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From the Institute of Internal Medicine and the Department for Anatomical Pathology, Royal Veterinary and Agricultural University, Copenhagen, the Department for Experiments with Pigs and Horses, National Institute of Animal Science, Copenhagen, the Risø National Laboratory, Roskilde, and the Division of Chemical Determination of Nutrients, National Food Institute, Søborg, Denmark

SELENIUM AND VITAMIN E DEFICIENCY IN PIGS

II. INFLUENCE ON PLASMA SELENIUM, VITAMIN E, ASAT AND ALAT AND ON TISSUE SELENIUM*

By

M. G. Simesen, H. E. Nielsen, V. Danielsen, G. Gissel-Nielsen, W. Hjarde, T. Leth and *A. Basse*

SIMESEN, M. G., H. E. NIELSEN, V. DANIELSEN, G. GISSEL-NIELSEN, W. HJARDE, T. LETH and A. BASSE: *Selenium and vitamin E deficiency in pigs. II. Influence on plasma selenium, vitamin E, ASAT and ALAT and on tissue selenium.* Acta vet. scand. 1979, *20*, 289—305. — The effect of dietary selenium (Se) and vitamin E (Vit. E) in pigs on Se and Vit. E in plasma and on Se in tissue from liver, heart, m. long. dorsi and m. psoas major was studied; and furthermore was the influence on the enzymes ASAT and ALAT studied.

Two levels of Se were used, 0.03 and 0.06 mg Se per kg feed. Within each Se level 2 levels of Vit. E were used, 15 and 45 i. u. per kg feed. This resulted in 4 groups: 1. low Se and low Vit. E; 2. low Se and high Vit. E; 3. high Se and low Vit. E; 4. high Se and high Vit. E.

Ten % of all pigs fed low Se, and 4 % of the pigs fed low Se and high Vit. E diet died with severe symptoms of Se deficiency. None of the pigs fed the high Se diet died with such symptoms. Plasma Se determinations have been shown to indicate the Se status in pigs almost as accurately as liver Se determination. ASAT and ALAT enzyme determinations were not of any diagnostic value.

There was a good agreement between dietary Vit. E level and the corresponding levels in plasma. Oxidized herring oil seems to enhance the Vit. E need.

selenium; vitamin E; hepatosis dietetica; mulberry heart disease; selenium in plasma and tissue; vitamin E in plasma; ASAT; ALAT; pigs.

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Selenium/vitamin E deficiency has been described in most animal species. The Nordic countries are among the geographical areas, where the Se/Vit. E deficiency syndrome is commonly seen. In pigs, the 3 main clinical manifestations are hepatosis dietetica (HD), mulberry heart disease (MHD) and nutritional muscular degeneration (NMD), occurring alone or in combination. The lesions are associated with combined Se/Vit. E deficiency in the young growing pig. It has been possible to reproduce these clinical and pathological manifestations in experiments of relatively short duration (*Lannek & Lindberg* 1975). It remains, however, to demonstrate genuine Se deficiency during adequate Vit. E supplementation, and vice versa. In birds the development of advanced Se deficiency during Vit. E sufficiency has been shown to require 2 generations (*Thompson & Scott* 1967).

The purpose of these experiments has been over a period of 2 generations to try to produce Se deficiency or Vit. E deficiency, or the combination of these two.

MATERIAL AND METHODS

The trial started in 1972 and was completed in 1976.

Feed

Two levels of Se were used, 0.03 and 0.06 mg Se per kg feed. Within each level of Se, 2 levels of Vit. E were used, 15 and 45 i. u. per kg feed (for details see *Nielsen et al.* 1979).

Animals

Sixty piglets were weaned at 3 weeks of age. Twenty-eight castrated males and 32 females were distributed into groups for 4 different diets.

The female pigs were mated, the castrated males slaughtered at approx. 85 kg live weight. Two sows from each of the 4 groups were selected for further mating. When possible a total of 4 litters were taken from each of these sows. For details of this breeding program and animal caretaking see *Nielsen et al.* (1979).

All pigs that died during the trial were necropsied.

Specimens of blood (plasma), liver, heart muscle, skeletal muscle (m. long. dorsi and m. psoas major) and kidney were

collected at slaughter and kept frozen until determinations of Se and Vit. E., *ASAT** (EC 2.6.1.1.) and *ALAT*** (EC 2.6.1.8.) were performed. The Se analyses were done in duplicate, using the fluorometric procedure described by *Olson* (1969). The Vit. E determinations were done according to *Hjarde et al.* (1973).

Transaminase determinations on plasma from the 1st generation of pigs were done ad modum *Morgenstern et al.* (1966) (ASAT) and *Reitman & Frankel* (1957) (ALAT). For samples from the 4 litters from the 2nd generation the transaminase determinations (ASAT and ALAT) were made according to *Keiding et al.* (1973).

Se, Vit. E, ASAT and ALAT concentrations were determined on plasma. Tissue samples were used for Se analysis only.

From the 1st generation and from the 1st and 2nd litters of the 2nd generation, plasma samples for Se, Vit. E, ASAT and ALAT determinations were drawn at the age of 3, 9, 15 and 21 weeks and at slaughter. From the 3rd and 4th litters, plasma samples were taken only at the age of 3 weeks and at slaughter.

