

Effects of barley-bound organic selenium compared with inorganic selenite on selenium concentration and structure of tissues in pig

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Abstract. A study was made of the effects of barley feeds containing varying levels of natural grain selenium derived from fertilizer and of the effects of sodium selenite on selenium concentrations in the tissues of growing pigs and on the histological structure of myocardium and longissimus muscle.

The results indicated that organic grain selenium affects the selenium levels of porcine tissues significantly more than sodium selenite supplementation of the same amount. The organic selenium concentrations of feed and organs were found to be linearly correlated. Spleen, lung and heart were the most reliable indicators of tissue selenium level in pigs.

The selenium concentration of muscles of pigs fed on naturally seleniferous feed (270 $\mu\text{g Se/kg DM}$) was at the internationally level (500 $\mu\text{g Se/kg DM}$ in meat), whereas the same amount of selenite in feed increased the selenium level of muscle only slightly (to 200 $\mu\text{g Se/kg DM}$).

The histological structures of heart and muscle of pigs fed on naturally seleniferous feed were normal, whereas in the selenite group there were signs of muscle dystrophy when the selenium level was below 200 $\mu\text{g Se/kg DM}$. In addition, the pigs given selenite were subsequently placed in a lower, fat-containing carcass class. Feed with a natural organic selenium level of 150—200 $\mu\text{g Se/kg DM}$ is preferred to selenite selenium supplement in pork production.

Introduction

The low selenium content in Finnish fodder plants (OKSANEN and SANDHOLM 1970, KURKELA 1982) makes it necessary to add selenium compounds to feeds during their production. Inorganic selenium supplements in

feed have not always had the desired effect. Nutritional muscular dystrophy (NMD) has occurred even when the selenium content of the feed has been 0.1 mg Se/kg DM.

Selenium-containing fertilizers can also be used to elevate the selenium content of feed. The changes in the selenium contents of

animal tissues resulting from the use of these fertilizers for growing feed grain should be investigated and the effects of elevated tissue selenium contents examined from the point of foodstuffs hygiene.

The object of the present study was to investigate the effects of feed with varying levels of organic and inorganic selenium on

- health
- organ selenium content
- macroscopic and histological structure of the muscles in the pig.

Materials and methods

Animals

Twenty-five Yorkshire pigs with an initial weight of 25 ± 0.5 kg were used in this study. They were divided into four groups (6 + 6 + 7 + 6). Each group received the test feed shown in Table 1 over a period of 110 days. In addition, samples for comparison were taken from the slaughterhouse from healthy pigs and from pigs with NMD.

Feed and feeding

Kemira Oy had grown barley with different selenium levels by fertilizing it one month after sowing with 0 to 1.0 kg sodium selenite per hectare (KORKMAN 1980). The feeds were mixtured from these barleys and protein mineral concentrate containing 40 % raw protein and 18.5 % minerals (Table 2) and from sodium selenite (Table 1). All the feeds used in the trial contained 30 IU/kg of vitamin E. All animals were given the group I feed for the four weeks leading up to the trial.

Table 2. Feed concentrate

Fish meal	30 %
Meat & bone meal	8
Soya	24
Fodder yeast	9
Wheat bran	5
Pig vitamin	3
Ground chalk	5
Fodder phosphate	4
NaCl	2.5
Lignox	4
Molasses	1

15 % of this concentrate was mixed with barley in connection with grinding.

The pigs were kept in sties with concrete floors. They obtained test feed and water freely from automatic dispensers.

Follow-up

The animals were examined clinically once a week. They were slaughtered after 110 days. The carcasses were examined visually at the time the meat was inspected and classified in the slaughterhouse.

Samples

Blood and faeces samples were taken at the end of the trial. Samples of urine and bile were taken after slaughtering, together with tissue samples from the spleen, liver, muscle (dorsal longissimus muscle), lung, kidney, pancreas, brain, CNS, eye and coccyx. The tissue samples were taken by cutting a sagittal or otherwise representative sample from the organ or from one of its lobes. A sample

Table 1. Selenium concentrations of experimental feeds

Trial group	Feed (grain Se conc.)	Total feed Se concentration $\mu\text{g}/\text{kg DM}$
I	Barley 10 $\mu\text{g}/\text{kg DM}$ + 15 % feed concentrate	100
II	Barley 10 $\mu\text{g}/\text{kg DM}$ + 15 % feed concentrate + Na_2SeO_3	270
III	Barley 180 $\mu\text{g}/\text{kg DM}$ + 15 % feed concentrate	270
IV	Barley 680 $\mu\text{g}/\text{kg DM}$ + 15 % feed concentrate	700

of myocardium was taken from the papillary muscle and the left ventricle wall.

