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EFFECT OF DIETARY FAT QUALITY AND VITAMIN E ON THE ANTIOXIDANT POTENTIAL OF PIGS*

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THODE JENSEN, P., H. E. NIELSEN, V. DANIELSEN and T. LETH: *Effect of dietary fat quality and vitamin E on the antioxidant potential of pigs.* Acta vet. scand. 1983, *24*, 135—147. — A randomized blocked factorial experiment was conducted with 90 young pigs. From 4 to 11 weeks of age the pigs were kept in individual pens and fed a selenium supplemented basal diet consisting mostly of propionic acid treated barley, soybean meal and dried skim milk, and containing < 0.5 mg vitamin E per kg. The treatment factors during this period were 3 dietary levels of added vitamin E (nil, 10 and 30 mg/kg) and a 6 % supplement of fresh or oxidized fat (⅔ lard and ⅓ herring oil). From 11 weeks of age until slaughter at 90 kg the pigs received the vitamin E supplements but no fat or dried skim milk. The basal diet for this later experimental period was based on untreated dry barley. Blood samples collected during the period of investigation were examined for vitamin E and for resistance against erythrocyte lipid peroxidation (ELP) in order to evaluate the antioxidant status.

Analysis of variance and Student's t-test on least squares means showed the ELP to be influenced independently by the vitamin E supplement and, during the fat feeding period, by the quality of the fat supplement, with the highest peroxidation resistance (low ELP) in the groups fed fresh fat and a high level of vitamin E. Blood vitamin E level was only influenced — positively — by the vitamin E supplement although variations in the feed vitamin E level below 10—15 mg vitamin E per kg did not result in corresponding variations in measurable blood vitamin E concentrations. In the same low range of vitamin E in the feed there was a statistically significant difference in ELP values between the different vitamin E treatment groups. No clinical manifestations of selenium-vitamin E deficiency were observed in the pigs.

The ELP and the plasma vitamin E levels observed would seem to suggest that a total of 15 mg vitamin E per kg barley-based feed will not always be sufficient for growing pigs.

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vitamin E; antioxidant status; fat quality; erythrocyte lipid peroxidation; pigs.

Dietary selenium, vitamin E, and fatty acids are known as the main determinants in the selenium-vitamin E deficiency syndrome. In pigs, the most prominent manifestations of this syndrome, which often leads to sudden deaths among young animals, are dietetic hepatosis (HD) and mulberry heart disease (MHD).

Supplementation of pig feed with selenium seems to prevent deaths from HD, while deaths from MHD may still occur (*Pedersen & Simesen* 1977, *Moir & Master* 1979). The field investigations conducted by these authors together with experimental studies, have provided evidence to suggest that the lesions of the heart are chiefly associated with vitamin E deficiency (*Van Vleet et al.* 1975, *Hakkarainen et al.* 1978, *Simesen et al.* 1982).

In spite of the fact that selenium supplemented pig feed has been used in almost all herds in Denmark since it was legalized in 1976, MHD is frequently diagnosed on routine post mortem examination of material submitted to the State Veterinary Serum Laboratory. During the same period very few cases of HD have been diagnosed. This is surprising, because, in addition to the Se supplementation, high levels (20—40 mg/kg) of vitamin E in the form of gelatin-coated dl-α-tocopheryl acetate, have been added to the feed in most of the herds concerned. In different other investigations the fat content of the feed, and especially a high level of polyunsaturated fatty acids and oxidized fat, have been found to influence the demand for vitamin E and to precipitate selenium-vitamin E deficiency diseases in pigs (*Swahn & Thafvelin* 1962, *Malm et al.* 1976, *Simesen et al.* 1979).

The biochemical reactions involved in selenium-vitamin E deficiency conditions, include oxidation of polyunsaturated fatty acids in biological membranes, with subsequent production of lipidperoxides (*Hoekstra* 1975). Factors which will reduce susceptibility to autooxidation are essential to prevent disease. The present experiment was designed to examine the effect of different dietary levels of vitamin E and of different fat qualities on the antioxidant status of young, growing pigs. Besides vitamin E in plasma, the resistance of erythrocyte lipids against peroxidation (ELP) was used as a measure of the antioxidant status of the pigs.

