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CLINICO-PATHOLOGIC FINDINGS IN YOUNG PIGS FED DIFFERENT LEVELS OF SELENIUM, VITAMIN E AND ANTIOXIDANT*

By

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SIMESEN, M. G., P. THODE JENSEN, A. BASSE, G. GISSEL-NIELSEN, T. LETH, V. DANIELSEN and H. E. NIELSEN: *Clinico-pathologic findings in young pigs fed different levels of selenium, vitamin E and antioxidant.* Acta vet. scand. 1982, 23, 295—308. — A randomized, blocked 2^3 factorial experiment was conducted with 48 young pigs. The treatment factors were: 2 levels of selenium (55 and 115 µg/kg), 2 levels of vitamin E (3 and 53 mg/kg) and 2 levels of the antioxidant feed additive Ethoxyquin (0 and 150 mg/kg). All pigs were kept in single pens and fed ad libitum throughout the experimental period of 9 weeks, i.e. from 3 to 12 weeks of age.
Plasma, heart, liver and muscle Se levels as well as whole blood glutathione peroxidase activity (EC 1.11.1.9 GSH-Px) were significantly higher in pigs given a dietary supplement of Se than in pigs given no supplement of Se (P ≤ 0.001). The Se-supplemented pigs showed a tendency to lower mean serum transaminase activity (ASAT and ALAT) than unsupplemented pigs, but the influence was significant (P ≤ 0.05) only for the ALAT activity.
Blood vit. E levels were higher for pigs receiving a supplement of vit. E than for unsupplemented pigs (P ≤ 0.001), and so was the resistance of red blood cells against lipid peroxidation (ELP), as expressed by lower ELP values.
There were no effects of Ethoxyquin supplementation on the biochemical variables included in the study.
The histological examination of heart muscle showed that the score for changes was negatively influenced by both Se and vit. E supplement (P ≤ 0.001) and to some extent also by Ethoxyquin supplement (P ≤ 0.05). The histological picture of m. long dorsi was influenced only by the vit. E supplement (P ≤ 0.01). No histological changes were found in the liver in this study. There were inverse relationships between whole blood GSH-Px defluorescence time and blood Se, and between ELP and whole blood vit. E (P ≤ 0.001).

selenium; vitamin E; antioxidant; glutathione
peroxidase; erythrocyte lipid peroxidation;
ASAT; ALAT; mulberry heart disease; dietetic
hepatosis; nutritional muscular dystrophy;
pigs.

Extensive studies have been carried out to clarify the etiology
of the 3 main clinical manifestations of selenium-vitamin E
deficiency in swine: Hepatosis Dietetica (HD), Mulberry Heart
Disease (MHD) and Nutritional Muscular Dystrophy (NMD) (cf.
e.g. *Lannek & Lindberg* 1975, *Underwood* 1977). Blood Se and vit.
E estimations have been shown to be reliable indicators of, re-
spectively the Se and the vit. E status in livestock (*Van Vleet*
1980). The Se contents in liver and heart have been found signi-
ficantly lower in pigs dying of MHD, than in normal pigs, but
higher than in pigs dying of HD (*Pedersen & Simesen* 1977).

Determination of glutathione peroxidase activity (GSH-Px)
and measurement of the resistance of erythrocytes against lipid
peroxidation in vitro (ELP) seem to be valuable and simple
methods for evaluating Se and vit. E status, respectively, in
young pigs, as described by *Fontaine & Valli* (1975) and, partly
based on the same data as used in the present study, by *Jensen
et al.* (1979). In another study, ASAT and ALAT estimations
were found to be without diagnostic value in this respect (*Sime-
sen et al.* 1979).

The present experiment was designed to examine the effect
of different dietary levels of Se, vit. E and antioxidant on the
clinico-pathologic and post-mortem findings in young pigs.

