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Cytosolic and mitochondrial ferritins in the regulation of cellular iron homeostasis and oxidative damage.

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Abstract

BACKGROUND: Ferritin structure is designed to maintain large amounts of iron in a compact and bioavailable form in solution. All ferritins induce fast Fe(II) oxidation in a reaction catalyzed by a ferroxidase center that consumes Fe(II) and peroxides, the reagents that produce toxic free radicals in the Fenton reaction, and thus have anti-oxidant effects. Cytosolic ferritins are composed of the H- and L-chains, whose expression are regulated by iron at a post-transcriptional level and by oxidative stress at a transcriptional level. The regulation of mitochondrial ferritin expression is presently unclear.

SCOPE OF REVIEW: The scope of the review is to update recent progress regarding the role of ferritins in the regulation of cellular iron and in the response to oxidative stress with particular attention paid to the new roles described for cytosolic ferritins, to genetic disorders caused by mutations of the ferritin L-chain, and new findings on mitochondrial ferritin.

MAJOR CONCLUSIONS: The new data on the adult conditional knockout (KO) mice for the H-chain and on the hereditary ferritinopathies with mutations that reduce ferritin functionality strongly indicate that the major role of ferritins is to protect from the oxidative damage caused by iron deregulation. In addition, the study of mitochondrial ferritin, which is not iron-regulated, indicates that it participates in the protection against oxidative damage, particularly in cells with high oxidative activity.

GENERAL SIGNIFICANCE: Ferritins have a central role in the protection against oxidative damage, but they are also involved in non-iron-dependent processes.

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