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Commentary

Significant Role of Ferritin in Atherosclerotic Cardiovascular Disease

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ABOUT THE STUDY

Processes required for maintaining life rely solely on iron. It can, however, also result in oxidative damage, which is regarded to be the main focus for a variety of chronic, including cardiovascular disorders. It is yet unclear how iron contributes to atherosclerosis' genesis. Macrophages serve a crucial role in the body's management of iron as well as the development, growth, and de-stabilization of atherosclerotic plaques. Through its actions on macrophages, iron itself may have an effect on atherosclerosis. Targeting iron metabolism inside macrophages, though, may have certain adverse side effects that interfere with the prevention of atherosclerotic plaque formation. Thus, the prevalent view that iron can hasten the development of coronary disease through lipid peroxidation may not fully account for the complex function that iron plays in the pathophysiology of atherosclerosis. Through iron-containing and -sequestering proteins and enzymes that maintain mitochondrial function, DNA synthesis and repair, as well as cell development and death, iron is needed for physiologic activities and plays a significant role in cellular metabolism. It is essential for erythropoiesis and oxygen transport because it is the primary component of haemoglobin. However, iron can also be harmful because of its capacity to produce ROS and oxidise biomolecules by producing poisonous hydroxyl radicals as a result of the Fenton reaction. The interaction of numerous iron-processing organs and cells, including as macrophages, erythrocytes, hepatocytes, and duodenal epithelial cells, as well as the hepcidin-ferroportin axis, tightly regulates iron homeostasis.

The reticulo-endothelial system recycles the majority of the iron needed for life from senescent red cells, with the additional need being fine-tuned by altering the quantity of iron taken by enterocytes. Disease can be caused by abnormalities in the intake or production of iron. The most frequent cause of anaemia is Iron Deficiency (ID), which is a major global health issue. Excess iron can enter the body in some disease conditions like hemochromatosis and affect parenchymal organs including the liver, pancreas, and heart.

90% of the body's daily iron needs are fulfilled by breaking down senescent erythrocytes that are recycled by macrophages. By modulating the quantity of iron that is absorbed, which comes in two primary chemical forms, it is possible to balance the amount of iron lost and the amount needed for growth, anaemia, and pregnancy. Most of it is non-haem ferric iron (Fe^{3+}), which is found in plants and must first be reduced by ferri-reductase in the gut mucosa before being absorbed. The bioavailability of haem iron is higher because haem ferrous iron (Fe^{2+}) from animal source meals can be directly absorbed. Only 1-2 mg of the normal daily intake of 12 to 15 mg iron is actually absorbed.

CONCLUSION

Complex networks efficiently manage iron metabolism to meet daily iron requirements and prevent iron overload. Iron homeostasis is mostly regulated by macrophages, and these cells' regulatory mechanisms are becoming well understood. In experimental models, changing macrophages' intracellular iron metabolism may change how they handle lipids and respond to inflammation, which may have an impact on atherogenesis and make it a viable therapeutic target in cardiovascular illnesses. Hepcidin, the primary regulator of iron homeostasis, may, however, have a dual function as atherosclerosis develops. Hepcidin inhibition may help early plaques by reducing macrophage pro-inflammatory activity, but it may have the opposite impact in areas of IPH due to its pro-angiogenic effects.

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