IRON METABOLISM – STATE OF THE ART 2014

Summary

Iron is biometal, existing in two main oxidation states, i.e. Fe(II)/Fe(III). The extensive range of redox potential available to this metal by varying its interactions with coordinating ligands, as well as its capacity to participate in one-electron transfer reactions, are the reasons why iron is essential for almost all living organisms. Iron is found in the active sites of a large number of enzymes that catalyze diverse redox reactions underlying fundamental metabolic processes, including respiratory oxidation, DNA synthesis, microRNA processing and oxygen transport. On the other hand, iron is toxic due to its capacity to catalyze *via* Fenton reaction the production of hydroxyl radical, a highly destructive oxidant. Cellular iron homeostasis consists in providing iron for a variety of biochemical processes and in limiting iron availability for Fenton reaction. Cellular iron homeostasis is mainly controlled by the iron regulatory proteins (IRP1 and IRP2) – two cytoplasmic RNA-binding proteins involved in the mechanisms that coordinate the synthesis of a number of key proteins responsible for cellular iron transport, storage and utilization. Systemic iron balance is largely based on a regulatory axis between the liver-derived peptide hepcidin and the iron exporter ferroportin proved to be fundamental for the coordination of iron fluctuations in the body and its distribution among the main sites of iron metabolism such as absorptive enterocytes, reticuloendothelial macrophages, hepatocytes and erythroid precursors of red blood cells. The article briefly resumes main discoveries within last 15 years, critical for the understanding iron homeostasis.