Selenium Concentration and Glutathione Peroxidase Activity in Selenium and Magnesium Deficient Rats

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ABSTRACT

To clarify the effects of selenium (Se) and magnesium (Mg) deficiencies on Se and glutathione peroxidase (GSHPx) status, weanling male Wistar rats weighing 50–60 g were placed on four kinds of diets divided by two levels of Se (0.5 or 0.019 mg/kg) and Mg (500 or 50 mg/kg) for 8 wk. Magnesium deficiency had an influence on distribution of Se, which was increased in muscle and decreased in other tissues. The changes in GSHPx matched those in Se. The levels of Se and GSHPx in most tissues were lower in Se-Mg-deficient rats than in Se-deficient rats. Thus, selenium and Mg deficiencies would make oxidant lesion more serious than Se deficiency.

Index Entries: Selenium, GSHPx, Se-Mg-deficiency, rat.

INTRODUCTION

Selenium (Se) deficiency is associated with Keshan disease, which is endemic cardiomyopathy, that occured in China. Its mechanism is unclear although Se supplementation could prevent it (1). The depletion in antioxidant ability may contribute to the disease because Se-dependent glutathione peroxidase (GSHPx, EC 1.11.1.9) activity decreases in Se deficiency (2,3). However, Se deficiency alone cannot explain the cause

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of Keshan disease. Magnesium (Mg) acts as a metallic cofactor in more than 300 enzymatic reactions; notably it is essential for all reactions requiring ATP (4). Magnesium also functions as a transmembrane and intracellular modulator of other ions. Altered Mg homeostasis, particularly deficiency, can cause alterations in metabolic functions that result in clinically recognizable events, such as heart disease. A report has shown a possible role for free radical participation in Mg deficiency (5). However, little information is available on the relationship between Se and Mg. In the present study, we examined the effects of Se and Mg deficiency on Se and GSHPx status in rats.

MATERIALS AND METHODS

Animals and Diets

Twenty-four weanling male Wistar rats (Oriental Bioservice, Kitayama Labesu Co., Kyoto, Japan), weighing 50-60 g were randomly and equally divided into four groups and fed diets containing adequate or deficient levels of Se (0.5 or 0.019 mg Se/kg of diet) and of Mg (500 or 50 mg Mg/kg of diet), but adequate levels of all other nutrients for 8 wk. The complete dietary composition is shown in Table 1. All rats were maintained in stainless steel cages with a raised wire bottom. The temperature was maintained between 23–24°C. Diet and deionized double-distilled water were given ad libitum. Diet intake and body wt were measured twice a week. Between 2 and 3 wk after starting, two Se-Mg-deficient rats and two Mg-deficient rats died.

Sample Collection and Preparation

After 8 wk rats were anesthetized with sodium pentobarbital, and blood was drawn from the abdominal aorta into heparinized syringes and centrifuged at 1000g for 15 min to obtain the plasma. Other tissues were removed and stored at −83°C together with plasma before analysis. Whole blood was taken for digestion before centrifuging.

Biochemical Analysis

The frozen tissues were placed in an ice-cold 0.1 mol/L potassium phosphate buffer, pH 7.4, and homogenized. The homogenate was centrifuged (Model 65P, RP-40 rotor, Hitachi Koki Co., Ltd., Tokyo, Japan) at 105,000g for 60 min, and supernatant was used to determine GSHPx activity by the coupled method described by Lawrence and Burk (8). The reaction mixture consisted of 50 mmol/L potassium phosphate buffer (pH 7.0), 1.0 mmol/L EDTA, 1.0 mmol/L NaN3, 0.2 mmol/L NADPH, 1.0 EU/mL GSSG-reductase, and 1.0 mmol/L GSH. Reaction mixture was mixed fresh at the beginning of each day. An enzyme source (0.1 mL) was