Samples from equal-sized control pigs of the same strain from the same slaughterhouse used as random sample material were taken from the longissimus muscle and the myocardium both from clinically healthy animals and from those with nutritional muscular dystrophy (NMD).

The samples were frozen immediately and kept at -20°C until analysed. Histological samples from the muscle, myocardium, liver and kidney were stored in 10 % formalin.

Chemical analysis and histological examination

The method employed in the selenium determination was SAARI and PAASO's (1980) modification of the hydride method coupled with atomic absorption spectrophotometry. The lowest measurable selenium content by this method is $10\ \mu\text{g Se/kg}$.

Statistical analysis

The following methods were used in the statistical analysis of the results:

— The differences in tissue trace element contents between the feed groups were tested for homogeneity of variances by Bartlett's test, differences between means by one-way variance analysis and significantly different means were discriminated by Tukey's test.

— the dependence of tissue selenium level on feed selenium level was tested by linear regression analysis with feed selenium content as the systematic variable. Both the logarithmic function and the linear model were tested in the analysis.

— The correlations between the Se-levels of different organs were calculated using a correlation coefficient.

— Quality classification of the carcasses and slaughtering weights were tested using Fischer's accurate four-field test, one-way variance analysis and Tukey's test.

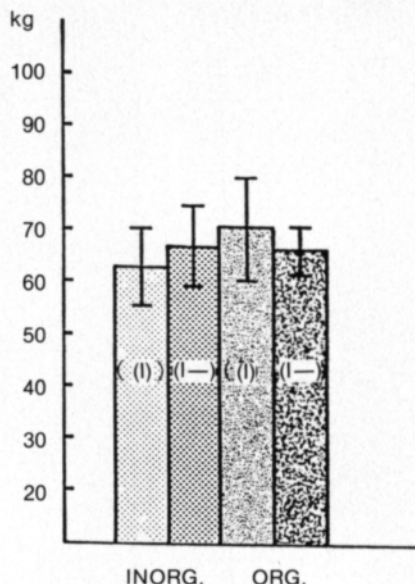


Fig. 1. Mean carcass weight and quality class

Results

The test pigs were clinically healthy during the tests. The mean slaughtering weights in the different groups are shown in Figure 1. No statistical differences were found in these.

In the quality classification, five pigs out of six in Group II were found to be fatty and were placed in class I—. The rest of the pigs were classified as belonging to class I (Figure 1).

The mean selenium contents of samples taken from test animals are shown in Table 3.

It was found that sodium selenite supplement in the basic feed ($270\ \mu\text{g Se/kg DM}$) in Group II did not statistically significantly elevate the selenium content of the muscle and other organs, whereas a corresponding selenium supplement in organic form (Group III) resulted in a statistically significant increase. In the muscle this difference in the elevation of the selenium contents was about 250 %.

The change in the organ selenium contents in Groups I, II and IV was linear. The dependence of muscle selenium content on the level

