Acta vet. scand. 1987, *28*, 81–92.

From the Department of Clinical Nutrition, College of Veterinary Medicine,
Swedish University of Agricultural Sciences, Uppsala, Sweden and the Department of Biochemistry,
College of Veterinary Medicine, Helsinki, Finland.

Bioavailability to Chicks of Selenium in Barley, Oats and Meat Meal

By *Saifeldin Hassan, R. V. Juhani Hakkarainen* and *Paul O. Lindberg*

Hassan, Saifeldin, R. V. Juhani Hakkarainen and Paul O. Lindberg: Bioavailability to chicks of selenium in barley, oats and meat meal. Acta vet. scand. 1987, 28, 81–92. – Day-old White Leghorn chicks deficient in selenium (Se) were fed a low Se basal diet (containing adequate level of vitamin E) for 2 weeks depletion period before they were given the experimental diets containing different levels of Se for 4 weeks. Dietary treatments contained 0.03, 0.06, 0.09 or 0.12 mg Se/kg as sodium selenite, barley, oats, meat meal or their extracted counterparts. Plasma GSH-Px activity was observed at weekly intervals, while the Se concentration of plasma and liver were determined at the end of the study. The biological availability of Se in the test ingredients was measured by the induction of plasma GSH-Px activity. In comparison to sodium selenite (100 %) it was: 77 % for barley, 80 % for extracted barley, 37 % for oats, 62 % for extracted oats, 20 % for meat meal, and 26 % for extracted meat meal. Using the retention of Se in plasma as a criterion, the following biological availability of Se was observed: barley 151 %; extracted barley 102 %; oats 90 %; extracted oats 107 %; meat meal 40 %; and extracted meat meal 47 %. Similarly, the efficiency of the test ingredients in increasing the Se concentration in liver was: barley 82 %; extracted barley 90 %; oats 67 %; extracted oats 98 %; meat meal 26 %; and extracted meat meal 31 %. The greater biopotency of the natural Se sources for increasing the Se concentration of the chick tissues than for inducing the plasma GSH-Px activity in comparison to sodium selenite, indicated that proportionally less amounts of the Se retained in the chick plasma from the natural sources were incorporated into the metabolic active form of Se, i.e., GSH-Px. Therefore, the plasma GSH-Px activity was suggested as the more reliable criterion to be used for the evaluation of the bioavailability of Se.

selenium bioavailability; glutathione peroxidase; selenium retention.

Introduction

The activity of the enzyme glutathione peroxidase (GSH-Px, glutathione-hydrogen peroxide oxidoreductase, EC 1.11.1.9) in plasma and whole blood has been utilized to estimate the biological availability of selenium (Se) in chicks (*Gabrielsen & Opstvedt* 1980), rats (*Sunde et al.* 1981, *Alexander et al.* 1983), turkey (*Cantor & Tarino* 1982) and pigs (*Sankari* 1985).

Cantor et al. (1975a) have shown that for the prevention of exudative diathesis (ED), 60 to 90 % of the Se present in plant materials and less than 25 % of that present in animal products is biologically available for the chick, as compared to sodium selenite. However, a lower biological availability of Se in plant materials and a higher availability of Se in fish meal has been reported (*Ikumo & Yo-*

shida 1981, *Gabrielsen & Opstvedt* 1980). Recently, *Huque & Jensen* (1985) and *Hassan* (1986) observed that approximately 60 to 70 % of the Se in fish meals is biologically available for the chick for the restoration of plasma GSH-Px activity. Thus the literature contains conflicting information about the bioavailability of Se.

It has been stated that vitamin E and Se act synergistically in the prevention of ED (*Scott et al.* 1957). Therefore, if chicks are fed sufficient amounts of vitamin E, they may be depleted of Se (i.e., GSH-Px) without developing this deficiency disease. The assessment of GSH-Px activity in such chicks will give an estimation of the bioavailability of Se without additional complications due to differences in feed intake, growth rate, deficiency symptoms, or by having trace amounts of vitamin E in the diet which exerts a curative effect against ED.

The purpose of this study was to evaluate the bioavailability of Se in barley, oats and meat meal as compared to that of sodium selenite for restoring plasma GSH-Px activity and for increasing the Se content of plasma and liver when fed to chicks which have been kept on a low Se diet with adequate amounts of vitamin E.

Materials and methods

Experimental animals

Four hundred and seventy-four 1-d old, unsexed, White Leghorn chicks were obtained from hens depleted of Se as described by *Hassan* (1986). Six chicks were immediately killed. Plasma samples and the liver were obtained and stored at –20°C. The remainder of the chicks were randomly allotted to 39 groups of 12 chicks each and kept in four-tiered electrically heated batteries with wire floor, as described previously (*Hassan* 1986).

