Interaction Between Dietary Carbohydrate and Copper Nutriture on Lipid Peroxidation in Rat Tissues

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ABSTRACT

The effects of the interactions between dietary carbohydrates and copper deficiency on superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) activities and their roles in peroxidative pathways were investigated. Weanling rats were fed diets deficient in copper and containing either 62% starch, fructose, or glucose. Decreased activity of SOD was noted in all rats fed the copper-deficient diets regardless of the nature of dietary carbohydrate. However, the decreased activity was more pronounced in rats fed fructose. Feeding the fructose diets decreased the activity of GSH-Px by 25 and 50% in the copper-supplemented and copper-deficient rats, respectively, compared to enzyme activities in rats fed similar diets containing either starch or glucose. The decreased SOD and GSH-Px activities in rats fed the fructose diet deficient in copper were associated with increased tissue peroxidation and decreased hepatic adenosine triphosphate (ATP). When the fructose in the diet of copper-deficient rats was replaced with either starch or glucose, tissue SOD and GSH-Px activities were increased and these increases in enzyme activity were associated with a tendency toward reduced mitochondrial peroxidation when compared to the corre-

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sponding values for rats fed fructose throughout the experiment. Dietary fructose aggravated the symptoms associated with copper deficiency, but starch or glucose ameliorated them. The protective effects were more pronounced with starch than with glucose.

**Index Entries:** Copper-deficient rats, and carbohydrate and lipid peroxidation; corn starch, and copper and lipid peroxidation; fructose, and copper and lipid peroxidation; glucose, and copper and lipid peroxidation; copper deficiency, and ceruloplasmin activity; copper deficiency, and copper superoxide dismutase; copper deficiency, and glutathione peroxidase; copper deficiency, and lipid peroxidation.

**INTRODUCTION**

Dietary copper deficiency in rats results in the reduction of superoxide dismutase (SOD) activity in several tissues (1-7). SOD is involved in the prevention of tissue peroxidation (8) and its reduction has been associated with enhancement of cell damage (6,9). Copper is utilized in peroxidation pathways that are closely interrelated to those utilized by selenium (7). Recently it has been reported that feeding rats diets based on evaporated milk deficient in copper decreased the activities of liver SOD and the selenoenzyme, glutathione peroxidase (GSH-Px) (10). In contrast, copper deficiency in diets sufficient in all essential nutrients, including selenium, did not have any effect on tissue GSH-Px in the rat (7,11). In another study (12), signs of copper deficiency in rats did not always correlate with the changes in the activity of GSH-Px.

Recently, we have demonstrated that the severity of copper deficiency can be enhanced by feeding rats diets containing fructose as compared to starch or glucose (13,14). However, as the diets in that study were not supplemented with selenium, the increased severity of copper deficiency might have been caused by lack of dietary selenium, which in turn could allow increased lipid peroxidation of tissues (6). The first purpose of the present study was to determine whether copper-deficient diets containing different types of carbohydrates, adequate in all essential nutrients including selenium, could differentially affect selenium status and consequently the activity of selenium-dependent enzymes. The changes in the activities of tissue SOD and/or GSH-Px caused by altered copper and selenium status could impair lipid peroxidation of tissue membranes and in turn lower the ATP level of mitochondria.

The use of fructose in diets of the Western world may well become a problem when the food industry increases the use of high-fructose corn sweeteners. It is possible that the same type of interaction between fructose and copper status in which the severity of copper deficiency is more pronounced by feeding rats diets containing fructose compared to starch or glucose (13,14), also exists in humans. The second purpose of the present study was to determine whether the severity of the metabolic and cellular lesions caused by copper deficiency could be reversed by