Studies on Metabolism and Physiological Effect of Selenium

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Selenium, a part of glutathione peroxidase and thus an essential trace element, possesses a protective effect against oxidative stress by catalyzing the destruction of \( \text{H}_2\text{O}_2 \) or lipid hydroperoxides. Selenium deficiency or toxicosis as a physiopathological effect will occur if this trace element in the body is either deficient or excessive, so the safe range of the physiological selenium level may be extremely narrow. However, there has been little systematic investigation on the physiological effects of selenium on the borderlines between deficient and nutritional levels and between nutritional and toxic levels.

The present study was carried out to elucidate the relationships between metabolic behavior in excretion and physiological effect on the borderline between physiopathological and nutritionally physiological-selenium levels.

The effects of feeding several different levels of dietary selenium such as deficient (0 mgSe/kg Torula yeast-based Se-deficient diet), nutritional (0.31 mgSe/kg diet), sub-toxic (1.25 mgSe/kg diet) and toxic (5.00 mgSe/kg diet) levels were investigated with SD male rats. The amount of respiratory selenium ((\( \text{CH}_3 \))\(_2\)Se:DMSe) and urinary selenium ((\( \text{CH}_3 \))\(_3\)Se\(^+\):TMSe), and the selenium content and activity of glutathione peroxidase (GSH-Px) in various rat tissues were particularly determined. The dietary selenium intakes during 30 d at deficient, nutritional, sub-toxic and toxic levels were 0.42, 6.3, 26 and 95 \( \mu \)gSe/rat/d, respectively. Daily fecal and urinary excretion of selenium showed a high plateau in accordance with the amount of dietary selenium intake, except for the decline of urinary excretion of total selenium when administered at a deficient level. Urinary TMSe was not detected more than 10 d after administration of deficient and nutritional levels, but at sub-toxic and toxic levels the TMSe levels in urine were constant in accord with these selenium intakes. Respiratory DMSe was not detected at deficient or nutritional levels, at sub-toxic level it decreased daily and at toxic level the high amount of DMSe was excreted in respiration.

The activities of GSH-Px in tissues were lowered with decrease in the selenium content between deficient and nutritional levels. This phenomenon was confirmed by Sephadex G-150 chromatograms of liver cytosol from rats 30 d after administration of different dietary selenium levels. The depression of GSH-Px activity in tissues at sub-toxic and toxic levels, on the other hand may be caused by selenium greatly exceeding the level of nutritionally physiological-selenium.

Results of experiments with rats fed on Se-deficient diet for 75 d revealed, a linear relationship between the selenium content of liver, kidney or heart and GSH-Px activity. GSH-Px activity in the liver, which is measured by \( \text{H}_2\text{O}_2 \) as substrate and is Se-dependent, disappeared when a Se-deficient diet was offered; this may induce a resultant additional GSH-Px measured by cumen hydroperoxide as substrate which is not Se-dependent and is the same as glutathione-S-transferase.