Experimental diets

Selenium enriched barley was obtained by spraying sodium selenite in water solution on a low-Se field when the barley began to sprout (*Korkman* 1980). Oats was also obtained from a low Se field to which 4 kg Se (as Na_2SeO_3 in water solution) per hectare was sprayed 4 years before the production of oats for the present study (*Gissel-Nielsen* 1985). Barley and oats were milled separately for making the meals.

The meat meal was obtained from 2 Yorkshire pigs that were kept on a standard commercial ration containing 0.1 mg Se/kg since weaning. At 20 weeks of age, Se as sodium selenite was dissolved in water and sprayed in the commercial ration to supply 3 mg Se/kg diet. This high Se ration was fed to the 2 pigs for 7 weeks. The pigs were then slaughtered and the skeletal muscle was ground in an ordinary kitchen meat grinder and stored at –20°C. Small portions of the frozen ground meat were left to thaw at +4°C and then placed in thin layers on plates which were heated to 140°C for 3 h in a heating box connected to vacuum pump (Vacuum pump: VP 18, Alfa-Laval, Sweden). The ground and heat-dried meat was milled and this meat meal was stored at –20°C. The preparation of the meat meal in this manner was essential since the meat meal available commercially has a low Se concentration, reflecting the Se status of the soil in Sweden. The extracted counterparts of barley, oats and meat meal were prepared as described earlier (*Hassan* 1986, *Hassan et al.* 1987).

The composition of the low-Se basal diet fed to chicks is shown in Table 1. The contents of protein and Se in the basal diet fed to the chicks, in the low-Se casein and in the sources to be examined for their Se efficicacy are shown in Table 2. Sodium selenite ($Na_2SeO_3.5H_2O$) and the test sources were

added to the basal diet to supply levels of 0.03, 0.06, 0.09 or 0.12 mg Se/kg (Table 3). The content of protein in all dietary regimens was regulated to 23.5 %. This was attained by reducing the percentage of the low-Se casein in each experimental diet in response to the particular protein amount provided by the test Se source.

Table 1. Composition of basal diet fed to chicks (%)[1, 2].

Low-Se casein[3]	18.60
Extracted soybean meal	10.90
Gelatin	3.00
Skim milk powder	2.80
Amino acid mixture[4]	0.96
Vitamin mixture[5]	5.00
Mineral mixture[6]	5.60
Distilled fatty acids[7]	3.00
Cellulose	3.00
Glucose	47.14
Total	100.00

[1] By analysis contains 235.0 g crude protein/kg diet.

[2] Calculated content (g/kg) methionine 9.4, methionine + cystine 10.8, lysine 26.2.

[3] Produced as described by *Bengtsson et al.* (1974).

[4] Supplied the following amounts (/kg diet): DL-methionine 2.4 g, lysine 1.5 g, glycine 3.0 g, arginine 2.7 g.

[5] Supplied the following amounts (/kg diet): Retinol 18.3 mg, cholecalciferol 27.5 µg, menadione 1.4 mg, vitamin E 50.0 mg, (Vitamin E dry prowder, 50 %, Provimix E - 50 SE, F Hoffman-La Roche) thiamin 10.0 mg, riboflavin 12.0 mg, pantothenic acid 25.0 mg, nicotinic acid 55.0 mg, pyridoxine 8.0 mg, biotin 0.3 mg, pteroylmonoglutamic acid 4.0 mg, cyanocobalamin 0.03 mg, choline chloride 50 %, 4.0 g, 1.2-dihydro-6-ethoxy-2.2,4 trimethylquinoline (Ethoxyquin) 80.0 mg.

[6] Supplied the following amounts (/kg diet): $CaHPO_4.2H_2O$ 37.23 g, $MgSO_4.7H_2O$ 7.60 g, $CaCO_3$ 5.07 g, NaCl 5.00 g, $FeSO_4.7H_2O$ 0.50 g, $ZnSO_4.7H_2O$ 0.33 g, $MnSO_4.H_2O$ 0.26 g, $CuSO_4.5H_2O$ 39.3 mg, K_2SO_4 6.4 mg, KIO_3 5.1 mg.

[7] Obtained from soybean oil (*Hakkarainen et al.* 1983).

Table 2. Protein and selenium content of basal diet fed to chicks, low-Se casein and test ingredients.