MATERIAL AND METHODS

The experimental animals were piglets born to sows fed a diet
low in Se and vit. E. The piglets were weaned at 3 weeks of age.
Male pigs were castrated 3 days before they were assigned to
the experiment. At weaning, 8 piglets from each of 6 litters were
randomly allocated to 8 different treatments, in a blocked 2^3
factorial experiment (*Snedecor & Cochran* 1967). The treatment
factors were: 2 levels of Se, 2 levels of vit. E and 2 levels of the
feed additive antioxidant Ethoxyquin (Table 1). All pigs were
kept in single pens and fed ad libitum throughout the experi-
mental period of 9 weeks. The barley included in the diet had a
moisture content of approx. 20 % and was treated with 1 %

T a b l e 1. Experimental design. Basic diet and supplements for the 8 treatment groups.

Se-suppl. µg/kg (as sodium selenite)	0				60			
Vit. E suppl. mg/kg (dl-tocopheryl acetate, gelatin-coated)	0		50		0		50	
Ethoxyquin suppl. mg/kg	0	150	0	150	0	150	0	150
Number of pigs	6	6	6	6	6	6	6	6

Basic diet: 60.3 % barley; 5.0 % soybean meal; 12.0 % dried skim milk; 10.0 % dried yeast; 3.0 % sugar; 4.0 % lard; 3.3 % mineral-tracemineral mixture; 0.6 % vitamin mixture; 1.8 % lysine-methionine mixture. Native contents: 55 µg Se/kg, 3 mg vitamin E/kg.

propionic acid and then stored for at least 8 weeks before it was used. The purpose of this treatment was to reduce the content of vit. E (*Madsen et al.* 1973).

At the end of the experiment the pigs were sacrificed for post-mortem examination.

Blood samples were collected from the anterior vena cava immediately before the pigs were killed. In continuation of the blood sampling the pigs were anaesthetized with mebumal i.v. and exsanguinated. Se-analyses were carried out by the fluorimetric procedure described by *Olson* (1969). Vit. E determination was made by high pressure liquid chromatography of the unsaponifiable matter on a column of kieselgel and measuring the fluorescence of the eluate with exitation at 290 nm and emission at 327 nm (*Leth* 1978). GSH-Px activity was determined according to *Board & Peter* (1976) though with minor modifications, and resistance against erythrocyte lipid peroxydation by the method of *Fontaine & Valli* (1977) and modified as decribed by *Jensen et al.* (1979). With these methods a low numerical value indicates high activity or high resistance, respectively.

Transaminase determinations (ALAT and ASAT) were made according to *Keiding et al.* (1973).

Tissue samples for histological examination were fixed in formalin and embedded in paraffin. Slides were prepared and stained with hematoxylin-eosin according to conventional methods. Slides of m. longissimus dorsi were used for classification of muscle lesions. Slides without conclusive lesions suggestive of

Se-, vit. E deficiency were given score 0; Slides with conclusive lesions were given score 1 or 2, indicating respectively a low and a high degree of severity.

Arithmetic means, standard deviations, coefficients of correlation and simple linear regression equations were calculated by standard procedures. Analysis of variance was performed by the general linear model procedure of *Helwig & Council* (1979). The effect of each treatment as well as interactions between treatments was analyzed within blocks, i.e. litters.

RESULTS

Table 2 shows for each treatment group the mean value and the standard deviation (s) for the clinico-chemical variables, i.e. Se and vit. E in plasma, ASAT and ALAT activity in serum and GSH-Px and ELP in whole blood. In addition, average daily gain, feed conversion rate, body weight, weight of heart and liver, and content of Se in liver, heart and m. longissimus dorsi, are included in the table, as is also the degree of histological changes in heart and skeletal muscle. All livers were without changes suggestive of HD. The results of the analysis of variance are shown in Table 3. The F values are based on partial mean squares, i.e. they are calculated after correction for influence of the other factors, including litter effect. The F values provide a measure of the influence of the different feed supplements (treatments) on the dependent clinico-pathological variables measured. One pig fed basic diet, died the day before the experiment was terminated. For some of the variables the total number of observations is therefore limited to 47.

