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Effect of Methionine Supplementation on Glutathione Peroxidase Activity in Young and Old Murine Tissues

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Abstract

The effect of methionine (Met) supplementation on glutathione peroxidase (GSHPx) activity in young and 14 month-old rat and mice was investigated. GSHPx activity was more enhanced by methionine supplementation in young rats when selenium (Se) was given as selenite than given in the form of selenomethione (Se-Met). However, GSHPx activity was not influenced by Met supplementation in the old rats. When diets were low in Se, the biopotency of the enzyme by Met was facilitated. No significant differences in GSHPx activity was observed with Met supplement in growing mice when Met was given 0.3% and 0.8% in the diet at high levels of Se (2ppm). The peak GSHPx in liver and kidney occurred at day 18, thereafter it decreased. Particularly, the liver GSHPx at day 18 increased 4.2 times than that at day 4 by 0.5% Met supplementation, while the unsupplemented group remained only 2.5 times increase. It is considered that in some tissues Met requirement may be met by Se-Met when rats were fed a diet suboptimal in Met. In addition, at lower levels of Se the utilization of Se is more enhanced by Met than at higher levels of dietary Se. Therefore, GSHPx activity may be influenced greatly by Met status along with dietary Se.

Key words : selenite, selenomethionine, glutathione peroxidase activity, biopotency

INTRODUCTION

With the discovery that Se is an integral part of the enzyme GSHPx, linear correlations of erythrocyte or whole blood GSHPx activity with blood Se concentrations have been demonstrated for sheep and cattle[1,2], swine[3], horses[4] and rats[5].

The activity of the enzyme GSHPx significantly decreases in the absence of dietary Se and the activity is dose-related to the dietary level of Se. Omaye and Tappel[6] observed that the specific activity of the enzyme increases as a logarithmic function of the dietary Se level.

Se controls the appearance of the GSHPx in the cytosol and mitochondrial matrix space during the ribosomal protein synthesis. Cytosolic Se concentrations may have a role in GSHPx protein synthesis. Intracellular Se may be bound to proteins and intracellular storage sites such as mitochondria or microsomes.

Strong correlations between blood selenium and GHSPx activity have been observed in individuals consuming low levels of Se[7,8], but Schmidt and Heller[9] and Schrauzer and White[10] have reported a lack of correlation between blood GSHPx activity and blood levels of Se in individuals consuming adequate or high levels of Se. This is most likely due to the efficient incorporation of selenomethionine into blood proteins. In addition, blood Se and GSHPx were not correlated in the blood of pregnant women[11,12], indicating that other factors may be involved in the relationship of Se and GSHPx activity.

When selenite is supplied to mammals, Se can be recovered as GSHPx[13-15], dimethylselenide or the trimethylselenonium ion[16]. This assimilation of inorganic selenite into these organic forms represents a unique reduction by the animal cell of an inorganic ion. Other inorganic ions such as nitrate and sulfate are generally considered not to be reduced but rather oxidized by the animal cell.

The study of the biological potency of various Se compounds that provide Se for GSHPx synthesis provides practical information on the bioavailability of various dietary forms of Se. The biopotency of selenite, selenomethionine, and Se-cystine for GSHPx sy-

nthesis *in vivo* has been reported to be generally similar when nutritionally adequate levels of Se are fed. Pierce and Tappel[17] found that a single large dose of selenite or selenomethionine (300μg Se/90g rat) given to Se deficient rats resulted in similar increases in liver, kidney, small intestine, and stomach GSHPx activity within 48 hours of Se administration. The retention of Se in major animal tissues revealed that it was much higher when given as Se-Met than in an inorganic form as selenite or selenate[18-21]. Se bioavailability studies in humans using Se-Met, showed that approximately half of the dietary intake of Se was excreted in urine, and the remainder was excreted in the feces. Tracer studies using ^{75}Se-Met showed that 80% of the dietary intake of Se was absorbed, and suggested that food Se is highly bioavailable where the Se content of soil is low.

Many Se compounds have been isolated from plants including Se-methylselenome thionine, selenohomocysteine, selenocystine, selenite, selenate, seleninic acid, dimethyl selenide, dimethyl diselenide, and selenocystathionine. Selenomethionine was found to be one of the major Se compounds in seeds or forages consumed by livestock. The existence of different levels of Met in the diet not only affects the bioavailabilily of Se compounds *in vivo* but also influences the activity of GSHPx. Therefore in this study, the biopotency of the two major selenium compounds, selenite and Se-Met on GSHPx activity was compared in different growing stages of rats and mice.

