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Obesity Fitness Expo 2017: Obesity leads to iron retention in the duodenum of mice likely due to increased production of adipose-derived hepcidin- Shougang Wei- Capital Medical University, China

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Obese people and animals have higher rates of iron deficiency (ID) than their normal weight peers. It was still uncertain whether obesity-related ID is a true or functional deficiency of iron. This study was to determine the effects and the possible underlying mechanisms of obesity on duodenal iron absorption and liver iron accumulation. C57BL/6J mice were randomly divided into high-fat diet-induced obese (DIO) group and normal control (NC) group to be fed respectively for 16 weeks. Oral iron absorption was tested by measuring serum iron, liver iron and the retained duodenal iron 90 min after intragastric administration of 57 FeSO4 solution. The protein expression levels of iron transporters in duodenum and liver were evaluated by Western blotting. Hepcidin mRNA levels in the liver and adipose tissues were quantified by real-time RT-PCR. The results showed that DIO mice had significantly higher iron retention in the duodenum, lower iron concentration in plasma and liver than NC mice. The protein expression levels of ferroportin-1 (Fpn1) in duodenum and transferrin receptor-2 (TfR2) in the liver were markedly decreased in DIO mice. Hepcidin mRNA levels in visceral adipose tissue but not in the liver were higher in DIO mice than NC mice. In conclusion, obesity-related ID may attributed to impaired intestinal iron absorption of which iron being retained in the duodenal enterocytes, not to that iron being accumulated in the liver. Increased expression of visceral adipose hepcidin probably is the immediate cause for the malabsorption of iron in obesity by inducing reduction of the duodenal Fpn1.

Iron homeostasis is affected by obesity and obesity-related insulin resistance in many ways. On the one hand, iron deficiency and anemia are common in subjects with progressive stages of obesity. This phenomenon has been well studied in adolescents, women and obese subjects undergoing bariatric surgery. In contrast, hyperferritinemia with normal or slightly elevated transferrin saturation is observed in approximately one-third of patients with Metabolic Syndrome (MetS) or non-alcoholic fatty liver disease (NAFLD). This constellation has been called "Dysmetabolic Iron Overload Syndrome (DIOS)". High body iron stores and iron deficiency are detrimental to health and the evolution of obesity-related conditions. Iron deficiency and anemia can affect mitochondrial and cellular energy homeostasis and further increase inactivity and fatigue in obese subjects. The inflammation associated with obesity is closely linked to iron deficiency and involves impaired duodenal iron absorption associated with low expression of duodenal ferroportin (FPN) as well as high concentrations of hepcidin. This review summarizes the current understanding of deregulation of iron homeostasis in obesity.

Abnormal iron status parameters indicating iron deficiency or overload are common in overweight and obese subjects. Iron deficiency is a particular clinical problem in adolescence when iron requirements increase and in morbid obesity in adulthood. A deterioration in the functional state of iron is mainly linked to an inflammation of adipose tissue and to an increased expression of hepcidin, a systemic iron regulation protein. Cytokines such as TNF-α, IL-1 and IL-6 as well as adipokines (leptin, resistin) or hepcidin can represent signals of obese, inflamed AT, facilitating changes in physiological iron homeostasis. Due to its underlying mechanism of reduced iron absorption from the gut, treatment of iron deficiency with oral supplementation is often insufficient and parenteral substitution is therefore necessary, especially in bariatric surgery patients. As iron deficiency and overload can have detrimental effects on the evolution of obesity-related conditions, diligent screening
And treatment of both warranted
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