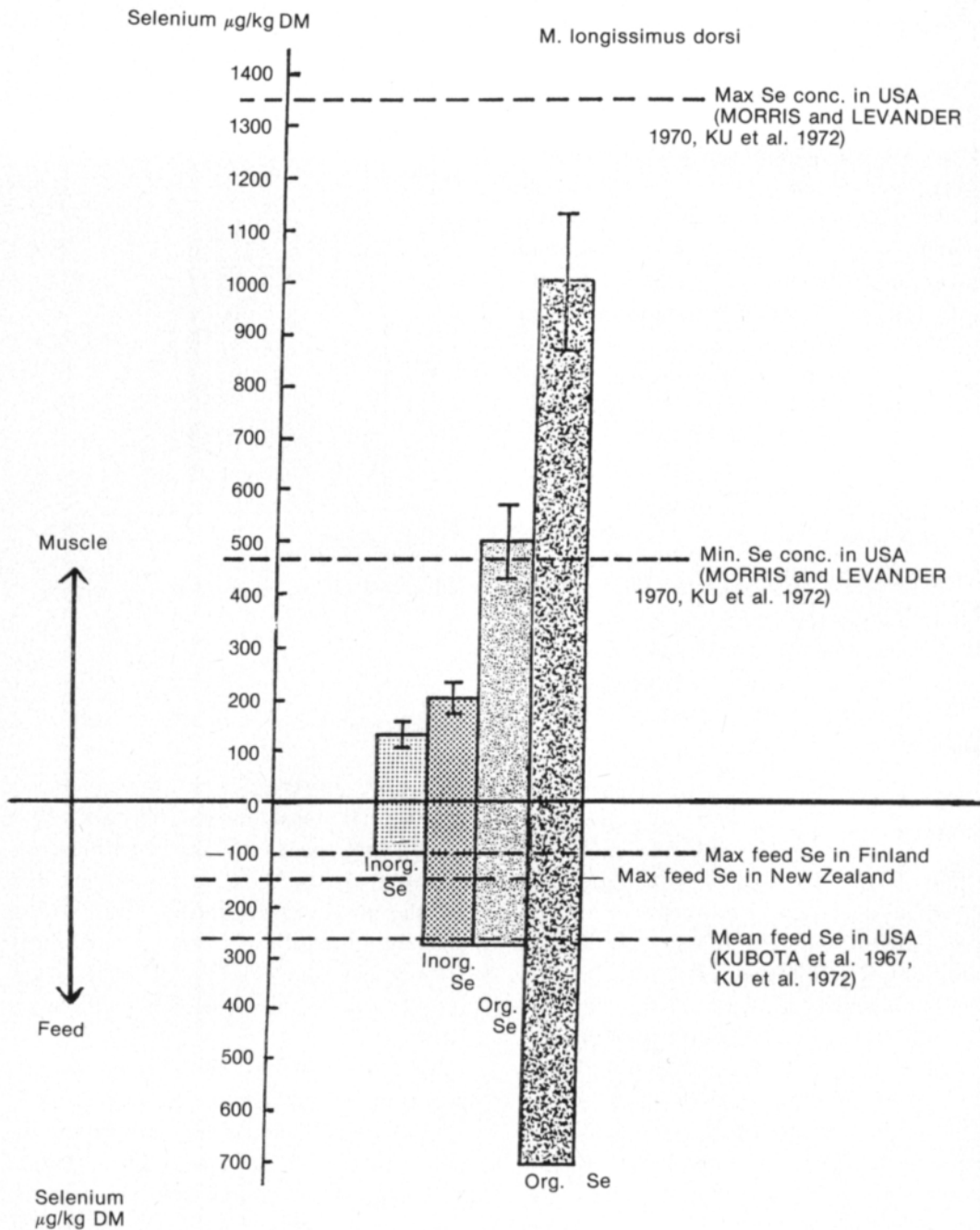


Fig. 2. Selenium content of muscle (*M. longissimus dorsi*) and associated selenium content in diet

of organic selenium in the feed is described by a curve of the second degree equation (Figure 3).

In the test groups, the selenium content of blood correlated best with the selenium con-

tents of the myocardium, liver, CNS and bile ($0.998 < r < 1$). Blood selenium content was only indicative of the selenium content of muscle. The most reliable indicator organs of the body selenium content were found to be

Table 3. Tissue selenium contents in test pigs ($\mu\text{g}/\text{kg DM}$, mean \pm SD)

Trial group Tissue	Trial			
	I	II	III	IV
Blood	550 \pm 64	566 \pm 47	614 \pm 63	1341 \pm 929
Spleen	1041 \pm 93	1125 \pm 121	1371 \pm 116	2200 \pm 360
Bone	141 \pm 34	191 \pm 34	414 \pm 115	541 \pm 263
Muscle	133 \pm 23	200 \pm 28	500 \pm 70	1016 \pm 134
Heart	683 \pm 23	733 \pm 131	1057 \pm 156	2550 \pm 150
Liver	1333 \pm 75	1617 \pm 227	1757 \pm 267	3433 \pm 320
Lung	691 \pm 53	766 \pm 79	950 \pm 96	1716 \pm 60
Kidney	5183 \pm 494	5666 \pm 1020	6128 \pm 832	8550 \pm 495
Pancreas		975 \pm 25	1325 \pm 375	
Brain	400 \pm 68	433 \pm 23	707 \pm 86	1450 \pm 150
Spinal cord	136 \pm 19	191 \pm 34	228 \pm 52	675 \pm 85
Eye	233 \pm 23	291 \pm 34	457 \pm 72	708 \pm 156
Bile, Combined sample	100	100	150	450
Urine, Combined sample	150	2100	1300	4700
Faeces, Combined sample	500	1800	750	1700