Source	Protein %	Se mg/kg
Basal diet	23.50	0.03
Low-Se casein	74.20	0.04
Barley	10.57	1.41
Extracted barley	11.40	1.50
Oats	11.50	0.14
Extracted oats	12.80	0.28
Meat meal	50.89	0.35
Extracted meat meal	82.86	0.50

Experimental procedure

The environmental conditions for the present study were similar to those as described previously (*Hassan* 1986). The low-Se basal diet was fed from hatching for 2 weeks depletion period. Prior to commencement of dietary Se treatments, 6 chicks from one group were killed and the remnants of the chicks in that group were discarded. Plasma samples were obtained and stored at –20°C together with the liver samples. The dietary treatments were the basal diet with no Se addition (control diet), the basal diet supplemented with graded levels of Se (0.03, 0.06, 0.09 or 0.12 mg Se/kg diet) provided as sodium selenite (standard diets), or the basal diet supplemented only with one of the test ingredients as a source of Se (Table 3). Each dietary treatment was fed to two groups of chicks. Feed and water were provided ad lib. Randomly selected chicks (4–7) from each duplicate groups on a dietary treatment were killed at 1, 2, 3 or 4 weeks of Se supplement. Blood was collected in heparinized tubes and centrifuged at 2500 × g for 10 min. The

Table 3. The content of test ingredients, low-Se casein and glucose in experimental diets containing natural ingredients as Se source.

Diet*			Amount %		
No.	Se source	Se added mg/kg	Se source	Low-Se casein	Glucose
6	Barley	0.03	2.13	18.30	45.31
7		0.06	4.26	17.99	43.49
8		0.09	6.39	17.69	41.66
9		0.12	8.52	17.39	39.83
10	Extracted barley	0.03	2.00	18.29	45.45
11		0.09	6.00	17.68	42.06
12	Oats	0.03	22.19	15.16	28.39
13		0.06	44.38	11.72	9.64
14	Extracted oats	0.03	10.79	16.74	38.21
15		0.09	32.37	13.01	20.36
16	Meat meal	0.03	8.61	12.69	44.44
17		0.06	17.22	6.79	41.73
18	Extracted meat meal	0.03	6.00	11.90	47.84
19		0.06	12.00	5.20	48.54

* Diet 1 was a control with no Se supplementation, while diets 2, 3, 4 and 5 were supplemented with 0.03, 0.06, 0.09 or 0.12 mg Se/kg, respectively, as sodium selenite ($Na_2SeO_3.5H_2O$).

plasma was obtained and kept at –20°C until GSH-Px and Se analyses. Also, the liver was immediately removed and kept at –20°C until Se analysis. Body weight of the chicks was controlled at 3 and 4 weeks of age on a group basis.

Analytical methods

Selenium analyses of diets and tissues were performed according to a fluorometric method (*Lindberg* 1968). GSH-Px activity in plasma was measured by the method of *Günzler et al.* (1974) as modified by *Sankari* (1985). Protein in feedstuffs and diets was analysed according to *A.O.A.C.* (1970).

Statistical analyses

Data on plasma GSH-Px activity, Se concentration in plasma and liver, and gain in body weight of chicks were subjected to one-way analyses of variance according to *Dunn & Clark* (1974). Pairwise comparisons were conducted using the least significant difference method, while in the unbalanced cases, the least-square means were used in the comparisons as described by the *Statistical Analysis System* (*SAS* 1982). The efficacy of Se in the test sources relative to that in sodium selenite for elevating plasma GSH-Px activity, or increasing the Se concentration in plasma and liver was estimated by comparing the slope of the dose-response line relative to that of sodium selenite according to *Finney* (1978). In this slope ratio assay the control group values of plasma GSH-Px activity, plasma and liver Se concentration were employed as a common intercept. Analysis of the data was conducted using the procedure GLM from SAS.

Results

Plasma GSH-Px activity

The effect of dietary Se supplementation on the plasma GSH-Px activity measured at weekly intervals is shown in Table 4. Significant interactions were observed between dietary Se level, Se source and time. Therefore, the results of treatments were compared separately each week.

The plasma GSH-Px activity of the control group showed no significant changes ($p > 0.05$) throughout the 4 weeks of Se treatment. Supplementation of Se increased the plasma GSH-Px activity above that of the control group by the first week of Se treatment. This increase continued throughout the remainder of the experiment. However, only the plasma GSH-Px activity observed in the chicks fed sodium selenite significantly increased ($p < 0.05$) at all Se dietary levels from the first week of Se treatment as compared to that of the control group. Differences in the length in Se supplementation time required to obtain a significant increase ($p < 0.05$) in GSH-Px activity by the different dietary treatments, as compared to that of the control group, were observed. However, the chicks fed meat meal at the lowest level of dietary Se showed no significant increase ($p > 0.05$) in GSH-Px activity throughout the whole experiment when compared to that of the control group.

The GSH-Px activity for each dietary Se source was closely related to the levels of Se supplementation. Significant differences ($p < 0.05$) in GSH-Px activity were observed between chicks fed the same source of Se due to different levels of dietary Se. These differences were observed in chicks fed sodium selenite, barley, extracted barley and extracted oats in the first week of Se treatment. However, similar significant differences in GSH-Px activity were observed at the second week or later of Se supplementation in chicks receiving Se from other sources.