RESULTS

The number of stillborn pigs as well as the number of deaths before the age of 3 weeks were highest in the groups with low Vit. E regimen (Groups 1 and 3). Between the age of 3 and 8 weeks deaths occurred only in the groups with low Se regimen (Groups 1 and 2). Detailed reports concerning the growth and feed consumption have been published (*Nielsen et al.* 1979), and descriptions of the gross and microscopic pathological lesions observed are to be published (*Basse et al.*).

Se concentrations in plasma

The plasma Se values in the pigs constituting the 1st generation started out with concentrations from 0.050 to 0.065 μg/g. During the first 15 weeks of the trial, the plasma Se concentrations for Groups 1 and 2 (low Se regimen) declined to about 50% and stayed at this level until slaughter. Groups 3 and 4

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* ASAT (Aspartate amino transaminase) formerly called SGOT — approximate conversion factor 4.05.

** ALAT (Alanine amino transaminase) formerly called SGPT — approximate conversion factor 2.50.

(high Se regimen) exhibited plasma values near the starting level, until about the age of 15 weeks. From then on, the plasma Se concentration increased moderately until slaughter (Table 1).

Table 1. Survey of Se plasma concentrations in the 1st generation (μg/g± 2 s).

Week \ Group	1	(n)	2	(n)	3	(n)	4	(n)
3	0.051±0.012	(12)	0.062±0.028	(13)	0.061±0.016	(13)	0.047±0.016	(13)
9	0.040±0.016	(15)	0.031±0.014	(14)	0.045±0.012	(15)	0.047±0.014	(13)
15	0.021±0.010	(15)	0.035±0.022	(14)	0.051±0.012	(15)	0.047±0.012	(12)
21	0.024±0.016	(15)	0.028±0.014	(14)	0.067±0.026	(15)	0.067±0.020	(13)
At slaughter	0.059±0.050	(7)	0.038±0.034	(6)	0.116±0.052	(8)	0.131±0.050	(7)

The plasma Se concentrations in the 4 groups are shown in Table 2 for the 2nd generation, 1st and 2nd litters. The plasma Se values differed in the 4 groups already from the very first sample. The low Se groups had plasma Se concentrations between 0.020 and 0.030 μg/g, whereas the high Se groups had plasma Se values between 0.040 and 0.060 μg/g. The nadir in the low Se groups was at the age of 15 weeks with plasma Se values between 0.015 and 0.020 μg/g. Between 15 weeks and slaughter, the plasma Se values for Groups 1 and 2 stayed almost constant. Groups 3 and 4, on the other hand, showed a moderate increase in plasma Se values. The plasma Se levels in these 2 groups were about twice as high as in Groups 1 and 2.

Table 2. Survey of Se plasma concentrations 1st and 2nd litters (μg/g± 2 s).

Week \ Group	1	(n)	2	(n)	3	(n)	4	(n)
3	0.026±0.016	(24)	0.023±0.012	(32)	0.050±0.020	(24)	0.043±0.012	(28)
9	0.019±0.010	(23)	0.023±0.012	(31)	0.041±0.014	(24)	0.038±0.022	(28)
15	0.017±0.012	(24)	0.019±0.012	(29)	0.036±0.018	(24)	0.030±0.020	(28)
21	0.021±0.016	(23)	0.017±0.012	(29)	0.045±0.018	(24)	0.044±0.026	(28)
At slaughter	0.034±0.034	(20)	0.031±0.022	(27)	0.077±0.028	(24)	0.081±0.032	(26)

In the 3-week sample from the 3rd litter the Se concentrations were about 0.030 μg/g for Groups 1 and 2. In the 4th litter, the plasma Se concentration in Groups 1 and 2 were 0.020 μg/g.

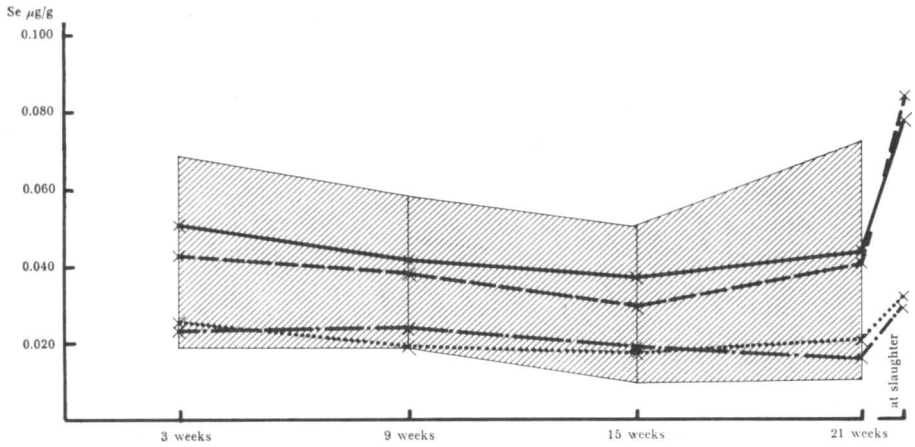

Figure 1. Plasma Se concentrations in growing pigs. Normal range ////////, mean Se ± 2 s for Group 4. Group 1 × .... ×, Group 2 × —.—.— ×, Group 3 × ——— ×, Group 4 × — — — ×, mean Se values, 1st and 2nd litter.