MATERIALS AND METHODS

The experimental animals were piglets born to sows fed a barley and soybean meal based diet with vitamins and minerals, and containing 0.1 mg supplemental Se per kg (as sodium selenite) and a supplement of 20 mg dl-α-tocopheryl acetate per kg. The piglets were weaned at 4 weeks of age 3 days after the males had been castrated. At weaning, 6 equally sized piglets from each of 15 litters were randomly assigned to 6 different diets in a factorial experiment with 3 levels of vitamin E and 2 qualities of fat (Table 1). The pigs were kept in single pens and fed ad libitum during the first experimental period of 7 weeks. From the end of that period until slaughter at 90 kg, the pigs still received the vitamin E supplement, but were fed no fat and no dried skim milk (Table 1). During this period the pigs were fed restrictively with rations ranging from approx. 1.4 kg per day at 11 weeks to approx. 3.0 kg per day at 90 kg. The barley for use during the first experimental period had a moisture content of approx. 20 %. After treatment with 1 % propionic acid it was stored for at least 8 weeks before it was used. The purpose of this treatment was to reduce the content of vitamin E.

Table 1. Experimental design. Basal diet and supplements for 6 treatment groups.

	Group					
	1	2	3	4	5	6
No. of pigs	15	15	15	15	15	15
4—11 weeks of age[1]						
Vit. E suppl., mg/kg	0	0	10	10	30	30
Fat quality	fresh	oxidized	fresh	oxidized	fresh	oxidized
Vit. E in whole ration, mg/kg	4.2	2.0	15.5	13.5	37.6	37.0
11 weeks — 90 kg live weight[2]						
Vit. E suppl., mg/kg	0	0	10	10	30	30
Vit. E in whole ration, mg/kg	6.5	6.5	14.6	14.6	35.2	35.2

[1] Diet: 49.2 % propionic acid treated barley, 35.0 % soybean meal, 6.0 % dried skim milk, 4.0 % animal fat, 2.0 % herring oil, 3.2 % mineral-tracemineral mixture, 0.6 % vitamin mixture without vitamin E.

[2] Diet: 74.9 % untreated barley, 22.0 % soybean meal, 2.6 % mineral-tracemineral mixture, 0.5 % vitamin mixture without vitamin E.

During the later experimental period, and for the sows, dry, untreated barley was used. The fat used consisted of lard (⅔) and herring oil (⅓). The fat for the low quality diets was oxidized by streaming hot air. No antioxidants were used. Pig mortality was recorded troughout the experiment, and dead animals were necropsied.

Heparinized blood samples were collected from the anterior vena cava when the pigs were 4, 7, 11, 12, 16 and 20 weeks old. The blood cells were tested for resistance against lipid peroxidation (ELP) by the method described by *Fontaine & Valli* (1977) and modified by *Jensen et al.* (1979). The method is based on the reaction of 2-thiobarbituric acid (TBA) with malonylaldehyde produced by in vitro peroxidation with hydrogen peroxide. Blood vitamin E was determined at 4, 11 and 20 weeks of age on 5 pools of plasma, each representing 3 pigs. Vitamin E determination was made by high pressure liquid chromatography of the unsaponifiable matter on a column of kieselgel, and measuring of the fluorescense (327 nm) of the eluate upon excitation at 290 nm. Plasma for vitamin E determination was kept at —20°C for a few weeks until analysed. Statistical analyses were made by the Statistical Analysis System described by *Helwig & Council* (1979). In addition to simple correlations the analyses included t-test on least squares means and analysis of variance, both performed by the general linear model (GLM-) procedure with block (litter), quality of fat, and levels of added vitamin E as independent variables.