During the first week of Se treatment, no significant differences in plasma GSH-Px activity ($p > 0.05$) due to source of Se were observed in the chicks fed the lowest level of dietary Se. However, this difference in GSH-Px activity was significant ($p < 0.05$) at all dietary Se levels between Se sources from the second week and thereafter.

Within each dietary Se level, chicks fed barley or extracted barley showed a comparable or significantly lower ($p < 0.05$) GSH-Px activity than that observed in chicks fed sodium selenite. Chicks fed oats, extracted oats, meat and extracted meat meals all of which had an almost similar GSH-Px activity, showed a significantly lower ($p < 0.05$) GSH-Px activity than that of chicks fed sodium selenite, barley or extracted barley. Chicks fed meals from extracted Se sources had a comparable or significantly higher ($p < 0.05$) GSH-Px activity than that of chicks receiving the same level of Se from the unextracted counterpart meals.

Plasma Se content

The content of Se in plasma is shown in Table 5. With the exception of chicks fed meat and extracted meat meals at the level of 0.03 mg Se/kg diet, a significant increase ($p < 0.05$) in the plasma Se was observed in the chicks in the other groups in comparison to that of the control group. The concentration of Se in plasma increased with increasing dietary Se levels. The most pronounced increase in Se concentration was observed in the plasma of chicks fed barley. The chicks that were fed meat and extracted meat meals were significantly less responsive ($p < 0.05$) to Se supplementation than were the chicks fed sodium selenite and barley.

Table 4. Effect of dietary Se source on GSH-Px activity in chick plasma at 1, 2, 3 and 4 weeks Se supplementation*.

Se repletion time	Added Se in diet	Source of Se							
		None	Sodium selenite	Barley	Extracted barley	Oats	Extracted oats	Meat meal	Extracted meat meal
weeks	mg/kg	Plasma GSH-Px, (U/ml)							
1	0.00	$0.20\pm0.01^{s\dagger+}$	———	———	———	———	———	———	———
	0.03	———	0.72 ± 0.04^{Aa}§"	0.54 ± 0.05^{Aa}	0.33 ± 0.03^{Aa}	0.33 ± 0.04^{Aa}	0.37 ± 0.08^{Aa}	0.35 ± 0.01^{Aa}	0.29 ± 0.04^{Aa}
	0.06	———	1.22 ± 0.07^{Ba}	0.92 ± 0.07^{Bab}	———	0.41 ± 0.04^{Ac}	———	0.48 ± 0.04^{Abcd}	0.53 ± 0.04^{Abcd}
	0.09	———	2.03 ± 0.29^{Ca}	1.19 ± 0.22^{BCb}	1.19 ± 0.22^{Bb}	———	0.96 ± 0.10^{Bb}	———	———
	0.12	———	2.89 ± 0.10^{Da}	1.57 ± 0.17^{Cb}	———	———	———	———	———
2	0.00	$0.35\pm0.01^{s\dagger+}$	———	———	———	———	———	———	———
	0.03	———	0.80 ± 0.05^{Aa}§"	0.52 ± 0.04^{Aa}	0.47 ± 0.03^{Aab}	0.47 ± 0.04^{Aab}	0.53 ± 0.06^{Aab}	0.39 ± 0.02^{Ab}	0.39 ± 0.02^{Ab}
	0.06	———	2.29 ± 0.01^{Ba}	1.51 ± 0.26^{Ba}	———	0.93 ± 0.13^{Bc}	———	0.66 ± 0.07^{Ab}	0.88 ± 0.04^{Bb}
	0.09	———	3.46 ± 0.19^{Ca}	2.40 ± 0.14^{Cb}	2.62 ± 0.04^{Bb}	———	1.84 ± 0.11^{Bc}	———	———
	0.12	———	4.08 ± 0.20^{Da}	3.57 ± 0.50^{Db}	———	———	———	———	———
3	0.00	$0.42\pm0.06^{s\dagger+}$	———	———	———	———	———	———	———
	0.03	———	1.38 ± 0.06^{Aa}§"	0.88 ± 0.10^{Ab}	0.95 ± 0.05^{Aa}	0.71 ± 0.06^{Ab}	0.95 ± 0.06^{Ab}	0.62 ± 0.09^{Ab}	0.62 ± 0.07^{Ab}
	0.06	———	3.41 ± 0.35^{Ba}	1.51 ± 0.14^{Cb}	———	1.23 ± 0.17^{Bc}	———	0.99 ± 0.25^{Ad}	1.38 ± 0.11^{Bc}
	0.09	———	4.65 ± 0.04^{Ca}	3.37 ± 0.18^{Cb}	3.60 ± 0.35^{Bb}	———	2.54 ± 0.19^{Bc}	———	———
	0.12	———	4.93 ± 0.27^{Ca}	4.52 ± 0.28^{Da}	———	———	———	———	———
4	0.00	$0.32\pm0.03^{s\dagger+}$	———	———	———	———	———	———	———
	0.03	———	2.03 ± 0.15^{Aa}§"	1.16 ± 0.11^{Ab}	1.28 ± 0.14^{Ab}	1.06 ± 0.09^{Ab}	1.09 ± 0.10^{Ab}	0.69 ± 0.05^{Ab}	0.84 ± 0.07^{Ab}
	0.06	———	4.38 ± 0.28^{Ba}	2.46 ± 0.12^{Bb}	———	1.65 ± 0.08^{Bc}	———	1.29 ± 0.10^{Bd}	1.86 ± 0.12^{Be}
	0.09	———	4.89 ± 0.26^{Ca}	4.06 ± 0.21^{Cb}	4.02 ± 0.15^{Ab}	———	3.75 ± 0.23^{Bb}	———	———
	0.12	———	5.30 ± 0.19^{Da}	4.82 ± 0.41^{Db}	———	———	———	———	———

