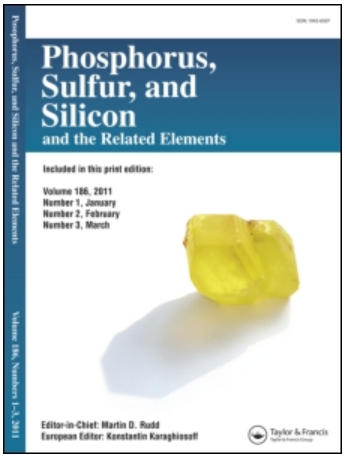

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EFFECT OF SELENIUM DEFICIENCY ON URINARY KETONE BODY EXCRETION IN STARVED RATS

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Abstract Rats were fed a Se-deficient (Se content: 0.011 μg/g) or a Se-adequate diet (the basal diet supplemented with 0.1 μg Se/g as sodium selenite). On the 22nd week of the feeding period, the rats were starved for 72 h and the urinary excretion of ketone bodies (acetoacetate and 3-hydroxybutyrate), urea, and creatinine were examined. The urinary excretion of ketone bodies were markedly higher in the Se-deficient rats than in the Se-adequate rats 24 h after the start of starvation, while the excretion of urea and creatinine were similar in the Se-deficient and Se-adequate rats. Se status did not effect on the plasma urea and creatinine contents as well as urinary *N*-acetyl-β-D-glucosaminidase activity; renal function was not impaired by Se deficiency. These results indicate that Se deficiency causes an increase of urinary ketone body excretion in starved rats and that the increase is ketone-specific with no changes in major urinary profiles.

INTRODUCTION

Selenium (Se) is an essential constituent of glutathione peroxidase (GSHPx) and several metabolic impairments caused by Se deficiency have been explained in connection with this enzyme. In recent years, however, several metabolic effects of Se not

associated with GSHPx have been recognized. Olsson showed, in his first report, increased urinary excretion of ketone bodies (acetoacetate (AcAc) and 3-hydroxybutyrate (3-OHBA)) in starved Se-deficient rats[1]. Subsequently, he observed that excretion of other major urinary components (sodium, potassium, urea, ammonium, and creatinine) was also increased in starved Se-deficient rats and he assumed that the increased ketone body excretion was not a ketone-specific change but was a side effect of a more basic metabolic impairment[2]. Although these findings are not fully explained by the present knowledge of Se nutrition, no affirmative re-examination has been performed by other researchers. Then author re-evaluated the effect of Se deficiency on excretion of urinary components including ketone bodies.

MATERIALS AND METHODS

Male weanling Wistar rats, weighing 50 to 60 g, were fed a *Torula*-yeast based diet (referred to as the basal diet) and the same diet supplemented with 0.1 μg/g of Se as sodium selenite. Analysis showed the basal diet to contain 0.011 μg/g of Se. In the present report, rats fed the basal diet are referred as Se-deficient rats and rats fed the Se-supplemented diet are referred as Se-adequate rats.

On the 22nd week, the rats in both groups were starved for 72 h and their urinary ketone bodies were measured enzymatically[3] for one day prior to three days following the start of starvation. Concurrently, blood and livers were collected from the rats without starvation and the GSHPx activity[4] and Se

content[5] were measured to assess the Se status. Urea and creatinine in the urine and plasma as well as urinary *N*-acetyl-β-D-glucosaminidase (NAG) activity were also determined by the use of commercial kits.

RESULTS AND DISCUSSION

During the whole experimental period, no difference was observed in animal growth, irrespective of the dietary Se supplementation; body weights (means±SD for 5 rats randomly selected from each group) of the Se-adequate rats and Se-deficient rats on the 22nd week were 459±22 g and 451±22 g, respectively. On the 22nd week, Se content and GSHPx activity in plasma, erythrocytes and livers of the Se-deficient rats were 4% to 17% of the values for the Se-adequate rats; a low Se status was completed by the feeding with the Se-deficient diet for 22 weeks.

