunit to the internal edge of the transmembrane domain and could play a role in membrane localization. Interestingly, non-acylated mutants mediate faster iron uptake than normal receptors Alvarez et al. The transferrin binding regions of the protein are unidentified Williams and Enns, a ; Williams and Enns, b. Efforts to crystallize transferrin receptor protein are underway. Iron is taken into cells by receptor-mediated endocytosis of monoferric and diferric transferrin Karin and Mintz, ; Klausner et al. Receptors on the outer face of the plasma

membrane bind iron-loaded transferrin with a very high affinity. The C-terminal domain of transferrin appears to mediate receptor binding Zak et al. Diferric transferrin binds with higher affinity than monoferric transferrin or apotransferrin Huebers et al. The dissociation constant Kd for bound diferric transferrin ranges from M to M at physiologic pH, depending on the species and tissue assayed Stein and Sussman, ; Sawyer and Krantz, The Kd of monoferric transferrin is approximately M. The concentration of circulating transferrin is about 25 mM. Therefore, cellular transferrin receptors ordinarily are fully saturated. After binding to its receptor on the cell surface, transferrin is rapidly internalized by invagination of clathrin-coated pits with formation of endocytic vesicles Figure 3. This process requires the short, 61 amino acid intracellular tail of the transferrin receptor molecule Rothenberger et al. Receptors with truncated N-terminal cytoplasmic domains do not recycle Rothenberger et al. This portion of the molecule contains a conserved tyrosine-threonine-arginine-phenylalanine YTRF sequence which functions as a signal for endocytotic internalization Collawn et al. Genetically engineered addition of a second YTRF sequence enhances receptor endocytosis Collawn et al. A number of stimuli reversibly phosphorylate the serine residue adjacent to the YTRF sequence, at position 24 by the action of protein kinase C Davis et al. The role of receptor phosphorylation is unclear. Despite removal of the phosphorylation site by site-directed mutagenesis, the transferrin receptor recycles normally Rothenberger et al. Receptor-mediated transferrin endocytosis Figure 3. Ferro-transferrin binds to transferrin receptors on the external surface of the cell. The complex is internalized into an endosome, where the pH is lowered to about 5. Iron separates from the transferrin molecule, moving into the cell cytoplasm. Here, an iron transport molecule shuttles the iron to various points in the cell, including mitochondria and ferritin. Ferritin molecules accumulate excess iron. Lysosomes engulf aggregates of ferritin molecules in a process termed "autophagy". An ATP-dependent proton pump lowers the pH of the endosome to about 5. The acidification of the endosome weakens the association between iron and transferrin. Even at pH 5. Conformational changes in the transferrin receptor also play a role in iron release Bali et al. Rather than entering lysosomes for degradation, as do ligands in other receptor-mediated endocytosis pathways, intact receptor-bound apotransferrin recycles to the cell surface, where neutral pH promotes detachment into the circulation Zak and Aisen, Thus the preservation and re-use of transferrin are accomplished by pH-dependent changes in the affinity of transferrin for its receptor Van Renswoude et al. Exported apotransferrin binds additional iron and undergoes further rounds of iron delivery to cells. The average transferrin molecule, with a half-life of eight days, may be used up to one hundred times for iron delivery Harford et al. Topologically, the cell exterior and the endosome interior are equivalent compartments. The primary role of the transferrin-transferrin receptor interaction is to bring iron into the vicinity of the cell surface, thereby increasing the likelihood of iron uptake. Following its release from transferrin within the endosome, iron must traverse the plasma membrane to enter the cytosol proper. The molecules effecting this transport have not been identified, but the process may be carrier-mediated Egyed, Their cells take up ferrotransferrin into endosomes, but fail to release iron into the cytoplasm Garrick et al. The molecular basis of the defects in these animals have not been elucidated. The endosomal transporter may reside on the plasma membrane of the cell prior to endocytosis Pollack, If so, it should be oriented to transport iron directly into the cell, without the assistance of transferrin. Such non-transferrin-bound iron uptake activities have been characterized in tissue culture. This uptake system could function constitutively but inefficiently. Coupling the transferrin cycle to transport across the plasma membrane might augment iron uptake by creating an iron-rich environment for the transporter within the endosome. This same elusive transport molecule could also be involved in intestinal iron uptake. Once inside the cell cytoplasm, iron appears to be bound by a low molecular weight carrier molecule, which may assist in delivery to various intracellular locations including mitochondria for heme biosynthesis and ferritin for storage. The identity of the intracellular iron carrier molecule s remains unknown. The amount of iron in transit within the cell at any given time is minuscule and defies precise measurement. Metabolically inactive iron, stored in ferritin and hemosiderin, is in equilibrium with exchangeable iron bound to the low molecular weight carrier molecule Figure 3. Both prokaryotes and eukaryotes produce ferritin molecules for iron storage. Ferritins are complex twenty-four subunit heteropolymers of H for heavy or heart and L for light or liver protein subunits Theil, L subunits are The

