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Vagal and splenic participation in the iron–hepcidin homeostasis of exercised obese and non-obese male Wistar rats

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Authors: Caroline de Maman Oldra , Giovana Fanhani Tessaro, Ellen Carol...

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Correspondence should be addressed to C de Maman Oldra: carol.dmaman@gmail.com

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Disruptions in iron homeostasis are common during obese states and are related to chronic inflammation and insulin resistance. Exercise exerts well-recognized anti-adiposity and anti-inflammatory effects, besides modulating iron control. The vagus nerve (VN) influences immune and metabolic responses, in a spleen-dependent manner with an unknown impact on iron. Here, we evaluated the effects of the absence of the VN and of the spleen on adiposity, metabolism, and iron homeostasis in non-obese and hypothalamic-obese rats submitted to swimming training. Hypothalamic obesity was induced by the administration of monosodium glutamate (MSG; 4 g/Kg) during the initial postnatal days (PNDs). Non-obese control (CTL) rats received equimolar saline. At PND 60, MSG and CTL were submitted to surgery consisting of bilateral subdiaphragmatic vagotomy (Sv), splenectomy (Spl), Sv + Spl, or sham surgery. At PND 80, the rats were subdivided into exercised (Ex) or sedentary (Sd). Exercised rats swam for 30 min/day for 40 days. At PND 120, the growth, adiposity, metabolism, and iron homeostasis of rats were evaluated. Major results indicate that the absence of the VN and spleen favors the anti-adiposity effects of exercise, particularly in MSG-obese rats. In CTL rats, exercise increased plasma iron, in association with changes in iron transport capacity and a reduction in circulating hepcidin levels, a response that is influenced by the VN and spleen. In contrast, in the MSG-obese animals, vagal and splenic absence resulted in increased hepcidin, including following exercise, via a response that is independent of systemic iron fluctuations, suggesting disturbed hepcidin–iron homeostasis during hypothalamic obesity.

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