Increased expression of hypothalamic NADPH-diaphorase neurons in mice with iron supplement

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Abstract

Iron deficiency is known as the most important nutritional problem in the world. The loss of appetite is a common characteristic of iron deficiency. Iron-containing heme is required as a cofactor for nitric oxide synthase (NOS) that produces nitric oxide (NO). NO acts as a modulator in the control of food intake mechanism. Therefore, we examined the expression of hypothalamic NOS in various levels of dietary iron. NADPH-diaphorase histochemistry was used to detect NOS neurons, since NADPH-d-positive neurons represent the amount of NOS. ICR mice (n =30) were randomly divided into three groups based on the level of dietary iron and fed experimental diets for 4 weeks: normal-iron diet group (6 g/kg diet, n=10), low-iron diet group (2 g/kg diet, n=10) and high-iron diet group (12 g/kg diet, n=10). The expression of NOS in the paraventricular nucleus (PVN) and lateral hypothalamic area (LHA) of hypothalamus was examined by histochemistry for nicotinamide adenine dinucleotide phosphate-diaphorase (NADPH-diaphorase). High-iron diet fed mice showed significantly higher staining intensity of NADPH-diaphorase-positive neurons in the PVN and LHA compare with normal and low-iron diet fed mice. Medium-sized stained neurons and fibers were scattered over the LHA of normal and low-iron diet fed mice. In the PVN regions, the number of small-sized stained cells and fibers were less in normal and low-iron diet fed mice than
those of high-iron diet, even though those cells and fibers were more frequently observed in high-iron diet fed mice. In this manner, iron supplement in anemic condition may potentially contribute to the improvement of food intake, and a clinical correlation should be further investigated.

Reference