The Se-levels in plasma, heart muscle, liver and skeletal muscle as well as the GSH-Px activity in whole blood, were significantly higher in pigs receiving a dietary Se-supplement than in pigs given no such supplement ($P \leq 0.001$). In contrast, Se-supplemented pigs had lower mean serum ALAT activity and also lower liver weight at slaughter, than unsupplemented pigs ($P \leq 0.05$).

The blood vit. E levels were higher for pigs given supplementary vit. E than for pigs not given vit. E and so was the resistance of red blood cells against lipid peroxidation as expressed by lower ELP-values ($P \leq 0.001$). Vit. E supplementation had apparently a positive influence upon feed utilization, since pigs receiving vit. E had lower feed-to-gain ratio than pigs given no dietary vit. E supplement ($P \leq 0.05$).

T a b l e 2. Mean values and s (in brackets) for each treatment with respect to measured variables (B = basic diet).

Measured variable	B	B + Se	B + Vit. E	B + Ethox.	B + Se + Vit. E	B + Se + Ethox.	B + Vit. E + Ethox.	B + SE + Vit. E + Ethox.
Blood-Se, μg/kg	.024 (.008)	.079 (.029)	.025 (.006)	.028 (.011)	.068 (.017)	.088 (.015)	.025 (.006)	.079 (.022)
Blood-vit. E, μg/kg	.03 (.04)	.07 (.12)	.32 (.31)	.03 (.08)	.40 (.28)	.05 (.08)	.47 (.34)	.35 (.19)
ASAT, μkat/ml	5.2 (6.1)	1.1 (.7)	1.9 (2.3)	2.4 (2.7)	3.0 (3.6)	1.5 (.8)	2.8 (2.4)	1.2 (.5)
ALAT, μkat/ml	1.6 (1.3)	.7 (.1)	.8 (.2)	1.2 (.5)	.9 (.4)	1.0 (.7)	1.3 (.8)	.8 (.2)
GSH-Px, deflourescense, min.	55.0 (15.4)	21.3 (5.5)	65.5 (17.1)	60.8 (19.9)	19.8 (5.2)	18.8 (5.8)	64.2 (18.6)	17.8 (5.0)
ELP, O.D. 535 nm	0.155 (0.047)	0.126 (0.031)	0.018 (0.001)	0.121 (0.049)	0.018 (0.002)	0.112 (0.056)	0.018 (0.003)	0.019 (0.002)
Avg. daily gain, g	396 (97)	373 (127)	386 (67)	410 (73)	421 (56)	435 (81)	424 (55)	433 (47)
Feed-to-gain ratio	2.03 (.20)	2.13 (.23)	1.97 (.16)	2.02 (.13)	1.94 (.05)	1.97 (.15)	1.95 (.15)	1.90 (.10)
Body weight, kg	30.9 (6.2)	29.3 (8.6)	29.7 (5.0)	31.1 (4.5)	32.2 (4.9)	32.4 (5.6)	31.5 (4.1)	32.1 (3.6)
Liver weight, g	928 (138)	800 (186)	853 (112)	843 (74)	858 (96)	804 (87)	856 (168)	837 (110)
Heart weight, g	149 (16)	126 (35)	127 (14)	134 (21)	135 (20)	135 (19)	131 (7)	135 (13)
Liver-Se, mg/kg	.086 (.021)	.253 (.057)	.081 (.015)	.076 (.009)	.261 (.078)	.248 (.050)	.093 (.012)	.272 (.063)
Heart muscle-Se, mg/kg	.053 (.006)	.116 (.015)	.056 (.006)	.051 (.004)	.119 (.011)	.119 (.014)	.062 (.005)	.125 (.028)
M. long. dorsi, Se mg/kg	.031 (.006)	.051 (.006)	.029 (.006)	.029 (.006)	.048 (.005)	.051 (.006)	.029 (.005)	.050 (.006)
Heart muscle-histol. changes (score 0—2)	1.3 (.5)	0 0	.2 (.4)	.3 (.5)	0 0	.2 (.4)	0 0	0 0
M. long. dorsi histol. changes (score 0—2)	1.3 (.5)	1.3 (1.0)	.7 (.5)	1.2 (1.0)	.2 (.4)	.7 (.8)	.5 (.8)	.3 (.5)

There were no effects of Ethoxyquin supplementation on the biochemical or production variables included in the study. With respect to the haematological variables i.e. haemoglobin and haematocrit estimations there were no differences between the 8 groups of pigs.