subunits of the ferritin molecule form a sphere with a central cavity in which up to atoms of crystalline iron is stored in the form of poly-iron-phosphate oxide Theil, Eight channels through the sphere are lined by hydrophilic amino acid residues along the three-fold axes of symmetry and six more are lined by hydrophobic residues along the four-fold axes; [Harrison et al. Strong interspecies amino acid conservation exists in the residues that line the hydrophilic channels, while marked variation exists in those along the hydrophobic passages. Hydrophilic channels terminate with aspartic acid and glutamic acid residues , and are lined by serine, histidine and cysteine residues all of which potentially bind metal ligands. The evolutionary conservation of the hydrophilic channels suggests that they provide the route for iron entry and exit from the ferritin shell, but this contention remains unproved. Little is known about how iron is released from ferritin for use. Although the two ferritin chains are highly homologous, only H ferritin has ferroxidase activity. A mechanism involving dioxygen converts ferrous to ferric iron, promoting incorporation into ferritin Levi et al. The composition of ferritin shells varies from H-subunit homopolymers to L-subunit homopolymers, and includes all possible combinations between the two. Isoelectric focusing of ferritin from a particular tissue reveals multiple bands representing shells with different subunit compositions.

Chapter 4 : Pirin (PIR, Iron Binding Nuclear Protein) - US Biological: P | calendrierdelascience.com

The transferrins are iron-binding proteins with molecular weights of around 80,, which interact with a maximum of two ferric atoms per each protein molecule. The best known transferrins are the serotransferrins from animal sera, lactoferrins from milk, and conalbumin from egg-white. The iron.

Iron Use and Storage in the Body: Ferritin and Molecular Representations Iron in Biology: Louis, MO Key Concepts: Nonpolar Interactively view a molecule in this section. These questions are fundamental to the study of medicine and to many chemists, biologists, and engineers. We know that our bodies are matter, and thus must be composed of atoms that have been specially arranged to produce the molecules and larger structures that sustain our lives. We know that the properties of an atom e. Hence, to study the human body as a complex organization of molecules that undergoes a wide array of interrelated chemical reactions, we should begin by asking one of the most basic questions about any system of molecules: What sort of atoms does the system contain? The complete answer to this question will have two main parts: Hence, our discussion of the human body as a chemical system begins by answering the question, "What type of atoms does the body contain? In fact, only 24 different elements are thought to be essential to the human body. Other elements, such as mercury, are sometimes found in the body, but do not perform any known essential or beneficial function. The other elements in the body, such as calcium, phosphorus, iron, and copper, are known to physiologists as mineral elements and trace elements. These elements must be present in the body in the proper amounts, and they must be available to react with other elements to form critical molecules and participate in important chemical reactions. In this tutorial, we will describe the importance of one essential trace element in the body, iron. Although iron comprises only 0. Metal Complexes in the Blood " tutorial that iron is necessary for oxygen transport in the blood. Recall that iron is the central atom of the heme group, a metal complex that binds molecular oxygen O2 in the lungs and carries it to all of the other cells in the body e. Without iron in the heme group, there would be no site for the oxygen to bind, and thus no oxygen would be delivered to the cells which would result in the cells dying. In addition to hemoglobin, other important proteins in the body that contain heme groups and therefore contain iron include myoglobin, which takes oxygen from hemoglobin and allows the oxygen to diffuse throughout the muscle cells, and the cytochromes, which supply the body with its energy currency. You will learn more about cytochromes in the Chem tutorial, " Energy for the Body: Other proteins, such as those needed for DNA synthesis and cell division, also rely on iron. Furthermore, iron is used to help produce the connective tissues in our body, some of the neurotransmitters in our brain, and to maintain the immune system. Hence, iron is necessary for allowing the cells that need oxygen to obtain O2, for supplying the body with a reliable source of energy, and for maintaining several other important structures and systems in the body. Iron Disorders Because iron plays such a crucial role in the body, it is important for us to maintain an adequate supply of iron to form hemoglobin and the other molecules in the body that depend on iron to function properly. Yet, our bodies continually lose iron in small amounts through everyday process such as urination, defecation, sweating, and sloughing off skin cells. Bleeding, particularly menstrual bleeding in women, contributes to further loss of iron from the body. To compensate for these losses and to maintain an adequate supply of iron, we should consume approximately 18 mg of iron daily. Certain conditions, including heavy bleeding and pregnancy, further increase the requirement for iron consumption. Good dietary sources of iron include red meat, liver, egg yolk, beans, nuts, and fortified cereals. When the deficiency becomes severe so that there are too few circulating red blood cells or the hemoglobin content of these cells is very low , the condition is diagnosed as iron-deficiency anemia. It is also possible to have too much iron deposited in the body tissues. This condition is known as iron overload. If the iron overload becomes severe usually when the total amount of iron in the body exceeds 15 g , the condition is diagnosed as hemochromatosis. A recessive genetic mutation can put some people e. Treatment for hemochromatosis consists of removing blood from the patient to decrease the amount of iron in the body, and treating the symptoms e. The Iron-Storage Protein How does the body regulate the amount of iron? Ferritin Figure 1 is the key to this important control of the amount of iron available to the body. Ferritin

