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SELENIUM CONCENTRATIONS IN TISSUES AND EGGS OF GROWING AND LAYING CHICKENS FED SODIUM SELENITE AT DIFFERENT LEVELS

By

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MOKSNES, KNUT and GUNNAR NORHEIM: *Selenium concentrations in tissues and eggs of growing and laying chickens fed sodium selenite at different levels.* Acta vet. scand. 1982, 23, 368—379. — Growing and laying chickens were fed graded levels of selenium in the form of sodium selenite. One day old Norwegian bred broiler chickens and 20 weeks old Norwegian bred White Leghorn chickens were divided into 5 groups each and fed a basal diet supplemented with 0, 0.1, 1.0, 3.0 or 6.0 μg Se/g for 6 and 31 weeks, respectively. At the end of the experiments significantly higher concentrations of selenium were found in the groups fed 1.0, 3.0 and 6.0 μg Se/g diet compared to the control group. Correspondingly higher concentrations of selenium were found in egg samples. The increase in egg yolk selenium was much higher than in egg white. Significant correlations were found between the amounts of selenium added to the ration and the selenium concentrations in liver, kidney, breast muscle, egg white, yolk and homogenized egg. There were no differences in body weight gain and egg production between the groups. A possible positive contribution to animal and human health of selenium supplementation of animals' diet above the required level is discussed.

dietary selenium; tissue; egg; selenium concentrations; growing chickens; laying chickens.

Until *Schwarz & Foltz* in 1957 demonstrated that selenium was the active component of "factor 3", selenium was regarded solely as a highly toxic element. Since then, the beneficial effect of selenium in a number of diseases, both in laboratory and farm animals, has been demonstrated. A level of 0.1 μg Se/g in the diet is considered satisfactory under most conditions to prevent deficiency symptoms in chickens (*Thompson & Scott* 1969). Toxic effects may be observed at levels of 5 ppm (*Ort & Latshaw* 1978) thus giving a safety margin of 1:50.

Natural selenium deficiency diseases in livestock and poultry have been reported in many countries. In Norway low tissue levels of selenium have been demonstrated in several domestic animal species, and feedstuffs of Norwegian origin are extremely poor in selenium (*Frøslie et al.* 1980). As a result, it was decided that 0.1 μg Se/g, in the form of sodium selenite, should be added to all domestic animal feedstuffs in Norway (*Landbruksdepartementet* 1979).

The optimum intake of selenium in farm animals is difficult to ascertain. This level may also differ depending on the criteria used for its assessment. However, it has been assumed that the optimum level is higher than 0.1 μg Se/g diet (*Pehrson* 1980, *Jönsson et al.* 1981).

The following study was made to determine the influence of dietary selenium on tissue and egg selenium levels, body weight gain and egg production in growing and laying chickens. Special emphasis was placed on evaluating possible risks for humans consuming meat and eggs from animals receiving high levels of dietary selenium in the form of sodium selenite.

MATERIAL AND METHODS

Growing chickens

One day old Norwegian bred broiler chickens were used for this study and were divided into 5 groups of 26 birds each. Five birds were killed before the dietary study began in order to establish basal tissue levels. The remaining chickens were fed a basal diet of broiler starter feed containing 0.13 μg Se/g, supplemented with 0, 0.1, 1.0, 3.0 or 6.0 μg Se/g in the form of sodium selenite. The composition of the diet is given in Table 1. The study lasted for 6 weeks. After 2 and 4 weeks, 5 birds from each group were killed, and liver and breast muscle samples analysed. After 6 weeks the remainder were killed and controlled for pathological lesions and body weight gain. Tissue samples from 10 chickens were analysed at the end of the experiment. One bird died during the experiment.

Laying chickens

Norwegian bred White Leghorn chickens were used for this study. The birds were 20 weeks old at the start of the experiment and had been fed an ordinary diet with no supplement of

Table 1. Composition of the diets.