RESULTS

The response at different ages to the main treatments is presented in Table 2 as mean values and standard errors of blood vitamin E, ELP, average daily gain, and feed to gain ratio. Fig. 1 presents, for each treatment group, the mean ELP values during the period of investigation. The results of the analysis of variance are shown in Table 3. The F-values are based on partial mean squares, i.e., mean squares corrected for influence of other factors, including litter effect or, with respect to vitamin E in blood, "pool" effect. The F-values provide a measure of the influence of the different feed supplements (treatments) on the dependent variables measured. One pig from treatment No. 1 and one from treatment No. 2 died 6—7 weeks old without conclusive lesions of Se-vitamin E deficiency and were excluded

Table 2. Mean values of measured variables (s.e.m. in brackets) for each treatment (feed supplement). Fat I = good quality fat. Fat II = oxidized fat. B = Basal diet with < 0.5 mg vit. E per kg (4—11 weeks) or 10 mg vit E. per kg (11 weeks to slaughter). Fat was included in the diet only during the age period 4—11 weeks.

	Treatments					
	B + 0 mg vit E		B + 10 mg vit E		B + 30 mg vit E	
Measured variables	Fat I	Fat II	Fat I	Fat II	Fat I	Fat II
Blood vit. E, μg/ml, 4 weeks	2.82 (0.34)	1.92 (0.33)	1.91 (0.32)	2.17 (0.28)	2.22 (0.41)	2.21 (0.55)
Blood vit. E, μg/ml, 11 weeks	0.05 (0.02)	0.02 (0.01)	0.24 (0.10)	0.04 (0.02)	0.50 (0.21)	0.45 (0.19)
Blood vit. E, μg/ml, 20 weeks	0.03 (0.02)	0.07 (0.05)	0.19 (0.08)	0.32 (0.14)	1.13 (0.26)	1.20 (0.23)
ELP, O.D. 535 nm, 4 weeks	.029 (.005)	.028 (.002)	.024 (.002)	.028 (.003)	.026 (.004)	.029 (.004)
ELP, O.D. 535 nm, 7 weeks	.201 (.026)	.210 (.022)	.141 (.029)	.168 (.024)	.093 (.021)	.132 (.028)
ELP, O.D. 535 nm, 11 weeks	.220 (.028)	.247 (.023)	.146 (.031)	.142 (.025)	.037 (.007)	.090 (.019)
ELP, O.D. 535 nm, 12 weeks	.166 (.021)	.175 (.023)	.081 (.016)	.052 (.012)	.028 (.007)	.039 (.008)
ELP, O.D. 535 nm, 16 weeks	.208 (.021)	.194 (.020)	.082 (.017)	.083 (.018)	.030 (.004)	.036 (.066)
ELP, O.D. 535 nm, 20 weeks	.165 (.019)	.151 (.012)	.047 (.008)	.042 (.008)	.018 (.001)	.026 (.004)
Av. daily gain, g, 4—11 weeks	470 (19)	449 (17)	440 (18)	436 (15)	446 (12)	431 (19)
Av. daily gain, g, 11 weeks to slaughter	726 (17)	718 (18)	715 (12)	696 (18)	741 (13)	733 (20)
Feed to gain ratio, 4—11 weeks	1.72 (0.04)	1.76 (0.03)	1.75 (0.03)	1.70 (0.04)	1.70 (0.03)	1.72 (0.03)
Feed to gain ratio, 11 weeks to slaughter	3.06 (0.07)	3.08 (0.07)	3.04 (0.04)	3.14 (0.08)	2.94 (0.06)	2.96 (0.06)

from the analysis; besides, a few blood samples coagulated and were therefore unfit for examination. Consequently, some of the calculations are based on a reduced number of observations.

From Table 3 it is seen that, plasma vitamin E levels were influenced by the levels of vitamin E supplement both at 11 and

Table 3. Analysis of variance for blood vitamin E, ELP, average daily gain, and feed to gain ratio.