MATERIALS AND METHODS

Animals

Sprague-Dawley rats and ICR Swiss mice were used in this sudy, maintaining under standardized laboratory conditions with the lights on from 06 : 00 to 18 : 00 and were fed low Se diet and distilled water *ad libitum*. At the end of the experiments they were anesthetized by ether and sacrificed by cervical disiocation. Blood was collected by heart puncture and major selenium pool organs including liver, kidney and spleen were taken, rinsed with saline solution and kept frozen until analysis.

Composition of Se-deficient diet

Torula yeast based Se-deficient diet was fed for approximately three weeks to deplete Se stores in the body before the start of each experiment. Composition of Se-deficient diet is shown in Table 1. Cod liver oil and sucrose were added as a source of essential fatty acids and a pure source of carbohydrate and energy, respectively.

Experiment 1

Rats were depleted of Se by feeding Se deficient diet prior to the experimeant. 0.5% Met was supplemented to the diet containing 0.05, 0.1, 0.2, 0.5 and 1.0 ppm Se either in the form of Se-Met or selenite.

Experiment 2

In order to further examine the effect of Met on GSHPx activity in old rats comparing to the previous experiment used growing rats 14 month-old rats were used. Animals were fed with 0.3 and 0.8% Met in the diet at 0. 15, 1.0 and 2.0ppm Se levels.

Experiment 3

Growing mice were maintained on a 2ppm Se in the diet for 80 days. One group was supplemented with 0.5% Met and the other remained unsupplementation, while the basal diet contained 0.3% Met. GSHPx activity was measured at 4, 10, 18, 28, 42 and 80days after feeding experimental diet containing 2ppm Se.

Measurement of glutathione peroxidase activity and protein determination

GSHPx activity in tissues and blood samples were

Table 1. Composition of Se-deficient diet

Torula yeast	30%
Mineral mixture (No Se)	5%
Tocopherol stripped lard	5%
Cod liver oil	3%
Vitamin mixture	1%
Sucrose	56%
$ZnSO_4 \cdot H_2O$	700mg/kg

AIN-76 vitamin mixture and AIN-76 mineral mixtures were used. Zinc sulfate was added to mineral mixture
Se was added as either in the form of selenite or Se-Met to a Se-deficient diet

measured by a modification of the method of Paglia and Valentine[22] as described by Stults *et al.*[15] with 0.25mM H_2O_2 as substrate. Assays were performed in 0.1M phosphate buffer, pH 7.0, containing 2.0mM GSH, 0.2mM NADPH, 1.0unit/ml GSH reductase, 1.0mM NaN_3, and 3.0mM EDTA. One unit of GSHPx activity is defined as the amount of enzyme required to oxidize 1μmole NADPH per minute.

Protein determinations were performed by the protein-dye binding method[23] using Coomassie brilliant blue G-250.

Statistical analysis

Data are expressed as mean and standard error of the mean. Statistical differences were examined using Student's *t*-test

RESULTS

Table 2 and 3 illustrate the increase of GSHPx activity in growing rats by 0.5% methionine supplementation in the diet. GSHPx activity at a lower level of Se tended to be enhanced more by methionine supplementation than that at a higher level of Se. The enzyme activity was almost doubled by methionine supplementation at 0.1ppm Se as selenite, while it increased 75.9% when Se was given as Se-Met. Overall, the biopotency of selenium was enhanced by methionine in the group fed selenite than Se-Met.

Fig. 1 shows the GSHPx activity of 14 month-old Sprague Dawley rats with 0.3% methionine supplementation at different Se levels. Enzyme activity was not enhanced by methionine supplementation in 14 month-old rats. Even at a 2ppm level of Se, enzyme activity was still increasing at 2ppm Se level, which was considered a toxic level to young rats.

Similar results were observed with the human studies by Thomson *et al.*[7] with New Zealand residents. They demonstrated that increases in Se concentrations in whole blood, erythrocytes, and plasma were greater after supplementing Se-Met-Se than after selenits-Se was consumed. Furthermore, Se concentrations tended to plateau after selenite-Se, while after Se-Met-Se they continued to rise as long as doing continued. This may be due to the fact that Se utilization is enhanced by Met through the protein synthesis including the synthesis of enzyme GSHPx.