Diet		Broiler starter feed*	All-mash cage laying feed 15 %*
Herring meal, extra quality	%	6.0	5.5
Meat and bone meal	„	3.5	3.5
Soyabean meal, extracted	„	13.3	2.4
Ground barley	„	5.0	5.0
Ground maize	„	12.8	20.0
Maize grits	„	6.2	7.8
Ground sorghum	„	11.5	14.2
Ground oats	„	18.0	17.0
Wheat brand	„	5.0	9.6
Grass meal	„	—	2.5
Fat	„	3.5	0.9
Molasses	„	2.0	2.0
Mineral mixture	„	2.5	9.1
Vitamins	„	0.5	0.5
Ground wheat	„	10.0	—
Limestone meal	„	0.2	—
Garanteed content:			
Crude protein	%	18—20	14—16
Digestible crude protein	„	15—17	11—13
Fat	„	6—7	—
Metaboliseable energy	kcal/kg	2940—3060	2600—2720
Calculated content:			
Crude fibre	%	4.0	4.4
Fat	„	—	3.9
Lysine	„	1.03	0.74
Methionine + Cysteine	„	0.75	0.57
Linoleic acid	„	1.20	1.20
Calcium (Ca) per kg	„	1.2	3.5
Phosphorus (P) „	„	0.7	0.7
Salt (NaCl) „	„	0.5	0.5
Selenium (analysed)	µg/g	0.13	0.17
Added per kg:			
Vitamin A	I.U.	15,000	12,000
Vitamin D_3	„	2,400	2,000
Vitamin E	mg	30	20
Vitamin K_3	„	2	0.5
Riboflavine (B_2)	„	7	5
Folic acid	„	1	0.5
Pantothenic acid	„	10	5
DL-Methionine	„	810	270
Niacin	„	15	—
Biotin	„	50	—
Pyridoxine (B_6)	„	2	—
Choline chloride	„	400	—
Manganese oxide	„	64	—

* Commercial concentrates from Arex-Møllesentralen, Oslo.

selenium. Five groups of 16 birds were individually caged and fed a basal diet of all-mash cage laying feed containing 0.17 μg Se/g, supplemented with 0, 0.1, 1.0, 3.0 or 6.0 μg Se/g in the form of sodium selenite (Table 1). Seven birds were killed before the dietary study began in order to establish basal tissue levels.

After 18 weeks (age: 38 weeks), 5 birds from each group were killed and liver, kidney and breast muscle samples analysed. At the end of the experiment, after 31 weeks (age: 51 weeks), the rest of the birds were killed and examined for gross- and histopathological lesions. Tissue samples from 10 birds were taken for selenium analysis.

Every 5 weeks 10 eggs were collected from each group. The total contents of 5 eggs were blended. The whites and yolks of the other 5 eggs were separated before pooling and blending. Records were maintained to calculate egg production. Eggs laid during the last 10 days were saved to calculate egg weight. The gain in body weight of all of the birds used in these experiments was monitored. Three birds died during the experiment, 2 due to lymphoid leukosis and 1 due to an adenocarcinoma.

Analytical methods

Breast muscle, liver and kidney samples were collected from each bird. The samples were stored at —20°C immediately after collection. The tissue and egg samples were analysed by a modification of the fluorimetric method (*Ihnat* 1974), *Norheim & Nymoen* 1981). Results are expressed as μg Se/g on a wet weight basis. Material for analysis was taken in a semifrozen state to prevent loss of fluid from the samples.

RESULTS

Growing chickens

The addition of sodium selenite to the diets increased the selenium level in tissues of growing chickens (Table 2). At the end of the experiment a higher liver selenium level was found in all groups supplemented with selenium compared to the control group. A significant correlation was observed between the liver selenium levels and the concentration of selenium added to the ration (Table 3). There also seemed to be a correlation between the selenium levels in breast muscle and selenium intake (Table 3), but the results were more scattered. However, at

Table 2. Selenium concentrations (mean ±s μg Se/g wet weight) in muscle and liver of growing chickens as affected by duration of selenium feeding and level of dietary selenium.

Added Se μg/g	Tissue	Duration of selenium feeding (weeks) 0 (n=1*)	2 (n=1*)	4 (n=5)	6 (n=10)
0	Muscle	0.22	0.16	0.21±0.01	0.21±0.02
	Liver	0.74	0.42	0.53±0.02	0.61±0.04
0.1	Muscle		0.21	0.21±0.03	0.21±0.01
	Liver		0.58	0.69±0.03	0.68±0.05
1.0	Muscle		0.28	0.28±0.01	0.24±0.04
	Liver		0.96	1.2 ±0.20	0.80±0.08
3.0	Muscle		0.25	0.24±0.02	0.28±0.02
	Liver		1.88	1.5 ±0.23	2.0 ±0.21
6.0	Muscle		0.26	0.25±0.01	0.36±0.04
	Liver		3.35	3.0 ±0.41	3.2 ±0.29

n = number of analyses.
* Each value represents 1 pooled sample of 5 animals.

the end of the experiment this correlation was also significant ($r = 0.85$, $P < 0.001$, $n = 50$). The selenium supplementation had no effect on the increase in body weight gain. The average body weight at slaughter was 1300 g.

