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Editorial

# Iron Overload, Insulin Resistance and Adiponectin: A Possible Pathway Linking

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Iron is an essential biometal for cellular life. All mammalian organisms require definite amounts of iron in order to satisfy metabolic needs or to perform specific functions. Iron is required for the production of red blood cells, the oxygen transport, DNA biosynthesis and energetic metabolism. The production of enzymes also depends on the biometal; these functions are due to its flexible chemistry and redox activity but the biometal can be also toxic due to its capacity to cause oxidative stress, fibrosis and cellular death, via the formation of reactive oxygen species (ROS) [1,2]. A complex of molecular reactions assures the correct iron balance considering both cellular requirements and minimized toxicity [2-5]. Iron perturbations were observed in patients with obesity and insulin resistance (IR): in particular it has long been known that increased iron stores have an effect on the clinical onset of obesity-related conditions [6-10] and that they are associated with increased risk of type 2 diabetes mellitus (T2DM) [11,12]. The pathways linking iron to the IR and the obesity and also the mediators of this association are poorly understood. Within the cross-talk among iron metabolism, diabetes and obesity we considered the role of iron overload in inducing IR and in modulating adipose tissue function and metabolism by considering the association among the markers of iron metabolism (ferritin in particular) and serum adiponectin, an insulin-sensitizing adipokine [13-15].

In order to underline a relation between iron and metabolic profile, in several studies reported some evidences of the association between the biometal and obesity-related conditions; in particular an excess of body iron stores coordinates the clinical course of metabolic dysfunction. On the other side, iron removal improves insulin sensitivity, delaying the clinical manifestation of diabetes. Serum ferritin has proved the main predictor of obesity: an inverse correlation between serum ferritin and the adipocyte-specific hormone, adiponectin, was reported. Adipocytes show an iron sensing role by using iron store levels to modulate adiponectin level: they regulate adipose tissue metabolism and organism metabolism by responding not only to the macronutrient amount but also to the iron status. Proof of this important aspect is the evidence of a regulation of adiponectin transcription by iron.

Iron overload and IR association is attributed to the iron capacity to catalyze oxidative stress by producing ROS: in insulin-resistant and diabetic patients treated with antioxidants an improvement of insulin sensitivity was observed. About the direct iron effect on glucose metabolism, iron induces insulin resistance of glucose transport in adipocytes.

In the cross-talk among iron, diabetes and obesity a very significant role is performed by adipokines signaling, in particular adiponectin, and its contribute to metabolic abnormalities.

## Disclosure Statement

The author declare that there are no conflicts of interest.

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