Mean values ± 2 s of plasma Se are calculated from all plasma samples taken from Group 4 (3 weeks to slaughter) and compared to the mean values of Groups 1, 2, 3 and 4 (Fig. 1).

Table 3 gives a regression analysis relating the Se content in piglets' plasma at 3 weeks of age to the selenium content in sow colostrum and sow milk. As can be seen there is a fair correlation between these (r = 0.58 and 0.66).

Table 3. Regression analysis relating Se concentration in piglets' plasma at 3 weeks of age to Se in sow colostrum and sow milk.

	b_0	b_1	r	P
Colostrum	0.016	0.0003	0.58	0.0001
Sow milk	0.012	0.0012	0.66	0.001

b_0 = intercept.
b_1 = regression coefficient.
r = correlation coefficient.
P = level of significance.

Se content of different tissues

The liver Se values in Groups 1 and 2 were lowest in pigs from the 2nd generation, 1st litter (Table 4). For Groups 1 and

Table 4. Se concentration in liver tissue (μg/g wet weight ± 2 s).

Group	1	(n)	2	(n)	3	(n)	4	(n)
1st generation	0.116±0.072	(7)	0.104±0.050	(7)	0.251±0.050	(7)	0.300±0.193	(7)
2nd generation								
1st litter	0.074±0.034	(19)	0.084±0.026	(25)	0.191±0.096	(13)	0.206±0.132	(19)
2nd litter	0.121±0.024	(4)	0.099±0.077	(4)	0.167±0.050	(8)	0.184±0.036	(8)
3rd litter	0.095±0.018	(6)	0.156±0.075	(5)	0.183±0.038	(6)	0.250±0.072	(5)
4th litter	0.109±0.032	(5)	0.152±0.180	(4)	0.199±0.040	(9)	0.227±0.070	(4)
Av. 2nd generation	0.103		0.119		0.200		0.234	

2, 2nd litter, the average liver Se values increased slightly. The average liver Se value for Group 1 for all litters was less than half of the liver Se values for Groups 3 and 4. The average liver Se concentration for Group 2 was also lower than for Groups 3 and 4. For the high Se groups (3 and 4), the liver Se concentrations stayed around 0.200 μg/g with a tendency to slightly higher Se values in Group 4.

The Se concentration in heart muscle was about half of the Se content in the liver tissue in all 4 groups and all litters. For the Se deficient groups, the Se concentration declined to a level about 0.060 μg/g, compared with about 0.120 μg/g in the high Se groups (Table 5).

Table 5. Se concentration in heart muscle (μg/g wet weight ± 2 s).

Group	1	(n)	2	(n)	3	(n)	4	(n)
1st generation	0.064±0.046	(8)	0.065±0.030	(7)	0.136±0.046	(7)	0.164±0.114	(6)
2nd generation								
1st litter	0.046±0.034	(19)	0.055±0.024	(25)	0.117±0.095	(16)	0.120±0.086	(20)
2nd litter	0.075±0.026	(4)	0.055±0.056	(4)	0.086±0.020	(8)	0.101±0.008	(8)
3rd litter	0.055±0.022	(6)	0.088±0.048	(5)	0.114±0.046	(6)	0.134±0.034	(5)
4th litter	0.063±0.022	(5)	0.094±0.085	(4)	0.107±0.046	(8)	0.117±0.054	(4)
Av. 2nd generation	0.061		0.071		0.112		0.128	

The Se concentration of muscle was about ⅓ that of liver in all 4 groups and all litters. In the Se deficient groups the Se concentration was about half of the Se concentration in the high Se groups (Table 6).

Table 6. Se concentration (av. double estimations M. long. dors. and M. psoas major) (μg/g wet weight ± 2 s).

Group	1	(n)	2	(n)	3	(n)	4	(n)
1st generation	0.038±0.026	(9)	0.045±0.034	(7)	0.082±0.036	(9)	0.080±0.036	(7)
2nd generation								
1st litter	0.030±0.018	(19)	0.036±0.024	(27)	0.059±0.018	(16)	0.058±0.026	(20)
2nd litter	0.047±0.020	(4)	0.037±0.018	(4)	0.060±0.022	(8)	0.061±0.018	(8)
3rd litter	0.033±0.012	(6)	0.033±0.017	(5)	0.054±0.030	(6)	0.083±0.038	(5)
4th litter	0.032±0.010	(5)	0.040±0.030	(4)	0.058±0.020	(8)	0.069±0.028	(4)
Av. 2nd generation	0.036		0.038		0.063		0.070	

Vit. E concentrations

The levels of Vit. E in plasma are closely associated with the 2 levels of supplementation (Fig. 2). Until the age of 3

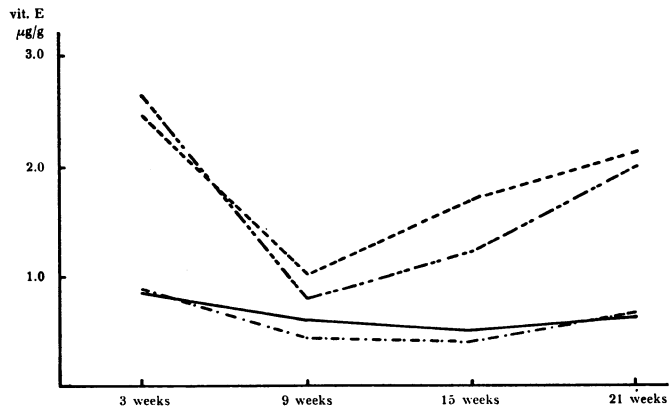

Figure 2. Survey of Vit. E plasma concentrations. Group 1 ——, Group 2 - - - -, Group 3 — - — -, Group 4 — - - — - -, mean values, 1st and 2nd litter.