* Prior to Se repletion period of 4 weeks, chicks were depleted on Se for 2 weeks.
† GSH-Px activity of the control group at day 0 of Se supplementation was 0.28 ± 0.05.
+ Mean ± SEM.
[4 5 6 7 8]Number of observations per mean.
§ Means within a column with no common uppercase superscript are significantly different (with at least $p < 0.05$).
" Means within a row with no common lowercase superscript are significantly different (with at least $p < 0.05$).
[1] Means significantly different from the control group (with at least $p < 0.05$).

Table 5. Effect of dietary source on Se concentration in chick plasma and liver*.

Added Se in diet	Source of Se							
	None	Sodium selenite	Barley	Extracted barley	Oats	Extracted oats	Meat meal	Extracted meat meal
mg/kg				Plasma Se ng/ml				
0.00	22.25±0.91$^{\dagger +}$	——	——	——	——	——	——	——
0.03	——	72.90±5.66$^{Aa,\S"}$	59.37±1.85^{Aab,}	54.32±6.33^{Aabc,}	47.83±2.15^{Aaef,}	45.83±3.24^{Abde,}	34.10±2.79^{Acf}	38.82±1.59^{Acf}
0.06	——	104.40±13.65^{Ba}	107.10±7.25^{Ba,}	——	81.05±4.00^{Bb,}	——	51.45±1.98^{Ac,}	64.00±4.41^{Bbc,}
0.09	——	123.30±3.09^{BCa,}	156.83±7.09^{Ca,}	115.52±8.02^{Ba,}	——	130.20±6.47^{Ba,}	——	——
0.12	——	128.05±3.49^{Ca,}	208.82±19.99^{Db,}	——	——	——	——	——
				Liver Se, µg/g dry matter				
0.00	0.47±0.01$^{\dagger +}$	——	——	——	——	——	——	——
0.03	——	0.82±0.02$^{Aa,\S"}$	0.79±0.03^{Aa,}	0.81±0.05^{Aa,}	0.73±0.04^{Aa,}	0.79±0.03^{Aa,}	0.56±0.02^{Ab,}	0.59±0.03^{Ab,}
0.06	——	1.50±0.06^{Ba,}	1.09±0.03^{Ba,}	——	1.05±0.04^{Bb,}	——	0.73±0.02^{Bc,}	0.85±0.10^{Bc,}
0.09	——	1.91±0.03^{Ca,}	1.60±0.04^{Cb,}	1.58±0.09^{Bb,}	——	1.69±0.06^{Bb,}	——	——
0.12	——	1.83±0.08^{Ca,}	1.76±0.05^{Da,}	——	——	——	——	——

* Prior to a repletion period of 4 weeks, chicks were depleted of Se for 2 weeks.
† Selenium concentrations in plasma and liver of the control group at day 0 of Se supplementation were 30.50 ± 4.46 ng/ml and 0.51 ± 0.06 µg/g dry matter, respectively.
+ Mean ± SEM of 6 observations per group.
§ Means within a column with no common uppercase superscript are significantly different (with at least $p < 0.05$).
" Means within a row with no common lowercase superscript are significantly different (with at least $p < 0.05$).
[1] Means significantly different from the control group (with at least $p < 0.05$).

Table 6. Bioavailability of Se (%)* in the test ingredients relative to sodium selenite as measured by the induction of plasma GSH-Px activity, and the Se content in plasma and liver as calculated from the slope ratio data.

Bioassay used	Source of Se					
	Barley	Extracted barley	Oats	Extracted oats	Meat meal	Extracted meat meal
Plasma GSH-Px activity†	76.58 ± 0.76	79.66 ± 1.12	37.35 ± 0.82	61.76 ± 5.64	20.41 ± 4.64	26.31 ± 1.0
Plasma Se concentration+	151.27	102.03	90.36	107.16	40.08	46.61
Liver Se concentration+	82.20	90.07	66.93	97.75	25.94	31.14

* Compared to sodium selenite as 100 %.
† Mean ± SEM of Se biological availability measured at 2, 3 and 4 weeks of Se supplementation.
\+ Measured at 4 weeks of Se supplementation.