Table I Effect of Se status on urinary ketone body excretion in starved rats

Hours after starvation	Acetoacetate +Se	Acetoacetate -Se	3-Hydroxybutyrate +Se	3-Hydroxybutyrate -Se
0	1.4±0.8	1.2±0.5	0.8±0.2	0.9±0.4
0-24	0.9±0.5	0.5±0.2	2.5±0.5	3.8±0.2
24-48	2.2±0.7	9.6±1.4***	2.2±0.3	25.1±8.4***
48-72	4.9±1.8	14.6±2.5***	1.9±0.8	50.0±21.1***

Values (means±SEM (n=5)) are expressed as μmol/24 h.
***, $p<0.001$

Table I shows the comparison of urinary AcAc and 3-OHBA excretion in the Se-adequate and Se-deficient rats. A significantly higher urinary excretion of these ketone bodies was observed in the Se-deficient rats than in the Se-adequate rats 24 h after the start of starvation. The increase of ketone body excretion was observed even in rats fed the Se-deficient diet for only 4 weeks (data not shown). These results are similar to the Olsson's first report[1]; increased urinary ketone body excretion undoubtedly occurred in starved Se-deficient rats.

Table II Urinary excretion of urea and creatinine

Hours after starvation	Urea (mmol/24 h)		Creatinine (μmol/24 h)	
	+Se	-Se	+Se	-Se
0	4.1±0.4^{c}	3.7±0.2^{b}	162±9^{b}	157±5^{b}
0-24	2.7±0.3^{b}	2.0±0.3^{a}	166±18^{b}	153±11^{b}
24-48	1.9±0.2^{ab}	2.1±0.2^{a}	173±17^{b}	160±7^{b}
48-72	1.0±0.1^{a}	1.2±0.1^{a}	134±8^{a}	126±8^{a}

Values are means±SEM (n=5). Means in the same column not sharing a common superscript differ significantly ($p<0.05$). The difference between "+Se" and "-Se" was not significant ($p>0.05$) for all items.

Table II shows the urinary excretion of urea and creatinine in rats fed the Se-adequate or Se-deficient diets for 22 weeks. Lowered urinary excretion of urea and creatinine, due to starvation, was observed. However, no significant differences in urea and creatinine excretion were observed between the

Se-adequate and Se-deficient rats. In addition, urine volume and ammonium excretion were also similar in the both groups (data not shown).

Olsson showed in his second report[2] that increases in urinary ketone bodies occurred with increases in urine volume and urinary excretion of urea and creatinine in Se-deficient rats; he inferred that the increase of urinary ketone bodies resulted from an impairment in filtration or reabsorption in the kidney. However, in the present experiment, the author observed that the Se status of the rats did not cause any changes in urine volume and urinary nitrogen excretion, irrespective of whether rats had been starved or not. These present results indicate that the increase of urinary ketone bodies due to Se deficiency is a ketone-specific change.

Table III Plasma urea and creatinine contents and urinary NAG activity

Items	+Se	-Se
Plasma urea (μmol/ml)	1.7±0.1	1.9±0.1
Plasma creatinine (nmol/ml)	83±3	85±4
Urinary NAG (munit/24 h)	379±22	371±41

Values are means±SEM (n=5). The differnce between "+Se" and "-Se" was not significant ($p>0.05$) for all items.

Table III shows plasma urea and creatinine contents as well as urinary NAG activity which were clinically used as indices for renal function. Difference was not observed between Se-deficient and Se-adequate rats in these indices. Thus, renal function was not

impaired by Se deficiency in the present experimental condition.

In Olsson's Se-deficient rats the activity of hepatic GSHPx was lowered to 3% of that in the Se-adequate controls[2], whereas in the present study hepatic GSHPx activity in Se-deficient rats was 11% of that in the controls; Se deficiency was more severe in Olsson's rats than the author's. The difference between the present results and Olsson's for urine volume and urinary nitrogen excretion may be associated with the difference in the extent of Se deficiency; a severe Se deficiency may cause changes in the major urinary profiles. However, the present results indicated that the increased urinary ketone body excretion occurred at an early stage of Se deficiency and was independent of the increases in urine volume and urinary nitrogen excretion. In conclusion, the increased urinary ketone body excretion is not a side effect of a renal dysfunction but is a primary effect of Se deficiency.

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