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Zn Effect on mRNA Expression of *ob* Gene (Leptin) and Neuropeptides (CRH, NPY) in Food Intake

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Zinc deficiency decreases food intake, acting like as satiety signal, which can cause anorexia and reduced growth. It has been proposed that zinc has been involved in food intake regulation, in somehow. Leptin, the *ob* gene product which is secreted from white adipose tissue, and neuropeptide corticotropin-releasing hormone(CRH), are also known as satiety signal, while neuropeptide Y(NPY) acts as hunger signal. We hypothesized that, on concerning both zinc deficiency and leptin/CRH as satiety signal, zinc deficiency may increase serum leptin level and, in turn, may affect on the leptin and CRH mRNA expression. Also, NPY as hunger signal would be examined.

Twenty six rats, weighing about 135.6±5.2 g, were fed with Zn-adequate(30mg/kg) for 4 weeks. Then one third(8rats) were killed as base line analysis. After then, Zn-depletion diet(1mg/kg) was fed for 2 weeks, then another one third(9 rats) were killed. For the last 2 weeks, Zn-repletion diet (50mg/kg) was fed, then the rest of rats were killed. Food intake was recorded daily and body weight weekly. Blood, various adipose tissues (epididymal, liguinal subcutaneous, abdominal, and omental), and hypothalamus were collected. Zinc in plasma, red blood cells, and mononuclear cells were measured. Serum leptin was measured using immunoassay. Leptin mRNA expression in adipose tissues and CRH/NPY mRNA expression in hypothalamus were measured by northern blots.

The growth rates were similar among the three different zinc dietary group during the entire experimental period. Food intake was also similar among the groups(18.6±SD1.6g~19.7±SD1.4g). Plasma zinc level was not significantly different among three zinc level, however, the pattern showed that it was decreased with Zn-depletion diet and again increased back with Zn-repletion. Zinc level in red blood cells ($p<0.05$) and mononuclear cells ($p<0.001$) decreased with feeding Zn-depletion diet, and increased back with Zn repletion diet. Serum leptin level in Zn-adequate diet (880.9±SD311.5pg/ml) was increased with feeding Zn-depletion diet(1236.0±SD323.9pg/ml), and decreased back with feeding Zn-repletion diet(998.3±SD471.4pg/ml)($p<0.05$). The pattern of serum leptin and dietary zinc level was consistent as acting satiety signal. However, leptin mRNA expression in various adipose tissue didn't show the consistent pattern with dietary zinc level. mRNA expression of CRH and NPY would be examined. However, not consistent trend was not shown at this study. Further study for mRNA expression of leptin in various adipose tissues and of neuropeptide in hypothalamus with different zinc level will be needed for investigation of relation among zinc, leptin, and neuropeptides in food intake.