Liver Se content

Data on the content of Se in liver are presented in Table 5. All the dietary Se treatments increased significantly ($p < 0.05$) the concentration of Se in liver, in comparison to that of the control group except in the groups where meat and extracted meat meals supplied 0.03 mg Se/kg diet.

Chicks fed sodium selenite had comparable or significantly higher ($p < 0.05$) liver Se concentrations than that of chicks receiving Se via other dietary sources. The increase of Se in the liver of chicks fed meat and extracted meat meals was similar ($p < 0.05$). However, this increase was significantly lower ($p < 0.05$) than that of chicks supplemented via other Se sources.

Bioavailability of selenium

Table 6 shows the biological availability of Se in the test ingredients in comparison to sodium selenite. This was measured by the induction of plasma GSH-Px activity and retention of Se in plasma and liver, as analysed by the slope ratio assay. After the first week of Se treatment, in general no significant differences ($p < 0.05$) in plasma GSH-Px activity were observed, between the chicks fed different dietary Se levels from the same source. Therefore, these results were not used for the slope ratio calculations.

Body weight

The body weights of the chicks, at 3 and 4 weeks of Se supplementation, showed no significant differences ($p > 0.05$) due to the various dietary treatments. Therefore, these data are not presented here.

Discussion

The plasma GSH-Px activity and Se concentration in plasma and liver of the control group showed no significant changes throughout the Se treatment period from the initial levels. Therefore the two weeks on the basal diet were sufficient to deplete the chicks of Se in the plasma and liver and the plasma GSH-Px activity. The rapid and significant increase in plasma GSH-Px activity observed at one week of Se supplementation in chicks fed sodium selenite, as compared to that of chicks fed Se from other sources corroborated recent results on the bioavailability of Se in wheat and fish meals (*Hassan et al.* 1987). It is evident that Se from sodium selenite is rapidly metabolized and more effectively used by the chick for plasma GSH-Px synthesis than is the Se found in natural ingredients.

The present results with respect to the biological availability of Se in barley, both for the induction of plasma GSH-Px and for increasing the Se concentration of liver, agree with the results of *Seier & Bragg* (1973), who measured the efficacy of Se in the prevention of ED. The high bioavailability of barley Se as measured by the retention of Se in plasma (150 %) was difficult to explain. In previous studies, high affinity of Se in some natural sources to specific organ or tissue has been observed (*Cantor et al.* 1975a, 1975b). The chemical form of Se in barley and oats is not known. However, the high retention of barley Se in this study suggests that a major portion of the Se in barley may be present in the same chemical form as in wheat, i.e., as selenomethionine (*Olson et al.* 1970), which has a high Se retention in animal tissues (*Alexander* 1983, *Hassan et al.* 1987).

A biological availability of 90 and 67 % was observed for oats Se as measured by Se retention in plasma and liver, respectively, while only 30 % was observed using the induction of GSH-Px. Similar low efficacy estimate for Se in oats to increase plasma GSH-Px has been observed recently (*Hassan* 1987). Apparently a higher proportion of the Se in plasma obtained from oats than from barley was unavailable for GSH-Px synthesis.

The low biological availability of Se observed in meat meal both for restoring plasma GSH-Px activity and for increasing the Se concentration in chick tissues agreed with the results of *Cantor et al.* (1975a), who observed a bioavailability of 15 % for meat meal Se in the prevention of ED in comparison with sodium selenite. However, the biological availability of Se in meat meal as established in the present study is much lower than that of Se in fish meal (*Hassan* 1986). One may speculate that the Se in meat meal is bound to animal protein in form(s) which are less available for the chick than those of fish meal.

The extracted meals used in this study as Se sources, were comparable or superior to their counterparts for the induction of plasma GSH-Px activity or for increasing Se retention in tissues. When using the prevention of Se-vitamin E responsive diseases as a criterion for the evaluation of biological availability of Se in feed ingredients, a practical difficulty arises due to the synergism of Se and vitamin E. *Cantor et al.* (1975a) and *Combs et al.* (1980) used hexane and petrolium ether extraction, respectively, for about 24 h to eliminate vitamin E in the feed ingredients tested for the efficacy of Se in the prevention of ED. As feed processing which involves heat treatment has been observed to reduce Se bioavailability (*Whitacre & Latshaw* 1982, *Huque & Jensen* 1985), there is a possibility that the extraction procedure which was used in the above mentioned studies has a similar effect on Se utilization. However, according to the present study the extraction process does not reduce the bioavailability of Se for GSH-Px synthesis or for increasing the Se content in chick tissues. The reason for the lower biological availability values for Se in chicks fed the unextracted meals is difficult to understand. A possible explanation may be that factor(s) in the fat component which inhibit the utilization of Se were eliminated by the extraction process.

