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Changes in Leptin, Corticotropin-Releasing Hormone and Neuropeptide Y Gene Expression Induced by Zinc Deficiency in the Rat

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Dietary zinc-deficiency induces a striking reduction and a cyclic pattern of food intake in rodents. Also, leptin and CRH are known as satiety signal and NPY as hunger signal. To elucidate the relationship between zinc deficiency and the regulation of food intake, we studied the gene expression of leptin (the *ob* gene product) in white adipose tissue and hypothalamic neuropeptides, corticotropin-releasing hormone (CRH) and neuropeptide Y (NPY) during zinc deficiency. Male Sprague-Dawley rats (n=16) weighing 180-190 g were randomly divided into Zn-adequate (30 mg Zn/kg diet), Zn-pair fed, Zn-deficiency (1 mg/kg), and Zn-sufficient (50 mg/kg), n=4 per group. After 3 wk of feeding, rats were killed. Hypothalamus in the brain and white adipose tissues (epididymal and omental) were collected for RNA isolation for RT-PCR. During zinc deficiency, leptin mRNA expression in epididymal and omental adipose tissue was down-regulated, while hypothalamic NPY gene expression was up-regulated. This finding suggests clinically potential importance, since reduced leptin level and increased NPY level is shown during zinc deficiency, yet appetite is still low. Hypothalamic CRH gene expression was up-regulated during zinc deficiency, which may contribute to the low appetite assiciated with zinc deficiency in rats. It can be cautiously interpretated from our results that low leptin and high NPY during zinc deficiency may act in attempt to restore normal food intake levels, rather than being reduced and thereby contributing to low appetite during zinc deficiency.