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Hepcidin

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Abstract

Hepcidin is an iron-regulating peptide hormone made in the liver. It controls the delivery of iron to blood plasma from intestinal cells absorbing iron, from erythrocyte-recycling macrophages, and from iron-storing hepatocytes. Hepcidin acts by binding to and inactivating the sole cellular iron exporter, ferroportin, which delivers iron to plasma from all iron-transporting cells. In a classical endocrine feedback system, hepcidin production is stimulated by plasma iron and iron stores. Reflecting a likely role of hepcidin in innate immunity, hepcidin is also induced by inflammation. Increased erythropoietic activity suppresses hepcidin, which leads to increased iron absorption and release of iron from stores, matching iron supply to increased demand. This suppression of hepcidin is in part mediated by erythroferrone, a hormone produced by erythropoietin-stimulated erythroblasts. Hereditary hemochromatosis is caused by hepcidin deficiency or resistance to hepcidin, and hepcidin deficiency also mediates the hyperabsorption of iron in β -thalassemia and other iron-loading anemias. Pathologically increased concentrations of hepcidin are seen

in iron-refractory iron deficiency anemia, in anemia of inflammation, and anemia of chronic kidney disease where increased hepcidin limits the availability of iron for erythropoiesis. Its central involvement in a variety of iron disorders makes hepcidin an important target for diagnostic and therapeutic applications.

References (46)

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