Measured variable	F-values for treatments, d.f. in brackets				Residual variance	R^2 [b]
	Block 'litter'[a]	Vit. E	Fat quality	Vit. E × Fat		
Blood vit. E 4 weeks	5.18** (4)	0.64 (2)	0.79 (1)	2.14 (2)	431×10^{-3}	0.58
Blood vit. E 11 weeks	2.54° (4)	8.80** (2)	1.13 (1)	0.41 (2)	595×10^{-4}	0.60
Blood vit. E 20 weeks	4.07* (4)	42.2*** (2)	0.44 (1)	0.01 (2)	822×10^{-4}	0.85
ELP 4 weeks	5.62*** (12)	0.48 (2)	0.49 (1)	0.43 (2)	828×10^{-5}	0.54
ELP 7 weeks	10.2*** (13)	17.8*** (2)	4.15* (1)	0.39 (2)	334×10^{-3}	0.73
ELP 11 weeks	3.23*** (14)	36.1*** (2)	1.84 (1)	1.11 (2)	558×10^{-3}	0.65
ELP 12 weeks	3.12*** (14)	60.3*** (2)	0.04 (1)	1.34 (2)	246×10^{-3}	0.71
ELP 16 weeks	2.32* (14)	74.9*** (2)	0.04 (1)	0.32 (2)	284×10^{-3}	0.73
ELP 20 weeks	1.77° (14)	104.9*** (2)	0.16 (1)	0.51 (2)	133×10^{-3}	0.79
Av. daily gain, 4—11 weeks	5.72*** (14)	2.24 (2)	2.23 (1)	0.37 (2)	230×10^{1}	0.56
Av. daily gain, 11 weeks to slaughter	1.96* (14)	2.17 (2)	0.86 (1)	0.09 (2)	346×10^{1}	0.33
Feed to gain ratio, 4—11 weeks	4.75*** (14)	0.57 (2)	0.14 (1)	1.55 (2)	105×10^{-4}	0.51
Feed to gain ratio, 11 weeks to slaughter	1.38 (14)	2.61° (2)	0.46 (1)	0.33 (2)	595×10^{-4}	0.28

*** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$, ° $P < 0.10$.

[a] For blood vit E it is not a litter effect but a 'pool' effect, cf. 'Materials and Methods'.

[b] R^2 indicates the proportion of variation explained by the analytical model.

20 weeks of age ($P < 0.01$ and $P < 0.001$). Also ELP levels were influenced by the vitamin E supplement at all examinations from 7 to 20 weeks of age ($P < 0.001$). Mean ELP values for the blood samples pooled for vitamin E determination were positively cor-