Growing mice were fed 2ppm Se with 0.3% and 0.8% methionine supplementation and GSHPx enzyme activity was measured at different periods of the experiment (Table 4 and 5). No significant differences in the enzyme activity was shown between groups. The highest enzyme activity in both liver and kidney occurred at 18 days in both groups of mice. Particularly, the liver GSHPx at day 18 increased 4.2 times than that at day 4 by 0.5% Met supplementation,

Table 2. Increase of plasma GSHPx activity by methionine supplementation at various levels of dietary selenium in young rats

	Dietary selenium level (ppm)				
	0.05	0.1	0.2	0.5	1.0
Se-Met + No Met	3.2±0.21	5.8±0.62	13.9±0.78	24.5±2.24	27.2±3.21
Se-Met + 0.5% Met	3.8±0.15	10.2±0.35*	23.2±1.72*	27.2±3.26	30.6±2.15
Selenite + No Met	7.1±0.05	12.8±0.82	18.3±1.85	22.6±1.75	20.1±3.28
Selenite + 0.5% Met	5.2±0.22	23.7±2.12*	33.5±3.25*	37.2±4.25*	29.2±3.22

*Statistically significant at $p<0.05$ relative to Met unsupplemented control
A unit of enzyme activity is one μmole NADPH oxidized per minute per g plasma protein
Data represents mean±SEM

Table 3. Increase of GSHPx activity by methionine supplementation*

	0.05ppm Se	0.1ppm Se	0.2ppm Se	0.5ppm Se	1.0ppm Se
Se-Met	18.8%	75.9%	66.9%	11.0%	12.5%
Selenite	−26.8%	85.2%	83.1%	64.6%	45.3%

* The increment of the enzyme activity was calculated dividing the Met supplemented into the Met unsupplemented control, then the figure was expressed as percentage

while the unsupplemented remained only 2.5 times increased.

Enzyme activity showed a decrease after 18 days. Between 18 to 42 days, the enzyme activity decreased by half in both tissues.

The biopotency of Se compounds in growing animals increased by Met supplementation particularly when low Se was fed, suggesting that at lower levels of Se the utilization of Se is more enhanced by methionine than that at higher levels of dietary Se.

GSHPx enzyme activity was not changed by methionine supplementation in mature rats and growing mice. However, in mature rats as the Se level increases the GSHPx activity increased even at 2ppm Se supplementation in the diet. This may also be due to the fact that Met may be involved in Se detoxification *in vivo* and thus increasing the utilization of Se.

DISCUSSION

Of the various body compartments studied, particularly rat liver and plasma, GSHPx activity shows a

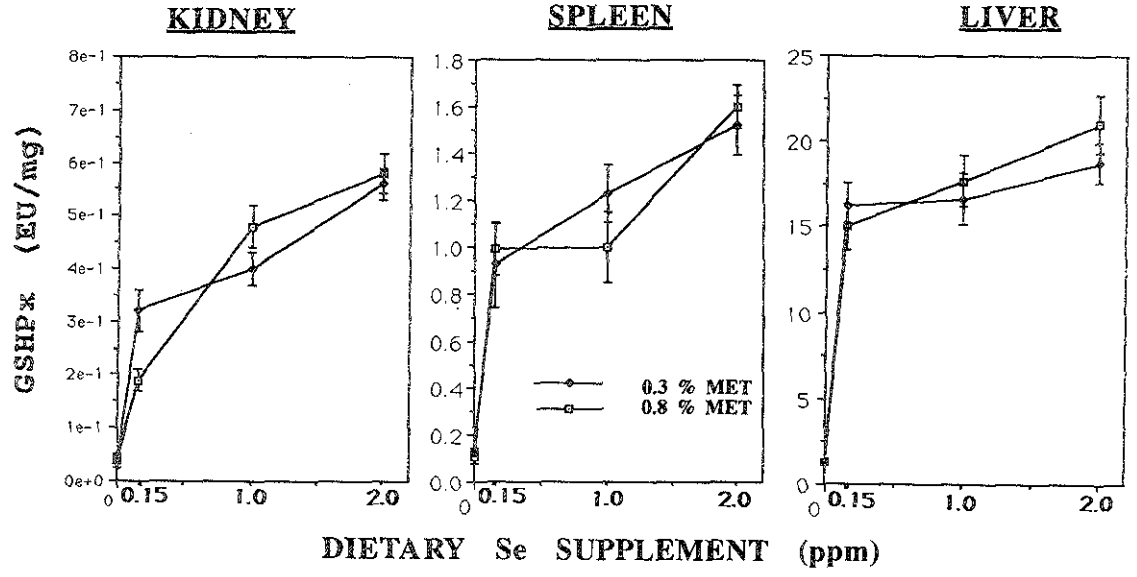

Fig. 1. GSHPx enzyme activity of 14 months-old Sprague Dawley rats in kidney, spleen and liver given methionine and selenium supplements.