The slope ratio results revealed that for each tested Se source, a higher biological availability of Se was observed when the retention of Se in plasma was used as a criterion than when the induction of plasma GSH-Px was the parameter, while the vise versa was observed for sodium selenite. This indicated a higher activity of plasma GSH-Px when expressed as enzyme units/μg Se in

chicks fed sodium selenite than in those receiving Se from other sources. Thus, it may be suggested that a high proportion of Se in the plasma of chicks fed natural ingredients is incorporated into chemical form(s) which are poorly utilized for GSH-Px synthesis, in comparison to the Se obtained from sodium selenite.

The body weight of the chicks was not affected by different sources or levels of dietary Se. This agrees with the observations of previous studies (*Cantor & Tarino* 1982, *Hassan et al.* 1987). This observation was expected and desired in order to overcome possible differences in Se intake due to differences in chicks growth rate. The basal diet fed to chicks contained sufficient amounts of vitamin E, sulphur containing amino acids and ethoxyquin, nutrients that prevent Se-vitamin E deficiency diseases in the chick and promote normal growth rate. The results indicated that the body weight is not a sensitive criterion for evaluating the bioavailability of Se when the chicks receive adequate amounts of vitamin E.

The present study established that Se from sodium selenite is more efficacious in restoring plasma GSH-Px activity than the Se obtained from natural sources. However, the greater potency of the natural Se sources for increasing the Se concentration of the chick tissues than for inducing GSH-Px activity indicated that the enzyme activity is the more reliable criterion to be used for evaluation of the bioavailability of Se.

Acknowledgements

The authors are grateful to Johan Korkman, Ph. D., and Kemira Oy, Helsinki, Finland and to Gunnar Gissel-Nielsen, Ph. D., Risø National Laboratory, Roskilde, Denmark, who have most generously placed selenium enriched barley and oats, respectively, at our disposal. They are indebted to Birgitta Vegerfors-Persson, M. Sc., and Gunnar Ekbohm, Ph. D., Department of Economics and Statistics, College of Agricultural Sciences, Uppsala, Sweden, for performing the statistical analyses. This investigation was supported by grants from the Swedish Council for Forestry and Agricultural Research.

References

Alexander AR, Whanger PD, Miller LT: Bioavailability to rats of selenium in various tuna and wheat products. J. Nutr. 1983, *113,* 196–204.

A.O.A.C.: In: Official Methods of Analysis of the Association of Official Analytical Chemists, 11th ed. Ed.: Horwitz W: Association of Official Analytical Chemists, Benjamin Franklin Station, DC 1970, pp. 16–17.

Cantor AH, Langevin ML, Noguchi T, Scott ML: Efficacy of selenium compounds and feedstuffs for prevention of pancreatic fibrosis in chicks. J. Nutr. 1975(a), *105,* 106–111.

Cantor AH, Scott ML, Noguchi T: Biological availability of selenium in feedstuffs and selenium compounds for prevention of exudative diathesis in chicks. J. Nutr. 1975(b), *105,* 96–105.

Cantor AH, Sutton CD, Johnson TH: Biological availability of selenodicysteine in chicks. Poult. Sci. 1983, *62,* 2429–2432.

Cantor AH, Tarino JZ: Comparative effects of inorganic and organic dietary sources of selenium on selenium levels and selenium-dependent glutathione peroxidase activity in blood of young turkeys. J. Nutr. 1982, *112,* 2187–2196.

Combs GF Jr, Barrows SA, Swader FN: Biological availability of selenium in corn grain produced on soil amended with fly ash. J. Agric. Food Chem. 1980, *28,* 406–409.

Dunn OJ, Clark VA: Applied Statistics. Analysis of variance and regression, 1st ed. John Wiley & Sons, New York. 1974, pp. 62–86.

Finney DJ: Statistical method in biological assay. 3rd ed. Alden Press, Oxford London. 1978, pp. 148–168.

Gabrielsen BO, Opstvedt J: Availability of selenium in fish meal in comparison with soybean meal, corn gluten meal and selenomethionine relative to selenium in sodium selenite for re-

storing glutathione peroxidase activity in selenium depleted chicks. J. Nutr. 1982, *110*, 1096–1100.

Gissel-Nielsen G: Personal communication. 1985.

Günzler WA, Kremers H, Flohé L: An improved coupled test procedure for glutathione peroxidase (EC. 1.11.1.9) in blood. Z. Klin. Chem. Klin. Biochem. 1974, *12*, 444–448.

Hassan S: Comparative effect of selenium in wheat, barley, fish meal and sodium selenite for prevention of exudative diathesis in chicks. Acta vet. scand. 1986, *27*, 461–478.