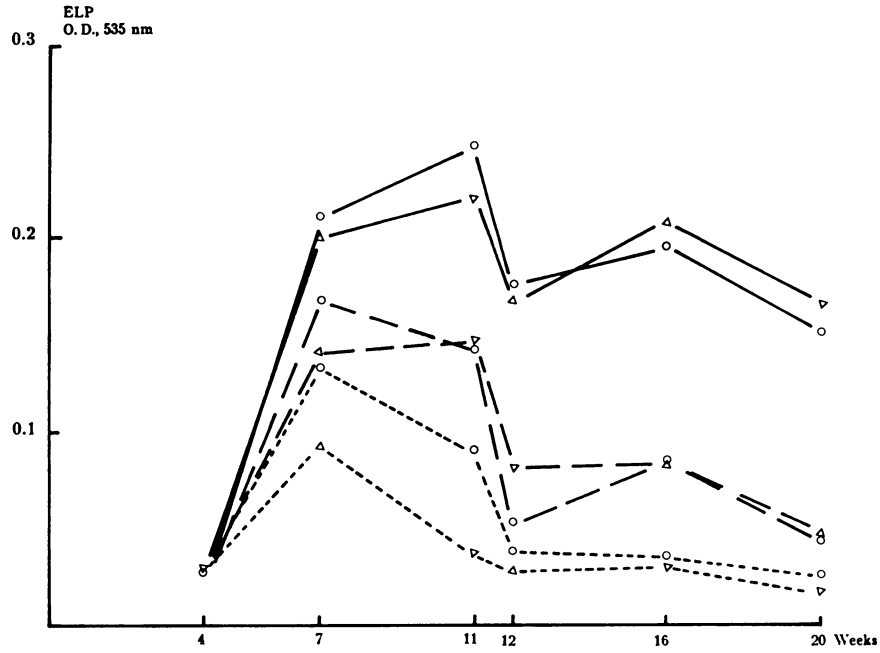

Figure 1. Mean ELP values for different treatments during the investigation periods, cf. 'Materials and Methods'. ▽ = fresh fat, ○ = oxidized fat (4 to 11 weeks). ——— 0 mg vit. E, — — — 10 mg vit. E, ---- 30 mg vit. E (per kg feed).

related to the reciprocal values of vitamin E in the pools at both 11 and 20 weeks of age ($r = 0.59$, $P < 0.001$ and $r = 0.73$, $P < 0.001$) but uncorrelated at 4 weeks ($r = 0.06$, $P = 0.74$). Only at 7 weeks did the fat quality influence the ELP values significantly ($P < 0.05$) with oxidized fat giving the highest ELP values. On the other hand, this investigation showed no significant influence of fat quality on blood vitamin E levels. The significant litter effect on ELP values decreased with age (Table 3).

As seen from Fig. 1 there was a notable fall in mean ELP values for all treatment groups from 11 to 12 weeks, which is the period immediately after the shift to a diet without fat and more rich in vitamin E.

For the different treatment groups at specified age, both the blood vitamin E and the ELP values were compared by Student's t-test on least squares means. At 11 weeks, after the period with

fat supplemented diet, the vitamin E supplement of 10 mg per kg feed to groups 3 and 4 did not result in significantly higher blood vitamin E mean levels in these groups than in the vitamin E unsupplemented groups, whereas the mean ELP values differed significantly (Table 4). At the same age a supplement of 30 mg vitamin E per kg feed resulted in both a significantly higher blood vitamin E mean level and a lower ELP value. At 20 weeks, after 9 weeks without fat supplemented diet, the blood vitamin E mean levels and mean ELP of the various groups were proportional to the vitamin E supplement.

Table 4. The influence of added vitamin E (mg per kg feed) on the blood vitamin E (μg/ml) and ELP (O.D., 535 nm) in pigs 11 and 20 weeks old. The treatments were compared by Student's t-test on least squares means (LSM).

Measured variable	Treatment		
	0 mg	10 mg	30 mg
Blood vit. E, 11 weeks	0.04[a]	0.14[a]	0.47[b]
Blood vit. E, 20 weeks	0.03[a]	0.23[a]	1.16[b]
ELP, 11 weeks	2.32[a]	1.43[b]	0.63[c]
ELP, 20 weeks	1.57[a]	0.45[b]	0.25[c]

[a,b,c] Means with different superscript within row differ significantly ($P < 0.05$)

The variations in fat quality and vitamin E levels in the feed did not influence the production results significantly at either 11 or 20 weeks of age. No significant interactions between supplements were seen.

There seems to be a difference in vitamin E content between the 2 fat qualities, cf. Table 1. Therefore, the effect of fat quality on ELP at 7 and 11 weeks was investigated separately by comparing least squares means (t-test) for the groups of pigs which were fed high vitamin E levels and for which there would therefore be little, and presumably negligible, difference in feed vitamin E between groups getting different fat qualities. By this investigation the influence of fat quality on the ELP was found significant at 11 weeks ($P = 0.01$) and almost significant at 7 weeks ($P = 0.11$), cf. Fig. 1. At the same time (11 weeks) no difference in blood vitamin E level was found between the

groups of animals fed good quality fat and those fed oxidized fat (Table 2).