weeks, the plasma concentrations in the Vit. E supplemented groups (2 and 4) remained at a nearly constant level, 3 times higher than the Vit. E level in the non-supplemented groups (1 and 3). Around the age of 9 weeks, the plasma levels of Vit. E had declined to about twice that of the non-supplemented groups (Table 7).

Table 7. Survey of Vit. E plasma concentrations from 1st and 2nd litters (μg/g).

Week \ Group	1 (n)	2 (n)	3 (n)	4 (n)
3	0.85 (3)	2.48 (3)	0.88 (4)	2.65 (4)
9	0.60 (2)	1.03 (3)	0.45 (4)	0.80 (4)
15	0.50 (3)	1.70 (3)	0.43 (4)	1.23 (4)
21	0.63 (3)	2.13 (3)	0.65 (4)	2.00 (4)

The average Vit. E levels in the 1st and 2nd litters, non-supplemented groups, were almost the same, whereas the Vit. E levels were clearly lower in the 2nd litter than in the 1st litter, Vit. E supplemented groups (Fig. 3). This may probably be explained by the fact that herring oil was given to 2nd-litter piglets from the age of 8 weeks to about 50 kg of live weight.

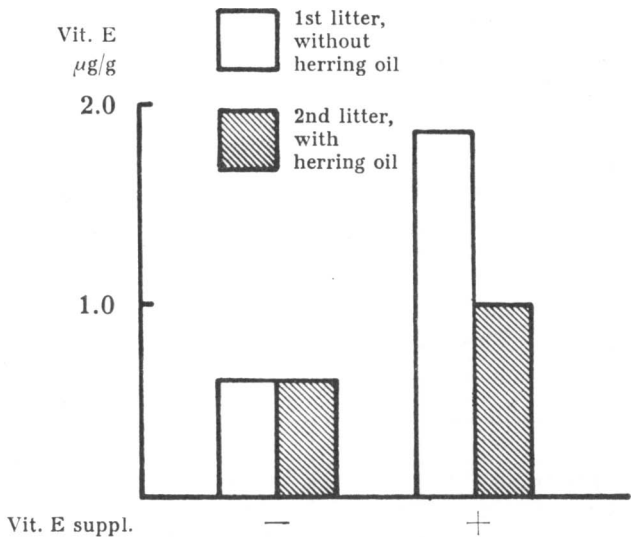

Figure 3. Vit. E content in plasma from pigs without (1st litter) and pigs with (2nd litter) addition of 2 % oxidized herring oil in the feed.

Transaminases

Using the plasma transaminase concentrations from Groups 3 and 4 (the Se-supplemented groups) as normal material for the 4 different age categories (3, 9, 15 and 21 weeks), the nor-

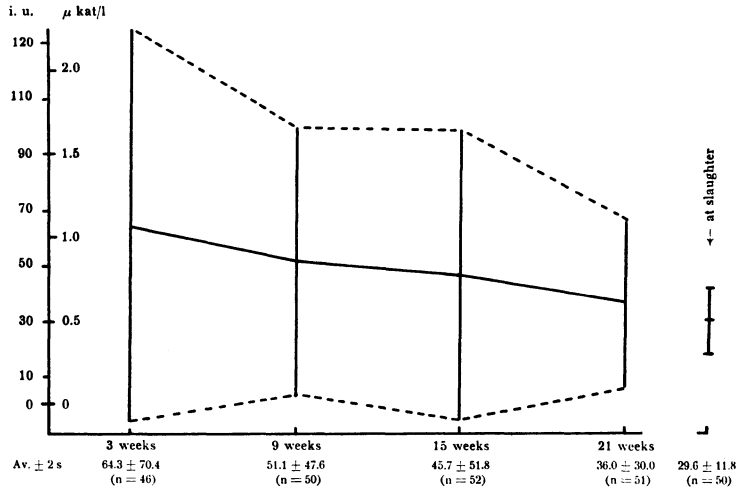

Figure 4. Normal plasma ASAT values in growing pigs. Mean ± 2 s, Groups 3 and 4.

mal range was defined as the mean value ± the double standard deviation (Figs. 4 and 5).