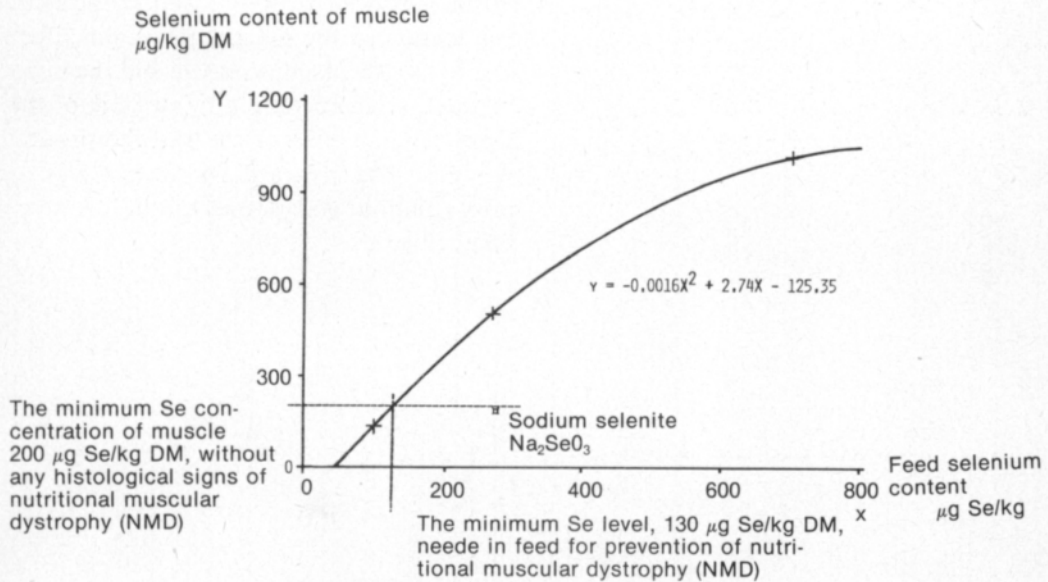


Fig. 3. Organic selenium in diet and associated selenium content of longissimus muscle of pig

the spleen and the lung, followed by the heart and the brain (Table 4).

The selenium content of the meat of clinically healthy pigs taken from the slaughterhouse and used as *control samples* was 150

$\mu\text{g Se}/\text{kg DM}$, and that of pigs with clinical signs of muscular dystrophy $378 \pm 79 \mu\text{g Se}/\text{kg DM}$. The corresponding selenium contents of the myocardium in clinically healthy pigs were $600 \mu\text{g Se}/\text{kg DM}$ and in pigs with

Table 4. The correlation matrix of selenium in the tissues examined.

	Blood	Spleen	Bone	Muscle	Myo- cardium	Lung	Kidney	Liver	Brain	Spinal cord	Eyes	Bile	Urine	Faeces
Blood	*													
Spleen	0	0												
Bone	0	*	*											
Muscle	**	**	0	*										
Myocardium	*	***	0	*	**									
Lung	*	**	0	*	*	**								
Kidney	**	**	0	*	*	**	**							
Liver	**	**	0	**	**	**	**	**						
Brain	*	***	0	**	**	**	**	**	*					
Spinal cord	**	*	0	*	**	**	**	**	**	0				
Eye	0	*	*	**	**	**	*	**	*	**	0			
Bile	**	**	0	*	***	**	*	**	*	**	0	0	0	0
Urine	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Faeces	0	0	0	0	0	0	0	0	0	0	0	0	0	0

0 = no significant correlation

* = almost significant correlation at 95 % certainty

** = significant correlation at 99 % certainty

*** = highly significant correlation at 99.9 % certainty

Corresponding correlation coefficients (r)

r = 0.950

0.950 r = 0.991

0.991 r = 0.998

0.998 r = 1

nutritional muscular dystrophy $641 \pm 159 \mu\text{g Se/kg DM}$, i.e. no statistically significant differences were found.