DISCUSSION

The results of this study confirm the close relationship between dietary vitamin E on the one hand and blood vitamin E and the peroxidation resistance of the erythrocytes (ELP) on the other hand, as previously demonstrated in pigs by *Fontaine & Valli* (1977), *Jensen et al.* (1979) and *Simesen et al.* (1982). This relationship seems to be relatively independent of the quality of the dietary fat, in that the variables mentioned were found not to be significantly influenced by interactions between fat quality and vitamin E in the feed. However, besides vitamin E the fat quality itself seems to influence the ELP, even though a difference in vitamin E content between the 2 fat qualities may tend to muddle the results of the analysis of variance. Unfortunately, the oxidized fat supplied the feed with 2.2 mg vitamin E less per kg than the good quality fat. Accordingly, the diet of the 2 vitamin E unsupplemented groups will have varied in vitamin E content from 4.2 mg per kg to 2.0 mg per kg (cf. Table 1). For the vitamin E supplemented groups this difference was relatively less, and for the two groups which received 30 mg vitamin E per kg feed it was considered to be negligible, in that these two groups differed only from 37.6 to 37.0 mg per kg. By comparing these two groups of pigs separately, a possible confounding effect is probably excluded. As the mean ELP values, both at 7 and 11 weeks of age, seem to be highest for the group of pigs fed the poor quality fat, it is concluded that fat of poor quality will increase the susceptibility of the red cells to peroxidation in vitro.

The apparently higher vitamin E supply needed by pigs fed oxidized fat (*Swahn & Thafvelin* 1962) may to a great extent be due to the low vitamin E content in such fat (*Bauernfeind* 1980). The results of the ELP determinations in the present investigation indicate that oxidized fat in the feed may by itself increase the susceptibility of tissues to oxidation, irrespective of the level of vitamin E. An influence of fat could be expected, since the polyunsaturated fatty acids in the biological membranes, which are a substrate for peroxidation, will to some extent reflect the fatty acid composition of the diet.

Stocks & Dormandy (1971) found the resistance of red cells against peroxidation to be influenced by haemoglobin. In the present investigation, preliminary calculations showed, as expected, haemoglobin concentration and haematocrit value to be highly correlated to litter effect. Furthermore, using the type I sum of squares in the SAS-GLM-procedure ELP was not invariably found to be influenced by haemoglobin concentration, whereas it was always influenced by block (litter) even after correction for haemoglobin influence. The haematological parameters when tested as dependent variables were found to be independent of the treatments. Since, in the light of these observations, it is doubtful whether haemoglobin has a real influence on ELP, haematological parameters were not included on their own account in the final calculations. It is true that, in man, biochemical abnormalities of the erythrocytes will influence their peroxidation susceptibility (*Stocks & Dormandy* 1971, *Stocks et al.* 1972), but in pigs, in which such abnormalities are extremely rare, ELP is assumed to be influenced mostly by physiological and nutritional factors, especially vitamin E.

Since the quality of the dietary fat had no significant influence on the blood vitamin E level, absorption of vitamin E is assumed not to have been influenced by the fat quality under the conditions of this experiment.

The blood vitamin E level of the 11 weeks old pigs supplied with 30 mg vitamin E per kg fat supplemented diet is the same as found in an experiment with a vitamin E supply of 50 mg per kg (*Simesen et al.* 1982). The changes in blood vitamin E level seen in groups 5 and 6 can not be explained directly. Whether the low level at 11 weeks is caused by an age effect or by the dietary fat supplement is an open question, but the negative influence of oxidized lard on blood vitamin E found by *Simesen et al.* (1979) gives reason to look upon the fat supplement as important.

It is remarkable that in the groups of pigs fed no supplementary vitamin E (1 and 2) the mean blood vitamin E levels were the same at 20 weeks as at 11 weeks although during the intervening period the pigs had received 6.5 mg vitamin E per kg basal diet. Since at 11 weeks the blood vitamin E level was almost the same in groups 3 and 4 as in groups 1 and 2, it looks as if, until a certain level, the concentration of vitamin E in plasma does not reflect the vitamin E concentration in the feed. On the

other hand, although at the age of 11 weeks there was no clear difference in blood vitamin E mean levels between the pigs given a supplement of 10 mg vitamin E per kg feed and the unsupplemented pigs, there was a highly significant difference in mean ELP values between these groups, Table 4. So, at low dietary vitamin E levels, ELP seems to be a more sensitive index of the vitamin E status of pigs than the blood vitamin E level.

