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INFLUENCE OF DIETARY SODIUM SELENITE ON TISSUE SELENIUM LEVELS OF GROWING PIGS

By

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MOKSNES, KNUT, SVERRE TOLLERSRUD and HANS JØRGEN LARSEN: *Influence of dietary sodium selenite on tissue selenium levels of growing pigs.* Acta vet. scand. 1982, *23*, 361—367. — Twenty Norwegian Landrace pigs were divided into 5 groups and fed a basal diet consisting of a mixture of dried skim milk and whey powder together with ground barley. The diet was supplemented with 0, 0.2, 0.8, 1.2 and 2.2 μg/g selenium as sodium selenite and was fed for 12 weeks. The muscle selenium level was increased by a factor of about 4 and the liver selenium by a factor of about 12 when the dietary selenium supplement was increased from zero to 2.2 μg/g. There was a significant linear correlation between dietary selenium and selenium concentrations in tissues. Possible benefit for humans consuming meat from animals having received the selenium doses used in this experiment are discussed.

dietary selenium; tissue levels; pigs.

Selenium — vitamin E deficiency in pigs is a nutritional disease showing one or more of the following symptoms: sudden death among fast growing animals, hepatic necrosis (hepatosis dietetica), degeneration of skeletal muscles, cardiac muscular degeneration or microangiopathy (mulberry heart disease) (*Grant* 1961).

Successful prophylactic control and therapeutic procedures are necessary to allow maximum profitable pig production in areas of the world where the soil is low in selenium.

Selenium moves continuously through a soil — plant — animal — man cycle. The cycle could be manipulated to supply adequate amounts of selenium to animals in deficient regions

by fertilizing soils with selenium salts, by supplementing animal rations with selenium compounds, by feeding foodstuffs naturally high in selenium (e.g. fish meal), or by giving parenteral injections of selenium to the animal.

To prevent selenium deficiency conditions, supplementation of selenium to animal feedstuffs is now permitted in a few areas of the world e.g. New Zealand, USA and Scandinavia.

As most feedstuffs of Norwegian origin are found to be extremely poor in selenium (*Frøslie et al.* 1980), this element has been added to all commercial concentrate mixtures in Norway since 1980 at the rate of 0.1 μg/g as sodium selenite (*Landbruksdepartementet* 1979).

The present experiment was designed to test the effect of different levels of selenium supplementation in the diet to growing pigs. A further intention was to evaluate the risk of human consumption of meat from these animals.

MATERIAL AND METHODS

Animals and feeding

Twenty Norwegian Landrace pigs were grouped according to live weight and sex into 5 groups, each consisting of 4 pigs. The initial mean weight was 21 kg. The experiment lasted for 12 weeks, live weight at slaughter being 95 kg. Two pigs died during the experimental period, 1 in Group 2 during blood collection and 1 in Group 4 due to swine dysentery.

The pigs were fed individually by hand twice daily. The basic diet consisted of a mixture of dried skim milk and whey powder blended with ground barley. The dried skim milk/whey fraction was kept at a constant level throughout the experiment, 0.5 kg per animal per day, whereas the amount of ground barley was increased every second week according to live weight, from 0.5 kg at the start to 2.3 kg at the end of the experiment. The ground barley was supplemented with 20 g dicalcium phosphate, 3.000 I.U. vitamin A and 300 I.U. vitamin D_3 per kg.

The mean selenium content of the feed components, as determined by analysis using a neutron activation method (*Steinnes* 1967), was 77 μg/kg for dried skim milk/whey powder, and 28 μg/kg for ground barley, inclusive of minerals and vitamins. The individual daily intake of selenium from the basic feed was thus 53 μg, corresponding to 0.05 μg Se/g feed, at the start of

the experiment and 103 μg, corresponding to 0.04 μg Se/g, at the end of the experiment.

Analysis of α-tocopherol was carried out using a spectrophotomertic method (*Lambertsen & Brækkan* 1959). The average α-tocopherol content of the basic feed was 18 μg/g. The daily intake of α-tocopherol from the basic ration thus ranged from 9 mg at the start to 41 mg at the end of the experiment.

The pigs in the control group (Group 1) were fed exclusively on the basic diet whereas the other 4 groups were given oral supplements of selenium as shown in Table 1. The supplement, a solution of sodium selenite, was increased gradually together with the amount of feed every second week. It was administered individually once daily with the feed.

At the end of the experiment the animals were slaughtered and examined for lesions, and tissue samples were obtained for histopathological examination and selenium analyses.

Analytical methods

Samples of liver and part of leg muscle were collected from each pig and frozen at —20°C immediately after collection. The tissue samples were analysed according to a modification of a 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

Supplementation of sodium selenite to diets low in selenium increased the selenium level in tissues of growing pigs (Table 1). The levels in liver increased relatively more than those in muscle in response to dietary selenite. Significant correlations were found between the amount of selenium (μg Se/g dry weight) added to the ration (x) and the selenium concentration (μg Se/g wet weight) in liver (y_1) and muscle tissue (y_2). The regression equations for liver and muscle were

$y_1 = 0.62x + 0.18$ ($r = 0.97$, $P < 0.001$), and

$y_2 = 0.065x + 0.066$ ($r = 0.93$, $P < 0.001$), respectively.

