Vitamin E, Selenium and Methionine Supplementation of Dystrophogenic Diets for Pigs

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ABSTRACT

Forty-eight weanling S.P.F. Yorshire pigs were used to study the influence of supplemental vitamin E (25 IU per kg of diet) selenium (0.5 ppm in diet) and methionine (0.1% in diet) on the incidence of hepatosis dietetica and mulberry heart disease when fed a torula yeast-corn diet. Vitamin E and/or selenium increased pig survival. Supplemental selenium resulted in increased liver selenium concentrations. No hepatosis dietetica was observed in any of the pigs. The addition of vitamin E and/or selenium at the levels used did not reduce the frequency of myocardial lesions; however, they prevented skeletal muscular dystrophy and exudative diathesis. The myocardial lesions were less severe in supplemented pigs compared with unsupplemented controls.

RÉSUMÉ

Les auteurs ont utilisé 48 porcelets guotobiotes Yorkshire, pour étudier l'influence de l'enrichissement d'une diète à base de levure torula et de maïs avec de la vitamine E (25 UI/kg), du sélénium (0.5 ppm) et de la méthionine (0.1%), sur la fréquence de l'hépatose diététique et de la cardiopathie mûriforme. La vitamine E et/ou le sélénium prolongèrent la survie des porcelets. L'addition de sélénium en augmenta la concentration dans le foie. L'hépatose diététique n'affecta aucun des porcelets. L'addition de vitamine E et/ou de sélénium, dans les proportions mentionnées plus haut, ne réduisit pas la fréquence des lésions du myocarde; elle réussit cependant à prévenir la dystrophie des muscles squelettiques et la diathèse exsudative. Les lésions du myocarde s'avérèrent moins prononcées chez les porcelets supplémentés que chez les témoins.

INTRODUCTION

A nutritional disease of swine, hepatosis dietetica (HD), which was responsive to vitamin E and the sulphur containing amino acids methionine and cystine was reported by Obel (10). Other lesions frequently observed along with the liver necrosis were myocardial and skeletal muscular degeneration and gastric ulcers. Later Eggert *et al* (4) observed that both vitamin E and selenium prevented liver necrosis in pigs. Grant *et al* (5) described a condition in swine called microangiopathy which was interpreted to be identical to mulberry heart disease. This condition could be prevented with the addition of tocopherol and sodium selenite to the diet. Reid *et al* (12) reported that supplementation of a basal diet, low in protein, with methionine protected pigs against liver necrosis. More recently, Michel *et al* (8) prevented dietary hepatic necrosis in swine fed a six percent protein diet containing torula yeast with the addition of selenium, high levels of vitamin E and more protein.

In another recent paper, Nafstad *et al* (9) were able to prevent mulberry heart disease, hepatosis dietetica, skeletal muscular dystrophy by the supplementation with DL-α-tocopherol acetate.

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Due to the apparent increase in the incidence of hepatic necrosis, mulberry heart disease and associated lesions in pigs in Otario during recent years, it seemed advisable to further investigate the effect of vitamin E, selenium and methionine on the incidence of these diseases when added to dystrophogenic diets (13).

A wide range of lesions has been associated with vitamin E and/or selenium deficiency in swine. The nomenclature used for various lesions in the literature is rather confusing. The following terms will be used in this paper. Mulberry heart disease (MHD) refers to myocardial degeneration, hepatosis dietetica (HD) is used for massive liver necrosis, skeletal muscle degeneration (SMD) for degenerative changes in skeletal muscles, exudative diathesis (ED) refers to subcutaneous accumulation of straw colored fluid, microangiopathy (MAP) means fibrinoid necrosis of capillarial and arterial walls, gastric ulcer means ulceration of the squamous epithelium in the pars esophagea of the stomach.

MATERIALS AND METHODS

Forty-eight Yorkshire S.P.F. pigs averaging 11.1 kg from six litters were distributed across eight treatments. The pigs were weaned at approximately four weeks of age and were maintained in individual pens for nine days prior to the initiation of the trial. During this period the pigs were fed a diet composed of ground corn, soybean meal, minerals and vitamins excluding supplemental vitamin E or selenium. The pigs were assigned such that the male pigs were fed diets 1214, 1224, 1225 and 1226, and the female pigs fed diets 1227, 1228, 1229 and 1230 while the female pigs from the last three litters were fed the first four diets and the male pigs the last four diets. The eight diets were formulated by supplementing a corn-torula yeast basal diet with vitamin E (E), selenium (Se), DL-methionine (Me) and the various combinations (Table I).

