Short Communication

Ferrotoxicity: Multiple Mechanisms of Action

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Increasingly, we are becoming aware that excessive/misplaced iron is a notable risk factor for a broad spectrum of diseases [1]. Moreover, the mechanisms of ferrotoxicity differ significantly among specific diseases.

Infectious diseases and cancers

Iron is dangerous because it is an essential growth factor for most bacterial, all fungal and all protozoan infections [2] as well as for all cancer cells [3]. Although viruses do not have independent metabolism, enhanced host iron is needed for viral synthesis [2].

Our bodies employ a constitutive iron withholding defense system that strives to prevent invader access to the metal. Moreover, the system is promptly and markedly upregulated at the time of threatened microbial and viral infections and cancer cell invasions [4].

Chronic diseases

Iron is dangerous because of its oxidative potential for highly sensitive key cells that are specific for the disease. Examples of the key cells include: (1) anterior pituitary cells that stimulate endocrine gland activities [5], (2) osteoblasts that rebuild bone [6] and (3) pancreatic beta cells that produce insulin [7]. These cells are killed by concentrations of iron that are several orders of magnitude lower than lethal quantities of iron for hepatocytes, macrophages, osteoclasts and pancreatic exocrine cells.

Thus it can be predicted that, in other chronic diseases, key body cells for relevant organ functions likewise will be found to be unusually sensitive to iron killing. A prime nominee is the ventricular cardiomyocyte, essential for heart health [8].

Conclusion

Excessive/misplaced iron, a ubiquitous component of disease causation, is dangerous in two ways. For infections and cancers, the metal is essential for invader growth. For chronic maladies, the metal is exceptionally lethal for key cells that are required for specific organ functions.

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