The mean selenium content of the meat of pigs with clinical muscular dystrophy was $378 \mu\text{g Se/kg DM}$, which deviated greatly from the corresponding means in Groups I, II and IV, while the mean selenium content of the myocardium, $641 \mu\text{g Se/kg DM}$, differed significantly from the mean selenium contents of Groups III and IV.

High biliary selenium contents were found in Groups III and IV, whereas in Group II it was the selenium content of the urine and faeces that was high. The biliary selenium contents were in agreement with the blood selenium contents and correlated significantly with the selenium contents of the myocardium and other internal organs.

Macroscopically, no changes were observed in the meat or organs of the test animals. Histologically in Groups I and II incipient degradation of the the myofibres was often seen in the longissimus muscle and the myocardium, as characterized by swelling of the myofibrils, absence of the sarcoplasm and myolysis. The myocardium showed loss of cross-striation and segmentation of myofibres (Figures 4, 5, 6).

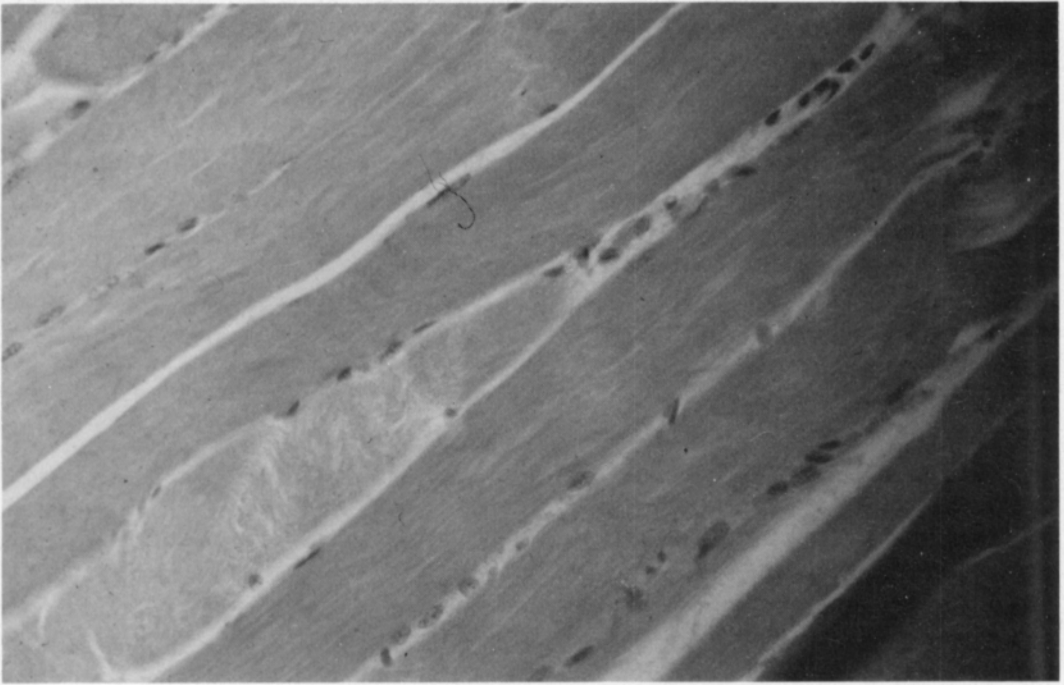


Fig. 4. Normal muscle, Se level over 200 μg Se/kg Dm



Fig. 5. Incipient muscular dystrophy (NMD), Se level 150–200 $\mu\text{g}/\text{Se}/\text{kg DM}$