T a b l e   3.   Analysis of variance for the clinico-pathological variables measured.

Measured variable	Block (litter) (5)	F-values for treatments; d.f. in brackets							Residual variance	R² (coefficient of determination)[a]
		Se (1)	Vit.E (1)	Ethox. (1)	Se × Vit.E (1)	Se × Ethox. (1)	Vit.E × Ethox. (1)	Se × Vit.E × Ethox. (1)		
Blood Se	2.6*	152.3***	1.8	1.9	1.2	1.0	.01	.1	218×10^{-6} (35)	.83
Blood Vit. E	5.0**	0	43.2***	.2	.2	1.3	.3	.7	301×10^{-4} (34)	.68
ASAT	2.7°	3.1°	.2	1.2	2.1	0	.2	3.6°	717×10^{-2} (35)	.40
ALAT	3.5*	6.3*	1.1	.1	1.6	.1	.9	5.8*	295×10^{-3} (35)	.47
GSH-Px	5.6***	195.8***	.6	0	1.4	.3	.1	.2	107 (34)	.87
ELP	2.0	1.1	149.4***	1.8	1.2	.4	1.8	.3	961 (34)	.83
Avg. daily gain	6.5***	.5	.5	3.1°	.4	.1	.1	1.1	372×10^{1} (35)	.52
Feed-to-gain ratio	4.3**	0	7.1*	2.7	.7	1.5	.6	.7	169×10^{-4} (35)	.50
Body weight	5.1**	.4	.5	2.7	.3	.1	.1	1.0	168×10^{-1} (35)	.47
Liver weight[b]	2.5°	4.2*	0	2.6	.7	.2	.6	.1	927×10^{1} (34)	.55
Heart weight[b]	3.9**	4.1°	5.4*	4.0	6.5*	3.4	3.1	1.7	653×10^{-1} (34)	.87
Liver Se	2.7*	211.8***	.9	.1	.2	.01	.7	.01	172×10^{-5} (35)	.87
Heart muscle Se	2.0	317.1***	2.6	.8	.1	.1	.5	.1	157×10^{-6} (35)	.90
M. long. dorsi Se	9.3***	309.7***	1.4	0	.3	.9	.7	.1	165×10^{-7} (35)	.91
Heart-histol. changes (0—2)	0	16.8***	16.8***	6.1*	10.8**	10.8**	2.7	6.1*	123×10^{-3} (35)	.67
M. long. dorsi-hist. changes (0—2)	.3	1.7	10.0**	0.9	0	0	.9	.9	599×10^{-3} (35)	.31

*** $P < .001$; ** $P < .01$; * $P < .05$; ° $P < .10$

a R^2, i.e. the proportion of variation explained by the analytical model.

b Total body weight is here included in the analytical model.

The score for histological changes in the heart muscle was negatively influenced by Se as well as by vit. E supplement (P $\leq$ 0.001) and to some extend also by Ethoxyquin supplement (P $\leq$ 0.05). With regard to the m. long. dorsi, only vit. E supplement showed influence (negative) on the score for changes (P $\leq$ 0.01).

