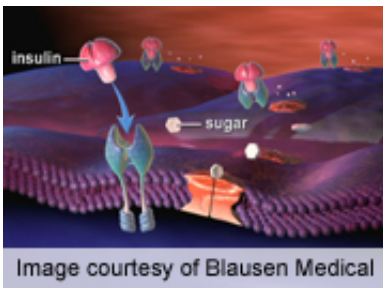


Adipose tissue macrophage iron content altered in obesity

February 4 2014



(HealthDay)—The percentage of adipose tissue (AT) macrophages (ATMs) that are iron rich is reduced in obesity, and those iron-rich ATMs (MFe^{hi}) undergo an inflammatory shift, according to research published in the February issue of *Diabetes*.

Noting that little is known about the role of alternatively activated resident M2 ATMs in AT homeostasis or their function in obesity, Jeb S. Orr, Ph.D., from the Vanderbilt University School of Medicine in Nashville, Tenn., and colleagues report the discovery of a population of alternatively activated ATMs.

The researchers found that there was a population of ATMs with elevated cellular iron content and an iron-recycling [gene expression](#) profile (MFe^{hi}), and the remaining APMs were referred to as MFe^{lo}.

About 25 percent of ATMs in lean mice were MFe^{hi}, and this percentage decreased in obese mice due to recruitment of MFe^{lo}. In obesity, MFe^{hi} ATMs underwent an inflammatory shift; in vivo, the [iron content](#) of MFe^{hi} was reduced, and there was decreased gene expression of iron importers and the iron exporter ferroportin. Exposure of primary peritoneal macrophages to saturated fatty acids in vitro correlated with altered iron metabolism gene expression. In [obese mice](#), the impaired MFe^{hi} iron handling coincided with adipocyte iron overload.

"In conclusion, in obesity, iron distribution is altered both at the cellular and tissue levels, with AT playing a predominant role in this change," the authors write. "An increased availability of [fatty acids](#) during [obesity](#) may contribute to the observed changes in MFe^{hi} ATM phenotype and their reduced capacity to handle iron."

One author is a minor shareholder of Cytoguide ApS; the rat anti-mouse CD163 monoclonal antibody used in the study was a gift from Cytoguide ApS.

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Citation: Adipose tissue macrophage iron content altered in obesity (2014, February 4) retrieved 20 April 2024 from
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