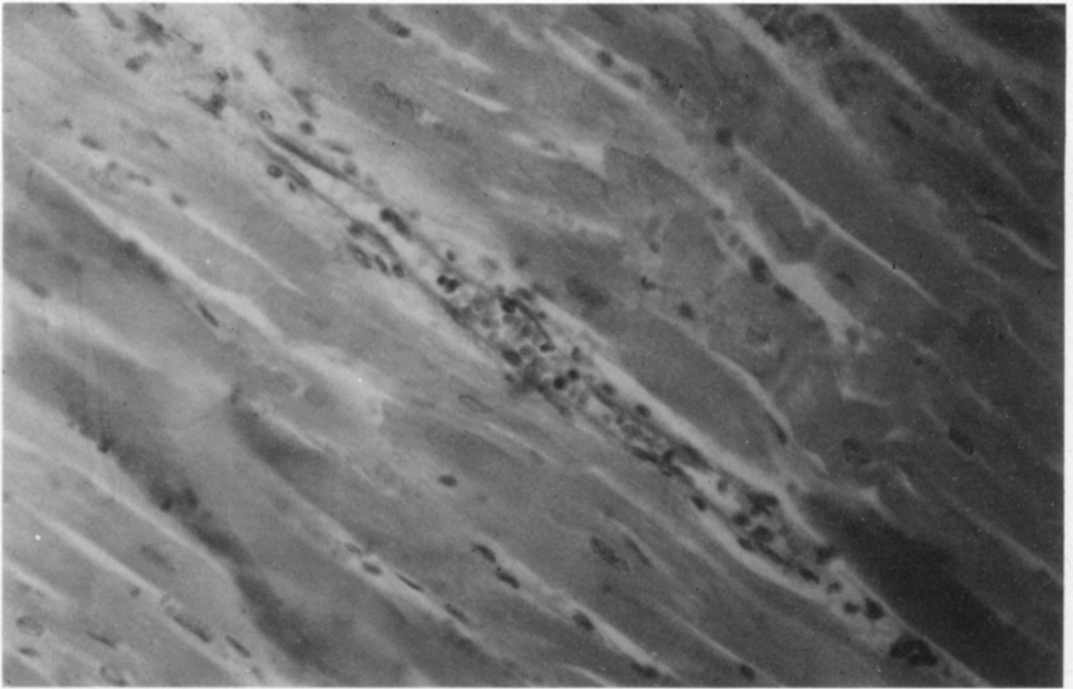


Fig. 6. Muscular dystrophy (NMD), Se level under 150 μg Se/kg DM

Similar changes due to nutritional muscular dystrophy were also found in clinically healthy pigs in the control group. The samples from Groups III and IV did not show any histological changes.

These degenerative muscular and myocardial changes were manifested progressively with increasing severity when the muscular selenium content fell to 200–150 μg Se/kg DM or below. In pigs with clinical nutritional muscular dystrophy, these histological changes were found in all samples of the longissimus muscle in which the selenium content was 200–450 μg Se/kg DM.

Progressively increasing degradation changes were observed in the myocardium of the pigs in Groups I and II and of the clinically healthy control animals when the myocardium selenium content fell to 600–700 μg Se/kg DM or below. The selenium content of the myocardium of pigs with clinical nutritional muscular dystrophy was 600–850 μg Se/kg DM.

Discussion

The clinical condition of the test animals was good during the trial. The selenium contents used did not adversely affect the health of the pigs. This was as expected, because the selenium contents of the test feeds were clearly between the selenium contents causing deficiency states (50 μg Se/kg DM) reported by LATSHAW *et al.* (1977), and the content found to be toxic in pigs (5000 μg Se/kg DM). The selenium content of the feed given to Groups II and III, 270 μg Se/kg DM, was of the same order as the mean natural selenium content of feed grain grown in the selenium-rich area of the USA (KUBOTA *et al.* 1967).

The different feeds used resulted in different selenium contents in the pig tissues. In Group I and in the healthy control group the selenium contents were 133 ± 23 μg Se/kg DM and 150 μg Se/kg DM, corresponding to the selenium content of Finnish pork reported by NUURTAMO *et al.* (1980), which in turn is lower than the selenium contents of foreign

pork, 450—1890 $\mu\text{g Se/kg DM}$ (LINDBERG 1968, MORRIS and LEVANDER 1970, KU et al. 1972, NORRMAN 1977).

In Group II (feed containing 270 $\mu\text{g Se/kg DM}$ as sodium selenite) the selenium content of the pork was 200 $\mu\text{g Se/kg DM}$. The elevation is small although the selenium content of the feed was three times greater than in Group I. In Group III, in which the feed contained the same amount of organic selenium of cereal origin, the selenium content of the muscle rose to 500 $\mu\text{g Se/kg DM}$ and in Group IV to 1000 $\mu\text{g Se/kg DM}$. The selenium level in the muscles was closely related to the organic dietary Se levels the pig received ($p < 0.001$).