The pigs were maintained in individual pens with a raised wire floor and had access to individual self-feeders and water nipples. The pigs remained on trial until death or for 85 days at which time the survivors were electrocuted.

Samples of hair and liver were obtained for chemical analyses. The hair samples, obtained from surviving pigs one week before the trial terminated, were washed 5X with 100 ml volumes of distilled water and dried in a dessicator before Se analysis. Assays for Se and α-tocopherol were according to procedures outlined previously (13).

All pigs were necropsied within 12 hours after death. Sections of liver, myocardium, skeletal muscle (semitendinosus), skin, femoral marrow, spleen, kidney and stomach were fixed in 10% formalin, embedded in paraffin, cut at 6u and stained with hematoxylin and eosin.

The performance data were evaluated using analysis of variance and Duncan's New Multiple Range test (3). The levels of liver and hair Se were evaluated by

TABLE I. Composition of Experimental Diets

Diet Number Supplement	1214 Basal	1224 E	1225 Se	1226 Me	1227 E Se	1228 E Me	1229 SeMe	1230 E SeMe
Ingredients								
Corn	74.5	73.5	73.5	73.5	72.5	72.5	72.5	71.5
Torula yeast	22.4	22.4	22.4	22.4	22.4	22.4	22.4	22.4
Vitamin-mineral premix[a]	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
Vitamin E premix[b]		1.0			1.0	1.0		1.0
Selenium premix[c]			1.0		1.0		1.0	1.0
Methionine premix[d]				1.0		1.0	1.0	1.0

[a]Supplied 0.5% cobalt iodized salt, 0.5% calcium phosphate, 1.5% limestone.
Trace minerals added in ppm of complete diet manganese 60, iron 70, copper 10, zinc 100.
Vitamins added per kg of diet; riboflavin 4.4 mg, pantothenic acid 8.8 mg, choline chloride 110.3 mg, B_{12} 19.9 µg, A 3, 309 IU, D_2 330.9 IU
[b]Supplied 25 IU of vitamin E per kg of diet
[c]Supplied 0.5 ppm Se in the diet
[d]Supplied 0.1% DL — methionine in the diet

analysis of variance with unequal observations per treatment (14).

RESULTS

Survival

Five of the six pigs fed the diet supplemented with E, Se and Me survived (Table II) and only one pig of six fed either the basal or Me supplemented diets survived the 85 day trial. Methionine supplementation did not appear to exert a protective effect when added to dystrophogenic diet. Survival of the pigs fed the other diets was intermediate between the two extremes. A significant difference ($P = 0.05$) in survival time among the replications was observed.

Pathology

No evidence of HD was observed in any pigs in this trial (Table II). Supplementation of the basal diet with E and/or Se and combinations of E Me, Se Me and E Me Se prevented SMD. The addition of Me alone had little effect on the frequency of SMD.

The addition of E and/or Se did not reduce the frequency of MHD at the levels administered. (Table II).

Exudative diathesis was not observed in pigs treated with E and/or Se or combinations of E Me and Se Me. Two pigs out of six treated with Me alone had exudative diathesis. Vitamin E and/or Se supplementation had no effect on the incidence of gastric erosions and/or ulcers. The group supplemented with Me alone had the lowest incidence of gastric lesions (one of six) when Me was given in combination with E and Se no effect was observed.

TABLE II. Frequency of Death and Pathological Lesions Observed in Pigs Fed a Torula Yeast-Corn Diet Supplemented with Vitamin E, Selenium and Methionine

Diet Number Supplement	1214 Basal	1224 E	1225 Se	1226 Me	1227 E Se	1228 E Me	1229 MeSe	1230 E SeMe
Died/Total	5/6	2/6	2/6	5/6	3/6	2/6	2/6	1/6
Myocardial degeneration	6/6[a]	4/6	6/6	6/6	5/6	6/6	6/6	6/6
Microangiopathy	6/6	4/6	6/6	6/6	5/6	6/6	6/6	6/6
Hepatosis dietetica	0/6	0/6	0/6	0/6	0/6	0/6	0/6	0/6
Skeletal muscle dystrophy	5/6	0/6	0/6	4/6	0/6	0/6	0/6	0/6
Exudative diathesis	4/6	0/6	0/6	2/6	0/6	0/6	0/6	0/6
Gastric erosions and/or ulcers	4/6	6/6	6/6	1/6	5/6	6/6	4/6	6/6

[a]Number of pigs with lesions of total receiving the treatment

TABLE III. Performance of Pigs Fed a Torula Yeast-Corn Diet Supplemented with Vitamin E, Selenium and Methionine