At 11 and 20 weeks, especially in groups 1 and 2, but also in groups 3 and 4, the low blood vitamin E levels seem to indicate a state of vitamin E deficiency. At the same time also the susceptibility of the blood cells to peroxidation (high ELP) indicates vitamin E deficiency in these groups.

As in a previous investigation (*Simesen et al.* 1982) addition of vitamin E to the diet had no appreciable influence on the daily gain. The vitamin E supplemented pigs had a little lower feed to gain ratio in the period from 11 weeks until slaughter. This result is not significant ($P = 0.08$) but it is in agreement with other reports (*Piper et al.* 1975, *Simesen et al.* 1982). The tendency toward a litter effect on the ELP values found in a previous study (*Jensen et al.* 1979) was found statistically significant in the present investigation, especially at the younger ages.

In conclusion, fat quality of the dietary fat apparently did not influence blood vitamin E levels, but oxidized fat was found to reduce the resistance of the red blood cells against peroxidation, and maybe also the resistance of other cells and tissues. At low levels of vitamin E in the feed, blood vitamin E was not as sensitive an index of the vitamin E status of the pigs as was the resistance of the blood cells against peroxidation. The results of the present investigation suggest that a total of 15 mg vitamin E per kg barley-based feed will not be quite sufficient for growing pigs.

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SAMMENDRAG

Betydningen af foderets fedtkvalitet og vitamin E indhold for den biologiske antioxydantstatus hos svin.

Et faktorielt forsøg blev gennemført med ialt 90 grise. Fra grisene var 4 til de var 11 uger gamle blev de holdt i enkeltstier og fodret med et selenberiget grundfoder bestående af propionsyrebehandlet byg, soyaskrå og skummetmælkspulver og indeholdende < 0,5 mg vitamin E per kg. Forsøgsbehandlingerne var 3 niveauer af vitamin E (0, 10 og 30 mg/kg) og et fedttilskud på 6 % bestående af frisk eller oxyderet fedt (⅔ svinefedt og ⅓ sildeolie). Fra grisene var 11 uger og indtil de blev slagtet ved 90 kg fik de fortsat vitamin E tilskudene, medens de ikke fik fedt eller skummetmælkspulver i foderet. Grundfoderet i denne forsøgsperiode var fremstillet af veltørret byg. Blodprøver, der var udtaget under forsøget, blev undersøgt for vitamin E indhold i plasma, ligesom blodlegemernes resistens mod peroksydering (ELP) blev bestemt for herved at vurdere grisenes antioxydantstatus.

Ved variansanalyse samt t-test på "least squares means" fandtes ELP værdierne at være afhængige af såvel foderets vitamin E indhold som af kvaliteten af det tilsatte fedt, således at et højt vitamin E indhold og god fedtkvalitet gav den største resistens mod peroksydering (lave ELP værdier). Der fandtes en positiv sammenhæng mellem blodets vitamin E indhold og vitamin E-indholdet i foderet, dog således at der i grupperne, der fik mindre end 10—15 mg vitamin E pr. kg foder, ikke fandtes tilsvarende variationer i det målelige vitamin E indhold i blodet. I modsætning hertil fandtes statistisk sikre forskelle mellem ELP værdierne for de grupper af grise der havde fået de to lave niveauer af vitamin E. Foderets fedtkvalitet havde tilsyneladende ingen indflydelse på blodets vitamin E indhold. Der blev ikke observeret kliniske tilfælde af selen-vitamin E mangel hos forsøgsgrisene. De fundne ELP værdier og plasma vitamin E koncentrationer gør det sandsynligt at 15 mg vitamin E pr. kg foder ikke altid er nok til at sikre en optimal antioxydantstatus hos grisene.

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