The differences could also be seen in tissue selenium determinations in different organs: the difference between the biological effect of sodium selenite and organic selenium of cereal origin is shown in the selenium contents of Groups II and III. The different chemical compounds of selenium, organic and inorganic, have different metabolic pathways in the organism (CARY et al. 1973). Selenomethionine and selenocysteine are biologically very active in pig (KU et al. 1972). In plants such as wheat and barley, most of the selenium is incorporated into protein as selenomethionine (OLSON et al. 1970). In fish meal, selenium is known to be present as selenocysteine, the bioavailability of which has been found to be lower than that of selenomethionine.

Monogastric mammals cannot incorporate selenite selenium directly into cysteine or methionine, although the bacteria of intestinal tract, e.g. *E. coli*, can synthesize organoselenium compounds from selenite, which may explain the conversion of Se to nonlabile reduced forms *in vivo* (ALLAWAY 1973).

The selenium bound in wheat or barley in the form of selenomethionine has a better bioavailability than the same quantity of selenium bound in meat or fish meal as selenocysteine (KU et al. 1972). This is probably one reason for the low selenium content of Finnish pork, since the selenium content of

Finnish feed grain is low, 5—20 $\mu\text{g Se/kg DM}$, and the pig feed mixtures used contain meat and fish meal as the source of protein. In the USA, soya is usually used instead of these (KU et al. 1972).

It seemed that selenite selenium, selenomethionine and seleniferous plants (non accumulator species) may follow different metabolic pathways leading to differences in tissue concentrations of selenium, even though all these forms of Se are about equally effective in preventing selenium response diseases (CARY et al. 1973, JAAKKOLA et al. 1982).

Variance analysis showed the selenium levels in the blood samples to be highly significantly dependent ($p < 0.001$) on the feed selenium content, but in the regression analysis the dependence of blood selenium level on feed selenium content was only suggestive (90 %, regression coefficient 1.4). Contrary to the results obtained with chickens (KÄÄNTEE and KURKELA 1980), blood was not among the most reliable tissue indicators of body selenium content in the pig. The spleen, lungs, heart and brain were more reliable. The selenium contents of all these tissues rose quite high due to organic selenium in the feed.

In all test groups, the mean selenium contents of the organs were the highest in the kidneys, varying between 5200 and 8600 $\mu\text{g Se/kg DM}$; the next highest contents were in the liver, 1100—3500 $\mu\text{g Se/kg DM}$, myocardium, 680—2550 $\mu\text{g Se/kg DM}$, spleen, 1000—2200 $\mu\text{g Se/kg DM}$ and brain 400—1450 $\mu\text{g Se/kg DM}$. The lowest selenium content was found in the spinal cord, eye and bone. The greatest differences in the selenium contents between different test groups were found in the heart, liver, brain, spinal cord and bone.

The low selenium content of Finnish feeds is compensated during manufacture by the addition of sodium selenite, which has a low biological availability. This is shown by the incidence of histologically verified nutritional muscular dystrophy in Groups I and II, but also in healthy and unhealthy animals

from the slaughterhouse. High selenium contents were found in both the muscle and myocardium of these unhealthy animals. This may be due either to the selenium administered to the animals in the form of medication or to mobilization of selenium from the body to the diseased areas (TOIKKA 1978).

The statistical results obtained indicate (Figure 3) that to produce histologically healthy pork with a selenium content of more than 200 $\mu\text{g Se/kg DM}$, the content of organic selenium in the feed must be over 130 $\mu\text{g Se/kg DM}$.

Since neither the international selenium content of pork nor even the selenium level required for production of histopathologically healthy meat is obtained by the addition of sodium selenite, even up to 270 $\mu\text{g Se/kg DM}$, the study supports the idea that the only way to elevate the selenium content of Finnish pork to the international standard is to raise the organic selenium content of pork feed to at least 150–200 $\mu\text{g Se/kg DM}$ (i.e. 0.15–0.2 ppm). This can be done safely by fertilizing the feed grain by selenium-containing fertilizers or by foliar spraying with solutions containing selenium (KORKMAN 1980).

Considering the selenium levels in Table 2, and the average pork consumption of about 71 grammes per person per day in Finland (NUURTAMO et al. 1980) the range selenium per pig diet studied in the present investigation accounts for a difference of not more than 3–25 μg selenium in the average daily intake in man. This 3–25 μg is insignificant compared with the toxic level of 2400–3000 μg selenium per day previously stated (NRC 1976), although it is of greater importance in terms of the proposed daily adequate amount of 50–200 μg (NBH Circular No. 1762/1981). The toxic level could be exceeded with a daily consumption of more than about 1.1 kg pig kidney or more than about 2.1 to 2.6 kg liver, from pigs whose diet contains between 100 and 700 $\mu\text{g Se/kg DM}$ (0.1–0.7 ppm).