Whole blood GSH-Px defluorescence time and blood Se were inversely related, as shown in Fig. 1 and by the regression equation: GSH-Px = 12.29 + 1.01 $\times$ Se^{-1} (r = —.86; P $\leq$ 0.001).
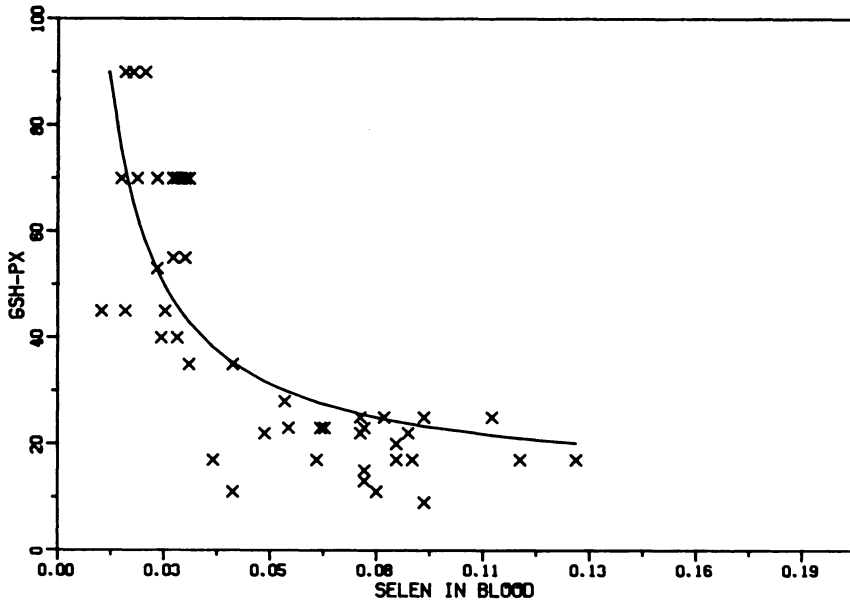
F i g u r e 1. The inversely related GSH-Px defluorescence time and blood Se. (GSH-Px = 12.29 + 1.01 $\times$ Se^{-1})

In many blood samples the vit. E content was below measurable level. In calculating the inverse relationship between ELP and blood vit. E such non-measureable levels were entered in the regression equation with a value of zero:

ELP = 27.51 + 1.12 $\times$ vit. E^{-1} (r = —.58, P $\leq$ 0.001).

The relationship is illustrated in Fig. 2.
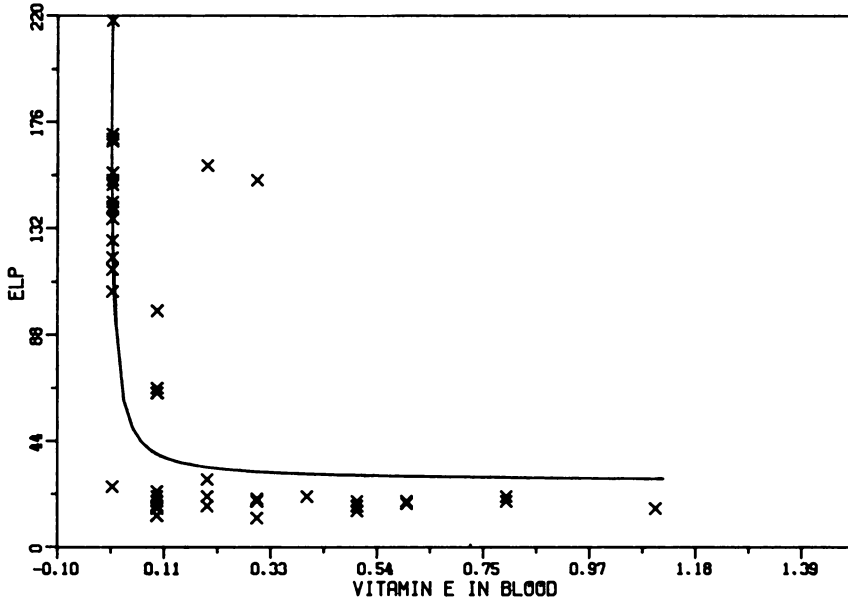
F i g u r e 2.   The inverse relationship between ELP and blood vit. E.
(ELP = 27.51 + 1.12 × vit. E^{-1})

DISCUSSION

The results of this study confirm the close relationship between dietary and plasma and liver Se previously demonstrated by *Hakkarainen et al.* (1978b), *Chavez* (1979a), *Simesen et al.* (1979), *Wegger et al.* (1980) and *Meyer et al.* (1981).