is a protein that stores iron and releases it in a controlled fashion. Hence, the body has a "buffer" against iron deficiency if the blood has too little iron, ferritin can release more and, to a lesser extent, iron overload if the blood and tissues of the body have too much iron, ferritin can help to store the excess iron. Figure 1 This is a three-dimensional representation showing ferritin, the iron-storage protein in the body. Ferritin has a spherical shape, and iron brown is stored as a mineral inside the sphere. This same molecule is shown in another type of representation in Figure 9 , below. The structure of this protein was determined using x-ray crystallography, the structure of the iron core is based on a simplified model compound, and the image was rendered using the Insight II molecular-modeling system from Molecular Simulations, Inc. To view this molecule interactively, please use Jmol , and click on the button to the left. How does ferritin store iron? Ferritin has the shape of a hollow sphere. Inside the sphere, iron is stored in the Fe III oxidation state. Then, the iron leaves through channels in the spherical structure. Molecular Representations Proteins e. To understand how ferritin or any of the many molecules that you will encounter in this course and throughout your experience in the sciences performs its job, we must be able to visualize the three-dimensional structure of the molecule, and understand the relationship between the structural features and the function of the molecule. We could make three-dimensional models to depict the structure of ferritin, but these models would be inconvenient for distributing the information widely. The most common formats for distributing information today- in books and on computer screens- necessitate that the image be displayed in two dimensions. Of course, there are many difficulties involved in converting all of the important structural information about a molecule into an easily understandable two-dimensional representation. No two-dimensional representation can show a three-dimensional structure in its entirety. In this tutorial, the 2D-ChemDraw, stick, CPK, and ribbon representations are used to examine the three-dimensional structure of ferritin. These four types of representations are described in the blue box, below. Types of Representations Used in this Tutorial Graphical computer modeling has greatly improved our ability to represent three-dimensional structures. One of the goals of graphical computer modeling is to create the computer-generated image such that the image seems three-dimensional. By replicating the effect of light on three-dimensional objects, computers can give the impression of depth to simulate the three-dimensional aspect. The ability of interactive molecular viewing e. By interactively rotating the molecules, a clear picture of the three-dimensional structure emerges. In addition, this increases our chemical intuition by looking at two-dimensional images and visualizing the three-dimensional structure in our brains. This tutorial uses different types of structural representations Figure 2, Table 1 , such as 2D-ChemDraw, stick, CPK, and ribbon, to illustrate the structure of ferritin. PDB files are also available for viewing the molecules interactively. By using these various representations to study the structure of ferritin, you will become familiar with the different types of information given by each type of molecular representation, as well as the strengths and limitations of each representation. Figure 2 This figure shows an alpha-helix from the " Hemoglobin and the Heme Group: Metal Complexes in the Blood " tutorial in four different types of computer-generated molecular representations. In the 2D-ChemDraw, stick, and CPK representations, carbon atoms are shown in gray black , nitrogen atoms are shown in blue, and oxygen atoms are shown in red. In this figure, hydrogen atoms light blue are shown in the 2D-ChemDraw representation but hydrogen atoms are not shown in the other representations. By examining the four representations in Figure 2, you can see that each picture tells us something different about the structure of the molecule. For instance, if we wanted to know how the atoms in an alpha helix are connected to one another, we would use the ChemDraw or stick representation. To see the relative sizes of the atoms in an alpha helix, we would use the CPK representation. Descriptions of the four types of representations, their major strengths, and their drawbacks are given in Table 1, below.

Chapter 5 : Iron-binding proteins - Wikipedia

The disease results from loss of function mutations (most often triplet expansion) in the FXN gene that lead to decreased expression of frataxin, a mitochondrial iron-binding protein that interacts with proteins involved in the mitochondrial Fe-S cluster biogenesis [34].

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Iron binding and activity depend on E, D, and H suggesting a direct interaction of Fe2+ with these residues. Furthermore, we demonstrate presence of iron in epithiospecifier protein and nitrile-specifier protein 3 from Arabidopsis thaliana (AtESP and AtNSP3).

Chapter 7 : Ferritin, the Iron-Storage Protein

Iron regulation is an important modifier of renal ischemia-reperfusion injury, but the role of iron-binding proteins during cardiopulmonary bypass remains unclear. The goal was to characterize iron-binding proteins throughout ischemia-reperfusion injury to determine their association with acute kidney injury development.

Chapter 8 : Transerrin and Iron Transport Physiology

"Iron-Binding Proteins" is a descriptor in the National Library of Medicine's controlled vocabulary thesaurus, MeSH (Medical Subject Headings). Descriptors are arranged in a hierarchical structure, which enables searching at various levels of specificity.

Chapter 9 : iron-binding proteins | WordReference Forums

Since transferrin is the primary iron-binding protein, the TIBC test is a good indirect measurement of transferrin availabilityâ€”the amount of transferrin that is available to bind to iron. In healthy individuals, transferrin is one-third saturated with iron.