Compared to these values, the ASAT values in 7 pigs in the 1st litter, Group 1, exceeded the upper limit, 2 in the 9-week sample, 6 in the 15-week sample (only 1 repetition), and 2 in the 21-week sample (1 repeat and 1 new). The corresponding ALAT values exceeded the upper normal limit in plasma samples from 5 animals. In 3 of these the ASAT values also exceeded

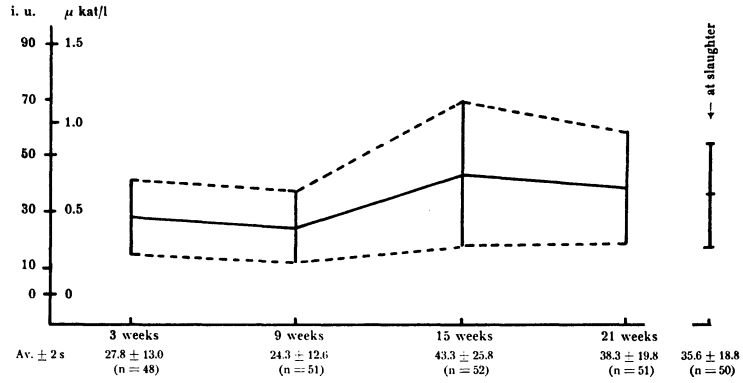

Figure 5. Normal plasma ALAT values in growing pigs. Mean ± 2 s, Groups 3 and 4.

the upper normal limit. Out of the 20 animals in Group 1 only 2 showed normal Se-values in all 4 plasma samples from the same animal. All the remaining had 1 or more subnormal plasma Se values.

In the 2nd litter, Group 1 (4 animals) only 1 pig had an ASAT value exceeding the upper limit (9-week sample). This pig at the same time had an elevated ALAT value. In 2 samples from this pig low plasma Se values were found. Another pig showed an elevated ALAT value twice (9 and 15 weeks), and at the same time the plasma Se values were below the lower normal limit. All 4 pigs in this group had, however, 1 or more plasma Se values below the lower normal limit.

Four pigs died in Group 1, 2 from the 1st litter and 1 from each of the 3rd and 4th litters. One 4 months old pig died from terminal ileitis. This pig had elevated ASAT, normal ALAT and normal blood Se values. Another pig died 4½ months old after a week with inappetance and elevated temperature. This pig had an increase in ASAT as well as ALAT values. Blood and tissue Se values were low. The 2 pigs from the 3rd and 4th litters died before any blood samplings were made. Both had low tissue Se values.

In Group 2 all average ASAT-ALAT values were found within normal range whereas the average blood Se values in several samplings were below the lowest normal range. Three pigs died from Group 2, 2 from the 1st and 1 from the 2nd litter. Of the 3 cases 1 showed normal ASAT-ALAT values, low blood and tissue Se values, 1 showed normal ASAT-ALAT values and subnormal blood and tissue Se values, and 1 case, which was a case of dietetic hepatosis, showed normal ASAT-ALAT and blood Se values 3 weeks before death. Tissue Se values were low.

In the 2 high Se groups 5 pigs died, 4 from the 3rd and 1 from 4th litter. The 4 pigs from the 3rd litter died few days old. No blood or tissue analyses were made. The 1 piglet from the 4th litter died 1 week old. Tissue Se values were normal.

In Table 8 a regression analysis relating Se concentration in plasma to the selenium content of different tissues and to plasma ASAT and ALAT values is shown. It is seen that there is a fair correlation between blood and tissue Se values, whereas there is no correlation between the blood Se values and ASAT and ALAT values at all.

Table 8. Regression analysis relating Se concentration in plasma to the selenium content of various tissues and to ASAT and ALAT values.

	b_0	b_1	r	P
Liver	0.044	2.77	0.70	0.001
Heart	0.031	1.47	0.49	0.001
M. iliopsoas	0.023	0.75	0.72	0.001
M. long. dorsi	0.030	0.58	0.57	0.001
ASAT	34	—119	0.19	0.14
ALAT	40	— 60	0.01	0.41

b_0 = intercept.
b_1 = regression coefficient.
r = correlation coefficient.
P = level of significance.

DISCUSSION

As can be seen in Tables 1 and 2 and Fig. 1 (21 weeks versus at slaughter) the plasma Se values showed a sudden, not feed-associated increase in blood samples taken in conjunction with slaughter (electrical stunning). This increase ($P < 0.01$) was higher in pigs with normal plasma Se status as compared with pigs with low Se values. As formerly pointed out by *Simesen & Danielsen* (1975) Se determination on plasma samples taken in conjunction with exsanguination (electrical stunning) is of no diagnostic value.

In this experiment about 10 % of the pigs in the low Se/low Vit. E group died, while only about 4 % in the low Se/high Vit. E group died with Se deficiency symptoms. Thus, the mortality was higher in the combined Se/Vit. E deficiency group than in the group where Se alone was lacking. In the 2 Se supplemented groups no death occurred which could be attributed to Se or Vit. E deficiency.

Young et al. (1977), in a similar experiment with a basal diet containing also 0.03 μg/g Se and supplemented to Se levels of 0.60 and 1.20 μg/g, encountered death within 57 days in 9 out of 10 pigs fed the basal diet. Seven of the 9 pigs were at necropsy found to have MHD and/or HD and/or gastric ulcers. No other pigs died or showed any lesions of Se or Vit. E deficiency.

It is obvious from Tables 1, 2, 4, 5 and 6 that the Se concentrations in blood and tissues reflected the level of dietary Se over a wide range. This is in good agreement with several authors, e. g. *Rasmussen* (1974), *Hitchcock et al.* (1978) and *Mahan & Moxon* (1978). Liver Se determinations have been regarded as the most reliable indicator for Se status of animals (*Van Vleet* 1975). The results in the present study seem to support the contention (*McDowell et al.* 1977) that plasma Se determination is almost as reliable (Table 8). The correlation between Se in plasma and Se in liver and m. iliopsoas is very high (Table 8).