There was a high correlation between GSH-Px and blood Se (cf. Fig. 1 and Table 3) and GSH-Px was closely related to dietary Se. This is in agreement with results obtained by *Jensen* (1977), *Sivertsen et al.* (1977), *Hakkarainen et al., Chavez* 1979b and *Meyer et al.*

With increasing dietary Se the curve for whole blood GSH-Px will rise to a certain level corresponding to enzyme saturation, and thereafter form a plateau. Plasma Se, on the other hand, will continue to increase curvilinearly with increasing dietary Se levels (*Nielsen & Rasmussen* 1979, *Meyer et al.*).

Se is incorporated into GSH-Px during erythrocyte maturation. Therefore the GSH-Px activity of the erythrocytes will reflect the mean Se supply during the last few months, whereas plasma GSH-Px seems merely to reflect feed Se changes of short duration and GSH-Px release from damaged and effeted cells

(*Thompson et al.* 1981). Therefore, while plasma Se and whole blood GSH-Px give a good picture of the total Se status of the pig (*Meyer et al.*), plasma GSH-Px appears to be a fast, simple and reliable method to evaluate the short-term intake of dietary Se (*Chavez* 1979 a). The high correlation found in this study between Se and GSH-Px activity in blood was to be expected since the pigs were fed only moderate Se levels. Furthermore almost all GSH-Px avtivity in pig erythrocytes is known to be Se-dependent (*Lawrence & Burk* 1978).

Likewise a close relationship was found between blood vit. E and ELP (Fig. 2). This confirms that as suggested by *Fontaine & Valli* (1977) red cell resistance against peroxidation is a reliable indicator of the vit. E status of pigs. The F values shown in Table 3, clearly demonstrate that the ELP values were influenced only by the vit. E supplements, and that vit. E had no influence on GSH-Px activity and Se values.

In man, red cell resistance against peroxidation is influenced not only by vit. E and lipids in the diet but also by biochemical abnormalities of the erythrocytes (*Stocks et al.* 1972). In pigs, biochemical abnormalities seem to be extremely rare. Therefore, on a herd basis ELP is assumed to reflect influence solely by physiological and nutritional factors, especially vit. E.

As previously described the whole blood GSH-Px and ELP were in this study found to be good indices of the Se and the vit. E status of the pigs, respectively (*Jensen et al.* 1979). From the analysis of variance (Table 3) it can be seen that the increased Se level in the feed gave a higher F value for GSH-Px than for blood Se concentration, and that the increased vit. E. feeding gave a higher F value for ELP than for the blood vit. E level. The difference in F value was especially great in the case of ELP and blood vit. E corresponding to the relatively low correlation between these two variables. As GSH-Px and ELP have biological rather than simple chemical implications, these results may be taken to suggest that GSH-Px and ELP are possibly more valuable indices of the Se and vit. E status of pigs than the respective blood concentrations.

As can be seen from Table 3 the histological features of the heart muscle were significantly influenced by dietary Se, vit. E and Ethoxyquin and even by interactions between these supplements. The histological picture of m. long dorsi was significantly influenced only by vit. E and the liver was not influenced by the

55 µg Se and the 3 mg vit. E per kg dry basic diet. The results are consistent with the results of experiments by *Van Vleet et al.* (1975) and *Hakkarainen et al.* (1978 a). *Van Vleet et al.* used a basic diet with only 30 µg Se/kg and the experiments by *Hakkarainen et al.* included groups with extremely low dietary Se and vit. E levels attainable only by using a semisynthetic diet. Like the present authors *Van Vleet et al.* and *Hakkarainen et al.* found the heart to be the main target organ for lower degrees of vit. E and Se deficiencies.

Small amounts of vit. E and Se in the feed have been found to be able to prevent/reduce the occurrence of HD. Thus in field investigations *Pedersen & Simesen* (1977) found that a mean supplement of 0.1 mg Se/kg dry feed was able to prevent HD in young Danish pigs. However, with this diet MHD continued to occur, in spite of the fact that most herds must have used vit. E supplemented feed. Also Australian investigations (*Moir & Master* 1979) have provided evidence to suggest that HD is chiefly associated with Se deficiency, whereas MHD is more associated with vit. E deficiency.