Summary

An investigation was made into the effects of barley feeds with varying levels of natural organic selenium introduced by means of selenium-containing fertilizer and of inorganic sodium selenite on the concentrations of selenium in the tissues of growing pigs (blood, spleen, bone, muscle, heart, liver, lung, kidney, pancreas, brain, spinal cord and eye), and on the histological structure of myocardium and longissimus muscle.

The results indicated that natural organic grain selenium affects the selenium levels of tissues in pig significantly more (muscle 250 %) than sodium selenite supplementation of the same amount. The natural selenium concentrations of feed and organs were linearly correlated. Spleen, lung and heart were the most reliable indicators of tissue selenium levels in pigs.

When the feed selenium level was supplemented to 100 $\mu\text{g Se/kg DM}$ (0.1 ppm) with sodium selenite, the selenium concentration of longissimus muscle was $133 \pm 23 \mu\text{g Se/kg DM}$. The selenium concentrations of muscles of pigs fed on naturally seleniferous feed, 270 $\mu\text{g Se/kg DM}$ were at the normal international level (500 $\mu\text{g Se/kg DM}$ in meat), whereas the same feed selenite selenium, 270 $\mu\text{g Se/kg DM}$, increased the selenium level of muscles only slightly (to 200 $\mu\text{g Se/kg DM}$).

The histological structures of heart and striated muscles of the pigs fed on 270 $\mu\text{g Se/kg DM}$ organic selenium feed were normal and healthy, whereas in the group given the same amount of selenite there were histological signs of muscle dystrophy (NMD). A grain selenium concentration of 700 $\mu\text{g Se/kg DM}$ did not have any adverse effect on experimental animals.

In the quality classification of the carcasses, 5/6 pigs in the group receiving sodium selenite were placed in Class I- because of their high fat content.

The study supports the idea that natural organic selenium of grain origin of 150–

200 μg Se/kg DM (0.15—0.2 ppm) is the most suitable for pork production and more effective than selenite-induced selenium content

both as regards the health of the animals and the elevation of the selenium content to the normal international level.

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Ohran orgaanisen seleenin ja epäorgaanisen seleniitin vaikutuksista sian elimistön seleenipitoisuuksiin ja lihaksiston rakenteeseen.

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Tutkimuksessa tarkastellaan ohran seleenin ja epäorgaanisen seleniitin vaikutuksia kasvavien sikojen elimistön seleenipitoisuuksiin sekä sydänlihaksen ja selkälihasten histologiseen rakenteeseen.

Tutkimukseen käytettiin 25 sikaa jaettuna neljään ryhmään sekä vertailuaineistona terveitä ja lihasrappeumaa sairastavia teurassikoja teurastamosta. Koeryhmiä ruokittiin 110 vrk:n ajan rehuilla joiden Se pitoisuudet olivat 100 µg—700 µg/kg.

Tulokset osoittivat että viljan orgaaninen seleeni kohotti porsaiden elimistön Se pitoisuutta huomattavasti enemmän kuin vastaava määrä natriumseleniittiä. Rehun seleenin ollessa viljaperäistä, 270 µg/kg, kohosi lihan Se pitoisuus tasolle 500 µg/kg kuiva-ainetta, rehun

seleenin ollessa natriumseleniittiä, 270 µg/kg, oli lihan Se pitoisuus vain 200 µg/kg kuiva-ainetta.

Porsaan elimistön Se tason parhaat ja luotettavimmat indikaattorit olivat pernan keuhkojen ja sydämen Se pitoisuudet.

Histologisesti todettiin ohraseleeniä saaneiden porsaiden lihakset ja sydämet terveiksi, kun taas seleniittiporsaille lihasten Se pitoisuuden alittaessa 200 µg/kg kuiva-ainetta ilmeni lihasrappeumaa.

Tulokset osoittivat ohraan sidotun seleenin kohottavan elimistön Se tasoa enemmän kuin vastaavat määrät natriumseleniittiä ja suojaavan elimistöä paremmin lihasrappeumalta.