Diet Number Supplement	1214 O	1224 E	1225 Se	1226 Me	1227 E Se	1228 E Me	1228 MeSe	1230 E SeMe
Average daily gain kg[a]	0.41	0.51	0.47	0.26	0.43	0.44	0.49	0.52
Average daily feed kg dry matter[a]	1.02	1.14	1.12	0.80	1.01	1.09	1.13	1.22
Gain/feed[a]	0.39	0.42	0.41	0.29	0.41	0.40	0.42	0.42
Liver selenium[b]	0.18^{1}	0.21^{3}	2.17^{3}	0.28^{1}	$1.90^{2,3}$	0.35^{1}	$1.80^{2,3}$	1.41^{2}
Number of samples	5	4	4	5	4	4	3	3
Hair selenium[b]	0.10^{1}	0.19^{1}	$0.41^{1,2,3}$	$0.29^{1,2,3}$	$0.46^{1,2,3}$	$0.26^{1,3}$	$0.51^{1,2}$	$0.63^{2,3}$
Number of samples	1	4	4	1	2	3	3	5
Feed selenium[b]	0.03	0.03	0.49	0.03	0.48	0.03	0.50	0.50
Feed tocopherol[c]	10.77	36.70	9.10	11.80	36.09	36.26	11.13	28.06

[a]For first 49 days of an 85 day trial
[b]Selenium μg/g dry basis except hair reported on as is basis
[c]α-tocopherol μg/g dry basis
^{1,2,3}-means bearing different superscripts differ significantly ($P = 0.05$)

Tissue Selenium

The addition of 0.5 ppm Se as sodium selenite to the torula yeast-corn basal diet increased the liver Se concentration over the non-supplemented pigs (Table III). No significant differences existed in the liver Se content of pigs supplemented with E and/or Me without Se. The pigs supplemented with Se alone had the highest liver Se content while those supplemented with a combination of E, Se and Me were significantly lower. The number of samples analyzed per treatment is indicated (Table III).

The Se content of hair tended to increase when pigs were supplemented with Se (Table III). The addition of either or both E and Me to the diet tended to result in a further increase in the hair Se content. These trends did not attain statistical significance and were based on only one observation in some cases.

The hair Se levels tended to parallel the liver Se levels; however, supplementation of the diet with E or Me tended to result in decreased liver Se levels and increased hair Se levels. These trends did not attain statistical significance and were based on only one observation in some cases.

Growth and Feed Utilization

Supplementation of the basal diet with E, Se or Me did not result in any significant differences in average daily gain, average daily dry matter consumption or feed efficiency for the first seven weeks of the trial (Table III).

DISCUSSION

In contrast to earlier reports (10, 12), Me supplementation did not appear to exert a protective effect when added to the dystrophogenic diets.

The basal diet used in these experiments was identical to that used in previous trials (13) in which a high frequency of HD was observed. The absence of HD lesions in the present trial may be due to the faster growing pigs developing myocardial lesions and dying before HD developed. Spontaneous cases of MHD generally occur in fast growing pigs. Supplemetation of the dystrophogenic diets with E and Se only reduced the severity but not the frequency of myocardial lesions in this trial. The lesions in supplemented pigs consisted primarily of microscopic areas of post-necrotic scarring. In contrast the myocardial lesions in the unsupplemented groups consisted of large areas of degeneration and hemorrhage which were evident grossly. Our results are only in partial agreement with those of other workers who prevented MHD with the addition of E and/or Se (5, 9).

Other workers (5, 9) did not prevent SMD with the addition of sodium selenite to experimental diets containing high levels of unsaturated fatty acids. Possibly the discrepancies between their results and ours may be explained on the basis of level of unsaturated fatty acids in the diet. Orstadius *et al* (11) protected pigs against SMD with intramuscular administration of sodium selenite.

Hidiroglou *et al* (7) observed a direct relationship between the Se content of forage, dam's hair and the incidence of white muscle disease in calves. A similar observation was made with respect to dietary selenium and hair selenium in the present trial.

Nafstad *et al* (9) obtained only a partial protective effect when E was added to known ulcerogenic diets. The addition of amino acids, Se and an increase in dietary protein did not have an effect on the incidence of gastric lesions in their work. The results reported here are in agreement with those obtained by Hannan *et al* (6) and Curtin (2) who were unable to prevent gastric ulcers with vitamin E. More recently, gastric ulcers could be prevented when ground barley was added to the diet at a level of 5-10 percent. (1)

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