Table 3 shows the regression of Se in sow colostrum and sow milk on content of Se in piglets' plasma at 3 weeks of age. It clearly shows that a high correlation exists between Se in colostrum or milk and level in piglets plasma.

Se values declined after weaning (at 3 weeks) in the 2 Se supplemented groups (Table 2 and Fig. 1). Nadir was reached around the age of 9—15 weeks. After this age, plasma Se values slowly increased. In the 2 groups fed the low dietary Se level, plasma Se values plateaued or decreased further. This result is in accordance with *Mahan et al.* (1977), who found a marked decline in serum and hepatic Se concentrations after weaning at 4 weeks. The lowest Se values in serum and liver were found in the period from 8 to 14 weeks of age.

Glienke & Ewan (1977) suggested that the growth requirement of pigs for Se does not exceed 0.07 mg per kg feed. Using Group 4 (0.06 mg per kg) of the present experiments the normal values were calculated for plasma Se from 3 weeks to slaughter (Fig. 1).

The liver Se values of the present experiments (Table 4) clearly indicated that the 4 litters in Groups 1 and 2 were at risk in regard to develop into clinical cases of hepatosis dietetica or mulberry heart disease (*Simesen & Pedersen* 1975 and *Pedersen & Simesen* 1977).

The increased rate of survival of the pigs on diets with low Se and high Vit. E concentrations compared with the pigs on diets low in both Se and Vit. E demonstrates that Vit. E may to a significant extent prevent development of the Se/Vit. E deficiency syndrome, even when the stores of Se have been almost depleted (*Glienke & Ewan*).

The ASAT values in this investigation were found to exceed

the upper limit of the normal range defined in 8 pigs in the first litter, Group 1, 2 pigs in the 9-week sample, 6 in the 15-week sample (only 1 replicate) and 2 in the 21-week sample (1 replicate and 1 new). The corresponding ALAT values exceeded the upper normal limit in plasma samples from 5 animals (1 double-replicate). In 3 of these, the ASAT values also exceeded the upper normal limit. Of the 20 animals in Group 1, none showed normal Se values in all 4 plasma samples (32 normal out of a total of 79 samples).

In Group 2, 1st litter, the ASAT values exceeded the upper normal range in 5 samples (no replicates). ALAT values exceeded the upper limit in 2 samples (not corresponding to the increased ASAT values). In 53 samples out of 107 in this group the blood Se values were below the lower limit.

In Group 1, 2nd litter, only 1 pig had an ASAT value exceeding the upper normal limit. Three ALAT values (from 2 animals) exceeded the upper limit. The single high ALAT value came from the same animal and sample as the high ASAT value. This animal had at the same time a low blood Se value. In total, however, 9 out of 11 blood samples from this group had low blood Se values.

In Group 2, 2nd litter, neither the ASAT nor the ALAT values exceeded the upper limit at any time. The blood Se value however, were lower than the lower limit in 8 out of 14 samples.

In the 2 high Se groups, 1st and 2nd litters, a total of 6 elevated ASAT and 5 ALAT values appeared. In 1 sample both ASAT and ALAT were elevated, and in 1 animal they were elevated but at different times.

Thus in these experiments it has not been possible to uncover any connection between a low Se status of an animal and increased ASAT-ALAT values. A regression analysis (Table 8) of Se concentration in plasma in relation to ASAT and ALAT values shows a correlation coefficient of 0.19 and 0.01, i. e. almost complete disassociation.

Lannek & Lindberg (1975) emphasize in their survey that determination of the concentration of serum enzymes has proved to be of great value for the diagnosis of liver necrosis and muscular and heart degeneration following the development of Se and Vit. E deficiency. This statement was not confirmed in the present experiments. With the time intervals used here, neither ASAT nor ALAT determinations have been able to reveal the

subclinical deficiency state, as is the case in cattle, sheep and horses. *Bengtsson et al.* (1978 a, b) reported on the basis of experimental deficiency trials with weaned pigs that the ASAT levels (normal/increased) were of an intermittent character with free periods followed by reactions. In our trials the few increases observed have all been more or less casual.

Evidence of the presence of subclinical diseases, including muscular stiffness, with significant increase in ASAT activities was recorded by *Van Vleet et al.* (1975). Such increase was to be expected, because tissue ASAT activities have been reported to be relatively high in a number of porcine organs, including liver, heart and sceletal muscle (*Cornelius et al.* 1959, *Wretlind et al.* 1959, *Tollersrud & Nafstad* 1970 and *Tollersrud* 1973).

In spite of the pronounced low Se plasma values found in Groups 1 and 2, and in spite of the Se/Vit. E deficiency deaths, we have not been able to identify an enzyme picture (ASAT and ALAT) similar to the picture observed for example in Se deficient cattle and sheep herds (*Oksanen* 1965 and several other authors).