Hassan S: Comparative effect of selenium in oats, meat meal, selenomethionine and sodium selenite for prevention of exudative diathesis in chicks. Zbl. Vet. Med. A 1987, *34*, 204–215.

Hassan S, Hakkarainen RVJ, Lindberg PO: Bioavailability of selenium in wheat and fish meal. Zbl. Vet. Med. A. 1987, *34*, 353–363.

Huque QME, Jensen JF: Biological availability of selenium and phosphorus in fish meal as affected by condition of fish and type of meal. Brit. Poult. Sci. 1985, *26*, 289–297.

Ikumo H, Yoshida M: Selenium content of feedstuffs and biological availability of selenium in chicks. Japan. Poultry Sci. 1981, *18*, 307–311.

Korkman J: The effect of selenium fertilizers on the selenium content of barley, spring wheat and potatoes. J. Sci. Agric. Finl. 1980, *52*, 495–504.

Lindberg P: Selenium determination in plant and animal material, and in water. Acta vet. scand. 1968, Suppl. 23.

Olson OE, Novacek EJ, Whitehead EI, Palmer IS: Investigations on selenium in wheat. Phytochemistry 1970, *9*, 1181–1188.

Sankari S: Plasma glutathione peroxidase and tissue selenium response to selenium supplementation in swine. Acta vet. scand. 1985, Suppl. 81, 1–127.

SAS: Statistical Analysis System User's Guide. SAS Institute Inc., Cary, North Carolina 1982.

Scott ML, Bieri JG, Briggs GM, Schwarz K: Prevention of exudative diathesis by factor 3 in chicks on vitamin E-deficient torula yeast diets. Poult. Sci. 1957, *36*, 1155.

Seier L, Bragg DB: Influence of vitamin E and antioxidant on the response of dietary selenium by the chick and the biological activity of selenium in feed grain. Canad. J. Anim. Sci. 1973, *53*, 371–375.

Sunde, RA, Gutzke GE, Hoekstra WG: Effect of dietary methionine on the biopotency of selenite and selenomethionine in the rat. J. Nutr. *111*, 76–86.

Whitacre M, Latshaw JD: Selenium utilization from menhaden fish meal as affected by processing. Poult. Sci. 1982, *61*, 2520–2522.

Sammanfattning

Biologisk tillgänglighet av selen i korn, havre och köttmjöl till kycklingar.

Daggamla vita Leghornkycklingar utarmade på selen utfodrades med en selenfattig basaldiet (innehållande adekvat nivå av vitamin E) under 2 veckor innan de sattes på försökskoster innehållande olika selennivåer under ytterligare 4 veckor. De olika dieterna innehöll 0.03, 0.06, 0.09 eller 0.12 mg Se/kg foder som natriumselenit, korn, havre, köttmjöl eller de tre sistnämnda foderkomponenternas extraherade motsvarigheter. Plasma GSH-Px aktiviteten mättes med en veckas intervaller medan selenkoncentrationen i plasma och lever bestämdes vid försökets slut. Selenets biologiska tillgänglighet i de testade foderkomponenterna mättes med induceringen av GSH-Px aktiviteten i plasma. Jämfört med natriumselenit (100 %) var den biologiska tillgängligheten av selen 77 % i korn, 80 % i extraherat korn, 37 % i havre, 62 % i extraherat havre, 20 % i köttmjöl och 26 % i extraherat köttmjöll. Då selenretentionen i plasma användes som kriterium kunde följande värden fastslås för selenets biologiska tillgänglighet: 151 % för korn, 102 % för extraherat korn, 90 % för havre, 107 % för extraherat havre, 40 % för köttmjöl och 47 % för extraherat köttmjöl. På motsvarande sätt och då selenkoncentrationen i levern användes som måttstock erhölls följande värden för selenets biologiska tillgänglighet i respektive fodermedel: 82 % i korn, 90 % i extraherat korn, 67 % i havre, 98 % i extraherat havre, 26 % i köttmjöl och 31 % i extraherat köttmjöl. Den högre biopotensen hos naturliga

selenkällor vad gällde ökningen av selenkoncentrationen i kycklingvävnader än vid induceringen av GSH-Px aktiviteten i plasma jämfört med natriumselenit, visade att proportionellt mindre mängder av selen, som retinerades i kycklingplasma från naturliga källor inkorporerades i metaboliskt aktiv form av selen, dvs. i GSH-Px. Därför fastslogs att plasma GSH-Px aktiviteten är ett mera pålitligt kriterium för bestämning och utvärdering av selenets biologiska tillgänglighet.

(Received November 7, 1986).

Reprints may be requested from: Saifeldin Hassan, Department of Clinical Nutrition, College of Veterinary Medicine, Swedish University of Agricultural Sciences, P. O. Box 7023, S-750 07 Uppsala, Sweden.