Addition of Se to the basic diet did not influence the daily gain nor the feed-to-gain ratio. This is in good agreement with findings by *Ewan et al.* (1969) and *Groce et al.* (1971). Nor did vit. E supplementation have any appreciable influence on the average daily gain. This agrees with results which appeared in a very comprehensive report from *NRC* (1974). Although differences between groups due to vit. E were rather small, they were statistically significant. As shown in Table 3 there were great variations in feed-to-gain ratio between blocks due to vit. E. This explains why the group differences were significant in spite of their apparent negligibility. Only few reports have shown significant differences in performance due to vit. E (*Piper et al.* 1975).

As appears from Table 3, a pronounced influence due to litter (block effect) was observed for most variables. It is especially interesting to note that Se in blood, liver and m. long dorsi, and GSH-Px and vit. E in blood, are closely related to litter, as were also average daily gain and feed-to-gain ratio.

It is well established that both average daily gain and feed-to-gain ratio are genetically influenced to a high degree, and the data from this study would indicate that this is also the case for Se in the body as well as for blood vit. E and GSH-Px activity. A genetic influence on blood GSH-Px activity has previously been

assumed by *Jørgensen et al.* (1977). ELP seems not to be genetically influenced.

Although blood and liver Se and blood vit. E have been regarded as the best indicators for Se and vit. E deficiencies, this experiment, among others, shows that determination of GSH-Px and ELP may be suitable as alternative in vivo tests for Se and vit. E deficiencies.

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SAMMENDRAG

Klinisk-patologiske fund hos unge svin, fodret med forskellige niveauer af selen, vitamin E og antioxidant.

Et 2^3 faktorielt forsøg blev gennemført med ialt 48 grise. Dyrene indgik i undersøgelsen ved en alder af 3 uger, og de blev aflivet ved 12 ugers alderen. Forsøgsbehandlingen var to niveauer af selen (55- og 115 µg/kg), to niveauer af vitamin E (3- og 53 mg/kg) og to niveauer af foder-antioxidanten Ethoxyquin (0- og 150 mg/kg). Grisene var under hele forsøget placeret i enkeltstier, og de havde adgang til foderet ad libitum. Ved forsøgets afslutning blev grisene obduceret med henblik på såvel patologisk-anatomiske som biokemiske undersøgelser.

Effekten af hver behandling samt interaktionerne mellem behandlinger blev beregnet ved hjælp af en variansanalyse.

Se-indholdet i plasma, hjerte, lever og m. longissimus dorsi samt

glutation peroxidase (GSH-Px) aktiviteten i fuldblod var signifikant højere for grise, der fik tilskud af Se i foderet end hos grise uden Se-tilskud (P ≤ 0.001). Grise, der fik Se-tilskud, havde gennemgående lavere ASAT og ALAT aktivitet i serum end grise, fodret uden tilskud af Se; kun for ALAT's vedkommende var indflydelsen af Se-tilskuddet statistisk signifikant (P ≤ 0.005).

Blodets indhold af vit. E var højere og blodlegemernes resistens mod peroxydering (ELP) var lavere for grise, der fik vit. E end for grise uden tilskud af vit. E (P ≤ 0.001). Der var ingen effekt af Ethoxyquin-tilskud på de biokemiske variable, der blev målt i forsøget.

De histologiske undersøgelser af hjertemuskulatur viste, at points for forandringer var negativt påvirket af såvel Se som vit. E tilskud (P ≤ 0.001). Der var ligeledes en tendens til negativ indflydelse af Ethoxyquin tilskuddet (P ≤ 0.05). På de histologiske forandringer i m. long. dorsi havde kun vit. E nogen indflydelse (P ≤ 0.01).

Hos ingen af grisene fandtes specifikke histologiske forandringer i leveren.

Der var omvendt proportionalitet mellem GSH-Px defluorescenstid i blod og Se i blod, og mellem ELP og vit. E i fuldblod.

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