The hypothesis of oxidative unstable fat in grain being the cause of the Se/Vit. E deficiency syndrome in swine has been proposed by *Thafvelin* (1960), *Grant* (1961, 1966), *Swahn & Thafvelin* (1962). However, *Carpenter et al.* (1966), *L'Estrange et al.* (1967) and *Conolly et al.* (1970) could not experimentally substantiate this hypothesis. These authors concluded that feeding of rations containing spontaneously oxidizing lipid to pigs had no adverse effects. The trials published in the present paper indicate that the oxidized herring meal, which was fed for a short period to the 2nd litter of pigs, had a much more pronounced influence on Vit. E need than oxidized lard, which was used for the subsequent litters. *Nielsen et al.* (1973) showed that oxidized herring oil was very efficient in enhancing the Vit. E requirement. As shown in Fig. 2, the level of Vit. E in plasma was quite low for the Vit. E supplemented groups (2 and 4). The addition of 2 % artificially oxidized lard was without any aggravating effect.

REFERENCES

Basse, A., H. E. Nielsen, V. Danielsen, G. Gissel-Nielsen, W. Hjarde, T. Leth & M. G. Simesen: To be published.

Bengtsson, G., J. Hakkarainen, L. Jönsson, N. Lannek & P. Lindberg: Requirement for selenium (as selenite) and vitamin E (as α-tocopherol) in weaned pigs. 1. The effect of varying α-tocopherol levels in a selenium deficient diet on the development of the VESD syndrome. J. Anim. Sci. 1978 a, *46*, 143—152.

Bengtsson, G., J. Hakkarainen, L. Jönsson, N. Lannek & P. Lindberg: Requirement for selenium (as selenite) and vitamin E (as α-tocopherol) in weaned pigs. 2. The effect of varying selenium levels in a vitamin E deficient diet on the development of the VESD syndrome. J. Anim. Sci. 1978 b, *46*, 153—160.

Carpenter, K. J., J. L. L'Estrange & C. H. Lea: Effects of moderate levels of oxidised fat (chicken, pig, rat). Proc. Nutr. Soc. 1966, *25*, 25—31.

Conolly, J. F., T. A. Spillane, D. P. R. Poole & D. M. McAleese: Nutritional effects of oxidized lipids in fresh and stored pig diets. Irish J. agric. Res. 1970, *9*, 39—58.

Cornelius, C. E., J. Bishop, J. Switzer & E. A. Rhode: Serum and tissue transaminase activities in domestic animals. Cornell Vet. 1959, *49*, 116—126.

Glienke, L. R. & R. C. Ewan: Selenium deficiency in the young pig. J. Anim. Sci. 1977, *45*, 1334—1340.

Grant, C. A.: Morphological and aetiological studies of dietetic microangiopathy in pigs ("Mulberry Heart"). Acta vet. scand. 1961, *2*, Suppl. 3.

Grant, C. A.: Diseases associated with auto-oxidation of diet fat (pig). Proc. Nutr. Soc. 1966, *25*, 18—24.

Hitchcock, J. P., E. R. Miller, K. K. Keahey & D. E. Ullrey: Effects of arsanilic acid and vitamin E upon utilization of natural or supplemental selenium by swine. J. Anim. Sci. 1978, *46*, 425—435.

Hjarde, W., E. Leerbeck & T. Leth: The chemistry of vitamin E (including its chemical determination). Acta agric. scand. 1973, Suppl. 19, 87—96.

Keiding, R., M. Hørder, W. Gerhardt-Hansen, E. Pitkänen, R. Tenhunen, J. H. Strømme, L. Theodorsen, J. Waldenström, N. Tryding & L. Westlund: Recommended methods for the determination of five enzymes in blood. The committee on enzymes of Scand. Soc. clin. Chem. and clin. Physiol., 2nd rep., March 1973, 24 pp.

Lannek, N. & P. Lindberg: Vitamin E and selenium deficiencies (VESD) of domestic animals. Adv. vet. Sci. comp. Med. 1975, *19*, 127—164.

L'Estrange, J. K., K. J. Carpenter, C. H. Lea & L. J. Parr: Nutritional effects of autoxidized fats in animal diets. 4. Performance of young pig on diets containing meat meals of high peroxide value. Brit. J. Nutr. 1967, *21*, 377—390.

Mahan, D. C. & A. L. Moxon: Effect of increasing the level of inorganic selenium supplementation in the post-weaning diets of swine. J. Anim. Sci. 1978, *46*, 384—390.

Mahan, D. C., A. L. Moxon & M. Hubbard: Efficacy of inorganic selenium supplementation to sow diets on resulting carry-over to their progeny. J. Anim. Sci. 1977, *45*, 738—746.

McDowell, L. R., J. A. Froseth, R. C. Piper, I. A. Dyer & G. H. Kroening: Tissue selenium and serum tocopherol concentrations in selenium-vitamin E deficient pigs fed peas (Pisum Sativum). J. Anim. Sci. 1977, *45*, 1326—1333.

Morgenstern, S., M. Oklander, J. Auerbach, J. Kaufman & B. Klein: Automated determination of serum glutamic oxaloacetic transaminase. Clin. Chem. 1966, *12*, 95—111.

Nielsen, H. E., N. J. Højgaard-Olsen, W. Hjarde & E. Leerbech: Vitamin E content in colostrum and sows milk and sow milk yield at two levels of dietary fats. Acta agric. scand. 1973, Suppl. 19, 35—39.

Nielsen, H. E., V. Danielsen, M. G. Simesen, G. Gissel-Nielsen, W. Hjarde, T. Leth & A. Basse: Selenium and vitamin E deficiency in pigs. I. Influence on growth and reproduction. Acta vet. scand. 1979, *20*, 276—288.