No gross lesions were observed at slaughter. Histopathological examination of the skeletal muscles showed signs of hyaline degeneration in the control group.

Table 1. Selenium concentrations (mean and range) of liver and muscle from pigs fed a basal diet supplemented with sodium selenite at different levels for 12 weeks. Data for total daily intake of selenium refer to start and termination of the experiment.

Pig group	Supplement of Se µg/g	Total daily intake of Se µg	µg Se/g (wet weight)	
			liver	muscle
1 (n = 4)	0	53—103	0.13 (0.11—0.16)	0.05 (0.05—0.05)
2 (n = 3)	0.2	253—663	0.49 (0.45—0.51)	0.09 (0.08—0.09)
3 (n = 4)	0.8	853—2343	0.65 (0.58—0.70)	0.12 (0.11—0.15)
4 (n = 3)	1.2	1253—3463	0.85 (0.83—0.91)	0.17 (0.16—0.19)
5 (n = 4)	2.2	2253—6263	1.6 (1.3 —1.8)	0.20 (0.18—0.21)

No significant effect of selenium supplementation was observed with respect to growth rate, feed efficiency and health.

DISCUSSION

There is a relationship between the amounts of selenium in feed and selenium levels in liver and muscle tissue of growing pigs. The muscle selenium level increased by a factor of about 4 and the liver selenium by a factor of about 12 when the dietary selenium supplement increased from zero to 2.2 µg/g. There was a significant linear correlation between dietary selenium and subsequent tissue concentrations. This is in agreement with previous studies by *Ku et al.* (1972) and by *Moksnes & Norheim* (1982) concerning chickens.

The muscle and liver selenium concentrations reported here are lower than the values given by *Ku et al.* (1973), but higher than the findings of *Ewan* (1971), *Groce et al.* (1971) and *Rasmussen* (1974). This may be due to many factors, such as the age of the pigs, different concentrations of natural selenium in the basic diet, length of the experimental period and weight of the pigs at slaughter.

Groce et al. (1973) postulated the existence of selenium thresholds in the tissues and serum of pigs. They suggested that once physiological storages of selenium are filled, excess of the

element is excreted and serum and tissue levels stabilize. Their data indicated that hepatic and skeletal muscle concentrations reach a maximum at 0.1 μg/g supplemented selenium. Our results from liver analyses showed no such trend, even at a dietary level of 2.2 μg Se/g. Similarly, *Rasmussen* (1974) could not show a leveling off in liver selenium concentrations at a dietary level of 1.0 μg Se/g.

Even 2.2 μg/g selenium in the diet showed no harmful effect on pig performance, and gave no signs of toxic effects. This agrees with previous studies by *Herigstad et al.* (1973). Two μg Se/g is suggested as a maximum dietary tolerable level for all animal species (*National Academy of Sciences* 1980).

Pigs fed the unsupplemented basal diet developed hyaline degeneration of the skeletal muscle which is one of the 3 main post mortem findings of selenium — vitamin E deficiency in pigs. This is in agreement with previous studies which implies that cardiac and skeletal muscular lesions are produced more readily than the hepatic form (*Sharp et al.* 1972 and *Van Vleet et al.* 1975).

Human consumption of meat from animals having received the selenium doses used in this experiment means a small increase of the daily selenium intake. Skeletal muscle is of special interest as this is the main tissue being used for human consumption. The highest values found here are in the order of 0.20 μg Se/g muscle (wet weight). This extra intake of selenium would be a positive contribution to the human selenium intake in Norway (*Moksnes & Norheim* 1982).

Pigs given naturally occurring selenium, being mostly selenomethionine, reach higher tissue selenium levels than pigs given sodium selenite (*Ku et al.* 1972, *Gissel-Nielsen* 1980). Selenomethionine is also less toxic than sodium selenite, at least to the rat (*Fishbein* 1977). If one intends to increase the selenium level in pork in order to enhance the human consumption of this element, selenomethionine would be more effective than sodium selenite. For this purpose selenomethionine would also give a larger safety margin when adding selenium to feedstuffs.

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SAMMENDRAG

Effekten av natriumselenitt i fôret på selennivåene i organer hos gris.

Tyve griser med en gjennomsnittsvekt på 21 kg ble delt i 5 grupper og fôret med et grunnfôr bestående av en blanding av skummet tørrmelk og mysepulver blandet med byggrøpp. Fôret ble tilsatt henholdsvis 0, 0.2, 0.8, 1.2 og 2.2 μg Se/g som natriumselenitt og det ble fôret i 12 uker. Gjennomsnittsvekten ved slakting var 95 kg. Selennivået i muskulaturen økte med en faktor på omtrent fire og selennivået i lever med en faktor på omtrent tolv når selentilskuddet i fôret økte fra null til 2.2 μg/g. Det ble funnet signifikante sammenhenger mellom mengden selen tillsatt i fôret og selennivåene i lever og muskel. Grisene i kontrollgruppen utviklet muskeldegenerasjon. Humant konsum av kjøtt fra griser som har fått de selendosene som er blitt benyttet i dette forsøket er diskutert.

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