Table 4. Liver glutathione peroxidase activity of methionine supplemented mice

	Days	Dietary methionine levels	
		0.3% Met (unsupplemented)	0.8% Met (0.5% Met supplemented)
GSH Peroxidase (EU/mg)	4	6.05±0.42	4.02±0.28
	10	8.72±0.61	8.48±0.34
	18	15.05±0.87	16.78±0.66
	28	12.02±0.37	12.85±0.58
	42	7.28±0.58	7.35±0.77
	80	8.81±0.46	9.21±0.84

Dietary Se level was 2ppm, fed to mice for 80days
A unit of enzyme activity is one μmole NADPH oxidized per minute
Data represents mean±SEM

Table 5. Kidney glutathione peroxidase activity of methionine supplemented mice

	Days	Dietary methionine levels	
		0.3% Met (unsupplemented)	0.8% Met (0.5% Met supplemented)
GSH Peroxidase (EU/mg protein)	4	1.20±0.26	1.23±0.34
	10	2.24±0.46	2.00±0.23
	18	3.96±0.38	3.85±0.49
	28	2.78±0.48	3.00±0.37
	42	1.60±0.52	1.78±0.47
	80	1.98±0.45	1.80±0.29

Dietary Se level was 2ppm, fed to mice for 80days
A unit of enzyme activity is one μmole NADPH oxidized per minute
Data represents mean±SEM

rapid decrease during Se depletion and a similarly rapid increase during Se repletion[24]. Oh *et al.*[25] have reported that tissue GSHPx activity plateaus at approximately 0.1ppm dietary Se for all tissues except erythrocytes and pancreas. The leveling off of GSHPx activity with increasing Se supplementation suggests that tissue GSHPx activity may be a better indication of effective Se status than tissus Se content.

In spite of the fact that the enzyme activity was almost doubled by Met supplementation at low levels of Se, Sunde *et al.*[26] claimed that dietary Met has no effect on the biopotency of GSHPx activity.

The enhanced GSHPx activity by methionine supplementation in group fed selenite rather than Se-Met indicates that Met requirement may be met by Se-Met in some tissues, particularly when rats were fed a diet suboptimal in methionine.

Selenite and selenomethionine have been reported to have equal bioptency in chicks[5]. However, when the diet of chicks was supplemented with Se at less than 0.1ppm for seven days, selenite was twice as potent as selenomethionine[27].

At low dietary levels of Se supplementation (less than 0.06ppm), selenite was generally more effective than selenomethionine for the synthesis of GSHPx. The biopotency of selenomethionine for GSHPx was reduced when suboptimal levels of methionine were fed to rats, whereas selenite biopotency was not affected.

Therefore, the relative biopotency of selenomethionine for GSHPx can vary according to the dietary methionine status of subjects.

However, selenomethionine was shown to be four times as biopotent as selenite for the prevention of pancreatic degeneration of chicks. Hawkes *et al.*[28] reported that selenomethionine was more effective than selenite in providing Se for GSHPx synthesis in liver slices *in vitro*. In contrast, Sunde and Hoekstra[29] have shown that selenite and selenide are more readily metabolized than selenocystine to a form of Se that can be incorporated into GSHPx.

Metabolic data suggest that when diets are low in Se and suboptimal in Met, GSHPx synthesis is facilitated by Met supplementation.

This may be due to a more efficient utilization of Se by Met for the synthesis of GSHPx. A Chinese study indicated that RBC GSHPx activity of people in low Se areas increased by Met supplementation. Similar results have been obtained in chicks[30]. These metabolic studies add interest to the experimental observations that Met may be involved in the synthesis of Se-Cys catalytic site of GSHPx.

Selenite Se can be metabolized and reduced to dimethyl selenide or trimethyl selenonium ions in mammals[16]. This assimilation of inorganic selenite into these organic molecules represents a unique reduction of Se by the animal of an inorganic ion.

The reductive pathway proposed by Esaki *et al.*[31] suggests that selenomethionine is incorporated into selenocysteine of GSHPx in pathways analogous to sulfur metabolism. Selenomethionine is not normally synthesized from either selenite or selenate in animals, for animals fed Se-Met accumulate Se in tissues compared to animals being fed selenite.