Table 3. The selenium concentrations (μg Se/g wet weight) in liver (y_1) and muscle (y_2) from growing chickens and liver (y_3), muscle (y_4), kidney (y_5), homogenized egg (y_6), egg yolk (y_7) and egg white (y_8) from laying chickens as a function of the selenium concentrations (μg Se/g dry weight) added to the ration (x) in the form of sodium selenite.

Regression function	n	r	P
$y_1 = 0.42\ x + 0.58$	81	0.97	< 0.001
$y_2 = 0.016x + 0.23$	81	0.66	< 0.001
$y_3 = 0.45\ x + 0.37$	82	0.94	< 0.001
$y_4 = 0.025x + 0.16$	82	0.93	< 0.001
$y_5 = 0.26\ x + 0.56$	11	0.96	< 0.001
$y_6 = 0.22\ x + 0.17$	30	0.97	< 0.001
$y_7 = 0.42\ x + 0.32$	30	0.99	< 0.001
$y_8 = 0.12\ x + 0.089$	30	0.97	< 0.001

n = number of samples analysed, r = correlation coefficient and P = probability.

Laying chickens

The feeding of diets supplemented with sodium selenite increased the selenium concentration in tissues of laying chickens (Table 4). Significant increases in liver, kidney and breast muscle levels occurred when the diet contained 1.0, 3.0 or 6.0 μg/g added selenium. Breast muscle selenium increased less than liver and kidney selenium in response to dietary selenite.

In the control group and the group supplemented with 6 ppm selenium the liver selenium level increased between the 18th and 31st week of the feeding period. In the other groups the liver selenium level decreased between the 18th and 31st week of the feeding period.

Significant linear correlations were found between the concentration of selenium added to the ration and the subsequent levels in liver, kidney and muscle tissue (Table 3). Despite the high correlation between muscle selenium and the dietary intake, the line describing this connection may be slightly curved.

Table 4. Selenium concentrations (mean ± s μg Se/g wet weight) in tissues of laying hens as affected by duration of selenium feeding and level of dietary selenium.

Added Se μg/g	Tissue	Duration of selenium feeding (weeks) 0 (n=7)	18 (n=5)	31 (n=10)
0	Muscle	0.16±0.01	0.15±0.07	0.14±0.01
	Liver	0.47±0.06	0.37±0.03	0.43±0.03
	Kidney*	0.56	0.44	0.51
0.1	Muscle		0.16±0.01	0.15±0.01
	Liver		0.46±0.04	0.43±0.03
	Kidney		0.62	0.62
1.0	Muscle		0.21±0.04	0.20±0.001
	Liver		0.77±0.09	0.67±0.10
	Kidney		0.86	0.35
3.0	Muscle		0.25±0.008	0.27±0.02
	Liver		1.9 ±0.19	1.5 ±0.36
	Kidney		1.3	1.4
6.0	Muscle		0.29±0.01	0.30±0.01
	Liver		2.4 ±0.59	3.4 ±0.62
	Kidney		1.7	2.5

n = number of analyses.

* Each value represents 1 pooled sample of all of the birds in each group.

The selenium supplementation had no significant effects on the increase in body weight gain or egg production. At the end of the experiment the chickens had an average body weight of 1800 g and the egg production reached a maximum of approximately 80 % after 10 weeks of the feeding period, one egg per chicken per day being 100 %. No macroscopical and histopathological changes were observed in any of the birds at necropsy.

Eggs

When 0.1 μg/g of selenium was added to the diet, the selenium content of egg white was almost the same as in the control group, but a detectable increase was found in yolk selenium. Feeding 1.0, 3.0 or 6.0 μg Se/g led to increased concentration in both egg white and yolk (Table 5). Corresponding levels were found in the homogenized egg samples. Significant correlations were found between the amount of selenium added to the ration and the selenium levels in egg white, yolk and homogenized egg (Table 3). There was a constant ratio between the selenium level

Table 5. Selenium concentrations (μg Se/g wet weight) in homogenized eggs, egg yolk and egg white as affected by duration of selenium feeding and level of dietary selenium. Each value represents 1 pooled sample of 5 eggs.