Oksanen, H. E.: Studies on nutritional muscular degeneration (NMD) in ruminants. Acta vet. scand. 1965, *6*, Suppl. 2, 110 pp.

Olson, O. E.: Fluorometric analysis of selenium in plants. J. Ass. off. anal. Chem. 1969, *52*, 627—634.

Pedersen, K. B. & M. G. Simesen: Feed supplementation with selenium in relation to the vitamin E-selenium deficiency syndrome in pigs. Nord. Vet.-Med. 1977, *29*, 161—165.

Rasmussen, O. K.: Selenium concentration and deposition. Performance and carcass levels of sodium selinate. Acta agric. scand. 1974, *24*, 115—125.

Reitman, S. & S. Frankel: A colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases. Amer. J. clin. Path. 1957, *28*, 56—63.

Simesen, M. G. & V. Danielsen: The diagnostic value of plasma selenium estimations on blood samples collected in conjunction with exsanguination of swine. Acta vet. scand. 1975, *16*, 551—553.

Simesen, M. G. & K. B. Pedersen: Selenium determinations in Danish swine affected with hepatosis dietetica. Acta vet. scand. 1975, *16*, 137—139.

Swahn, O. & B. Thafvelin: Vitamin E and some metabolic diseases of pigs. Vitam. and Horm. 1962, *20*, 645—657.

Thafvelin, B.: Role of cereal fat in the production of nutritional disease in pigs. Nature (Lond.) 1960, *188*, 1169—1172.

Thompson, J. N. & M. L. Scott: Selenium deficiency in chicks and quail. Proc. Cornell Nutr. Conf. for Feed Manufacturers, Ithaca, New York 1967, 130—136. (Role of selenium in the nutrition of the chick. J. Nutr. 1969, *97*, 335—342).

Tollersrud, S.: Changes in the enzymatic profile in blood and tissue in pre-clinical and clinical vitamin E-deficiency in pigs. Acta agric. scand. 1973, Suppl. 19, 124—127.

Tollersrud, S. & I. Nafstad: The vitamin E-deficiency syndrome in pigs. II. Investigations on serum and tissue enzyme activity. Acta vet. scand. 1970, *11*, 495—509.

Van Vleet, J. F.: Retention of selenium in tissues of calves, lambs and pigs after parenteral injection of a selenium-vitamin E preparation. Amer. J. vet. Res. 1975, *36*, 1335—1340.

Van Vleet, J. F., K. B. Meyer, H. J. Olander & G. R. Ruth: Efficacy and safety of selenium-vitamin E injections in newborn pigs to prevent subclinical deficiency in growing swine. Amer. J. vet. Res. 1975, *36*, 387—393.

Wretlind, B., K. Orstadius & P. Lindberg: Transaminase and transferase activities in blood plasma in tissues of normal pigs. Zbl. Vet.-Med. 1959, *6*, 963—970.

Young, L. G., R. B. Miller, E. D. Edmeades, A. Lun, G. C. Smith & G. J. King: Selenium and vitamin E supplementation of high moisture corn diets for swine reproduction. J. Anim. Sci. 1977, *45*, 1051—1060.

SAMMENDRAG

Selen- og vitamin E mangel hos svin. II.
Indflydelse på Se og Vit. E plasma- og Se vævsværdier samt plasma ASAT og ALAT.

Virkningen af peroralt tilført selen (Se) og vitamin E (Vit. E) på plasmaværdierne for Se, Vit. E og transaminaser (ASAT og ALAT) samt på vævsværdier for Se er blevet undersøgt.

To niveauer af Se blev tilført, nemlig 0,03 og 0,06 mg pr. kg foder. Hvert af holdene på de 2 Se niveauer blev igen opdelt i to, nemlig et hold med foder indeholdende 15 I. E. Vit. E og et med 45 I. E. pr. kg foder. Herved fremkom 4 grupper: 1. Lav Se, lav Vit. E; 2. Lav Se, høj Vit. E; 3. Høj Se, lav Vit. E og 4. Høj Se, høj Vit. E.

Ti % af grisene på lav Se, lav Vit. E, og 4 % af grisene på lav Se, høj Vit. E døde med udtalte symptomer på Se mangel. Ingen af de grise, som fik foder med højt Se indhold, viste sådanne symptomer. Plasma Se bestemmelserne viste sig at reflektere grisenes Se status næsten ligeså akkurat som Se bestemmelser på levervæv. ASAT og ALAT plasma enzymbestemmelser viste sig med de benyttede blodprøveudtagelsestidspunkter at være uden diagnostisk værdi. Plasma Se bestemmelser udført på blodprøver udtaget i tilslutning til aflivning med elektrisk bedøvning viste betydelig højere værdier end prøver udtaget umiddelbart før slagtningen.

Forsøgene viste, at der er god overensstemmelse imellem foderets Vit. E indhold og de tilsvarende niveauer i plasma. Oksyderet sildeolie synes at forøge grisenes Vit. E behov.

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Reprints may be requested from: M. G. Simesen, the Institute of Internal Medicine, Royal Veterinary and Agricultural University, Bülowsvej 13, DK-1870 Copenhagen V, Denmark.