The increase of GSHPx activity with Met Supplementation, suggests that there may be a methionine pathway for the efficient utilization of Se, especially when Se is suboptimal as observed in the enhanced GSHPx synthesis in either man or animals. Methionine metabolism is dependent upon hormones, enzymes and tissues. For example, Finkelstein *et al.*[32] reported that N_5-Methyltetra-hydrofolate-homocysteine methyltransferase activity contributes significantly to the regulation of methionine metabolism in mammals.

L-methionine is incorporated into proteins, acts as the methyl group donor via S-adenosyl methionine, is a precursor for cysteine, cystine and taurine and participates in the transamination process. The wide variation in the biological utilization of sulfur containing amino acids for growth of various animal species may be related to the relative susceptabilities of D-amino acids to oxidation to the corresponding keto acids by specific and nonspecific D-amino acid oxidases.

Although L-cystine is not an essential amino acid for rodents, less methionine is needed for growth in the presence of some L-cystine in the diet[33]. The mechanism of this so-called "sparing effect" is not well understood.

Selenomethionine follows the metabolic pathways of intact methionine, and thus when methionine is

limiting, selenomethionine will be incorporated into body proteins in place of methionine, where Se will be unavailable for GSHPx synthesis until these proteins turn over.

A recent study[34] using ^{75}Se-selenite and Se-Met indicates that a fraction of selenomethionine is deposited directly and nonspecifically into tissue proteins in place of methionine. The fact that selenomethionine biopotency is impaired when dietary methionine is limiting may also be an indirect evidence that methionine may contribute a portion of carbon source, which may then increase the activity of the enzyme GSHPx.

Evidence has also suggested that selenocysteine is synthesized from selenomethionine. Easki *et al.*[31] reported that the pathway of selenomethionine has a similar pathway as methionine leading to selenocysteine. This evidence suggests that Se in the form of selenomethionine can be converted to selenocystathionine then to selenocysteine for the immediately available form of GSHPx synthesis.

From the observations in this study, the enhancement of GSHPx activity by Met is apparently greater in the young animal than the old at low levels of Se than at high levels of Se. The recognition that methionine contributes to the endogenous synthesis of GSHPx when Se is limiting represents a discovery of a previously unrecognized function of Met and awaits further elucidation for the specific metabolism involved.

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성장기의 쥐와 늙은 쥐 조직의 Glutathione Peroxidase 활성에 대한 Methionine 투여의 효과

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요 약

자라나는 과정의 젊은 흰쥐와 14개월 된 늙은 흰쥐, 생쥐들을 대상으로 Met 투여에 따른 간, 혈장, 신장, 비장 등의 glutathione peroxidase (GSHPx) 효소 변화를 측정 비교하였다. 성장기의 쥐에 있어서 Se을 Selenite 형태로 투여했을 때 0.5%의 식이 Met 공급은 같은 Se을 Se-Met 형태로 공급했을 때 보다 효소 활성이 증가하였다. 그러나, 14개월 된 늙은 쥐에서는 Met 투여에 대한 효소 활성은 큰 변화가 없었다. 일반적으로 식이 Se 증가는 효소 활성과 거의 정비례하나 1ppm Se 수준 이상에서는 완만하게 증가 또는 거의 정체하는 상태를 보이지만, 늙은 쥐에서는 Se의 식이 수준이 2ppm이 되어도 여전히 효소 활성이 증가하는 경향을 보였다. 성장기의 생쥐 실험에서도 2ppm Se 수준에서의 0.3%와 0.8%의 식이 Met 투여는 효소 활성에 큰 영향이 없었다. 그러나 실험식이 투여 18일 경엔 4일째 보다 Met 투여군이 4.2배의 증가를 보인 반면 비투여군은 2.5배의 증가에 머물렀다. 18일경 이후 효소 활성이 감소하여 42일째는 반으로 감소함을 보였다. Se의 생체 활성은 특히 식이 Se 수준이 낮을 때 Met에 의해 증가되는 폭이 컸으며 Se 수준이 높을수록 상대적으로 감소함을 보였다. 위의 제반 결과에서 Met의 요구량이 Se-Met에 의해 대체되고 있음을 짐작할 수 있다. 그러므로 식이 Met 수준 상태에 따라 효소의 활성이 달라질 수 있다는 것을 시사한다.