Added Se	Sample	Duration of selenium feeding (weeks)					
μg/g		5	10	15	20	25	30
0	Homogenized eggs	0.20	0.22	0.17	0.19	0.19	0.21
	Egg yolk	0.35	0.38	0.36	0.33	0.33	0.39
	Egg white	0.12	0.12	0.07	0.10	0.12	0.10
0.1	Homogenized eggs	0.20	0.25	0.22	0.22	0.24	0.26
	Egg yolk	0.42	0.42	0.42	0.45	0.48	0.48
	Egg white	0.12	0.13	0.10	0.12	0.11	0.12
1.0	Homogenized eggs	0.31	0.33	0.32	0.35	0.37	0.35
	Egg yolk	0.60	0.59	0.50	0.67	0.60	0.60
	Egg white	0.16	0.17	0.17	0.17	0.16	0.15
3.0	Homogenized eggs	0.62	0.83	0.78	0.89	0.80	0.81
	Egg yolk	1.7	1.6	1.8	1.6	1.4	1.4
	Egg white	0.40	0.46	0.53	0.43	0.43	0.43
6.0	Homogenized eggs	1.4	1.3	1.4	1.7	1.4	2.1
	Egg yolk	2.7	2.5	3.1	2.8	2.9	3.1
	Egg white	0.83	0.54	0.90	0.79	0.71	0.99

in egg yolk, egg white and homogenized egg during the feeding period in all five groups.

Eggs laid during the last 10 days of the experiment showed that the dietary selenium had no effect on the egg weight. The average egg weight was 59 ± 4.3 g.

DISCUSSION

There is a close relationship between the amount of selenium added to the ration and the subsequent selenium levels found in tissues and eggs of growing and laying chickens. When using sodium selenite as a source of selenium, marked increases were found in the selenium concentrations in liver, kidney and egg, especially in the yolk. On average, selenium levels increased by a factor of 7 in liver, 4 in kidney and less than 2 in muscle when 6.0 μg Se/g was added to the ration, compared to the control group.

The present results are in agreement with corresponding results on growing pigs where high correlations were observed between the selenium intake and tissue levels of selenium (*Moksnes et al.* 1982). In sheep, however, a much more drastic increase in the hepatic selenium level was observed, whereas the muscular response was very small (*Moksnes & Norheim,* to be published). Thus it appears that different species vary in their tissue response to sodium selenite in the feed.

The amounts of selenium given to growing and laying chickens in the present study did not give any signs of toxic effects. The body weight gain and the egg production were not affected. This is in agreement with previous studies by *Ort & Latshaw* (1978). They found that the most sensitive criterion for toxic effects of selenite selenium was the hatchability of fertile eggs and that this was decreased by about 12 % at a selenium level of 5 μg/g in the diet. Egg weight was decreased at 7 μg Se/g and egg production at 9 μg/g as sodium selenite in the diet. Previously *Arnold et al.* (1973) found that egg size and percentage of fertile eggs hatching were decreased at 8 μg Se/g feed.

The tissue selenium levels, both in growing and laying chickens, varied somewhat during the feeding period. In the control group of growing chickens, the liver levels increased significantly from the 2nd to the 4th and the 6th week. The

muscle levels showed considerable variations in all 5 groups, probably because of the short duration of this experiment. Among the laying chickens a significant increase was found in the liver and kidney selenium levels from the 16th to the 31st week in the control group and the group fed 6.0 μg Se/g. In the other 3 groups a small and insignificant decrease was observed. These trends indicate a homeostatic mechanism regulating the tissue selenium levels after intake of sodium selenite and this mechanism is effective at least up to an intake of 3.0 μg Se/g in the feed. At an intake of 6.0 μg Se/g feed, selenium accumulates in the tissues analysed.

No significant changes in the selenium levels were observed in eggs from all 5 feeding groups over the experimental period. The highest selenium concentrations were found in yolk, the average ratio between egg yolk and egg white being 3.6. *Ort & Latshaw* (1978) obtained similar results. The ratio between egg yolk and egg white is also in agreement with previous studies where sodium selenite was added to the feed (*Latshaw & Osman* 1975). A different ratio is found when organic selenium compounds is used (*Moksnes,* to be published). *Herstad & Hvidsten* (1981) have also given selenium to chicks and laying hens and they found lower selenium concentrations than reported here. However, they analysed only a very small number of samples.

Feedstuffs of Norwegian origin are usually extremely poor in selenium, one exception being herring meal, and most of the naturally occurring selenium in concentrates comes from imported grains. The feed used in the control groups contained between 0.1 and 0.2 μg Se/g. Supplementing the basal diet with sodium selenite showed no effects other than increased selenium concentrations in tissues and eggs of the animals. Thus the present study does not give a clear indication of the optimum intake of selenium in growing and laying chickens.

In Norway selenium is added to all mixed concentrates and mineral mixtures at amounts equivalent to 0.1 μg Se/g feed. This selenium is added as sodium selenite. Possible errors in the working procedure may result in incorrect concentrations of selenium in the final product. However, the present study clearly shows that the addition of 10 to 20 times the recommended levels has no negative effects on growing and laying chickens. It has been stated that 2 μg Se/g diet has produced no unequivocally toxic signs, and this dietary concentration is suggested as a

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maximum tolerable level for all species (*National Academy of Sciences* 1980).

Oldfield (1981) has recently pointed out that when the animal diet is already adequate, i.e. containing 0.1 μg Se/g, supplementation of selenium is unnecessary and worthless. In low selenium areas with a suboptimal human intake of selenium this is not strictly true. Addition of selenium above the required levels for domestic animals' diet will lead to increased selenium levels in meat and other farm animal products, thus giving a positive contribution to the human selenium intake. This possibility is therefore worthwhile considering.

The addition of selenium to animals' diet has been shown to increase the immune response. Supplementing diets low in selenium also increases the resistance against enteric infections (*Teige* 1978). Thus it is possible that the supplementation of selenium above 0.1 μg Se/g feed would be beneficial for animal health.

It has been suggested that the optimum human intake of selenium is in the order of 200—300 μg Se/day (*Schrauzer* 1980, *Shamberger* personal communication). Based on this suggestion, available data on human nutrition in Norway seem to indicate a suboptimal selenium intake. It would therefore be a positive contribution to the human selenium intake if chickens were given the maximum tolerable level of selenium in the diet. However, more data on the human nutrition aspects are obviously needed. Addition of higher concentrations of sodium selenite than the 0.1 μg Se/g feed used today, would, however, give a smaller safety margin in the handling of sodium selenite. As will be shown in a later paper, the use of selenomethionine, instead of sodium selenite, as a source of selenium gives higher muscular levels of selenium. If one intends to increase the selenium level in meat and eggs, the use of selenomethionine would give a larger safety margin when adding selenium to feedstuffs.

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SAMMENDRAG

Selenkonsentrasjoner i organer og egg hos broilere og verpehøns som har fått forskjellige mengder natriumselenitt i fôret.

Det er blitt gjennomført to separate forsøk, ett med broilere og ett med verpehøns, for å se på hvilke effekter forskjellige mengder av natriumselenitt i fôret har på selennivåene i organer og egg. En dag gamle kyllinger og 20 uker gamle høner ble hver delt i 5 grupper og gitt et grunnfôr tilsatt henholdsvis 0, 0,1, 1,0, 3,0 eller 6,0 µg Se/g i henholdsvis 6 og 31 uker. Ved avslutningen av broilerforsøket ble det funnet høyere selenverdier i leveren hos alle broilere som hadde fått selentilskudd enn hos kontrolldyrene. Likeledes ble det funnet en signifikant positiv sammenheng mellom nivåene av selen i lever og muskel og den mengden selen som ble tilsatt fôret. Ved avslutningen av forsøket med verpehøns ble det funnet signifikante økninger av selennivåene i lever, nyre og brystmuskel når fôret var tilsatt henholdsvis 1,0, 3,0 eller 6,0 µg Se/g. Signifikante positive sammenhenger ble funnet mellom nivåene av selen i lever, nyre og brystmuskel og den mengden selen som ble tilsatt fôret. For eggenes vedkommende ble det også funnet en tilsvarende økning i de homogeniserte eggprøvene ved økende selentilsetning. Økningen av seleninnholdet i plommen var langt større enn i hviten. Signifikante positive sammenhenger ble funnet mellom selennivåene i egg, plomme og hvite og den mengden selen som ble tilsatt i fôret. De forskjellige selentilsetningene hadde ingen effekt på tilveksten eller eggleggingen. En mulig positiv effekt på dyrs og menneskers helse ved å øke tilsetningen av selen i dyrefôret utover det nivået som blir anbefalt i dag er diskutert.

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