Acta vet. scand. 1977, *18*, 140—142.

Brief Communication

THE GENERALIZED SHWARTZMAN REACTION INDUCED BY A SINGLE INJECTION OF ENDOTOXIN IN PIGS FED A VITAMIN E DEFICIENT COMMERCIAL DIET

An experimental generalized Shwartzman reaction (GSR) induced in pigs by endotoxin from Escherichia coli has been reported previously *(Teige et al.* 1973, *Quast* 1973). The diet given to the pigs seemed to influence the outcome of the experiments. It has been found difficult to provoke GSR in pigs fed a commercial diet, even when the E. coli endotoxin is given as a single, relatively large dose or as 2 properly spaced doses (*Teige et al., Nordstoga* 1976). *Quast* induced the GSR by continuous infusion of endotoxin. In connection with 1 experiment in pigs, changes indicative of GSR were observed among a group of animals fed a commercial diet and receiving a small single dose of endotoxin. These observations are presented in this paper.

Materials and methods

The commercial diet* was given to 5 pigs (Nos. 1 to 5) which had an average weight of 17 kg when the feeding period started. A sample of the same feed collected at the end of the feeding period contained 8 mg α-tocopherol per kg**. The diet had been given to pig No. 1 for 10 weeks, the other pigs for 12 weeks, when they were given a single intravenous injection of a partially purified endotoxin***. The examinations of blood samples, the killing of the animals and necropsies were performed in the same way as described in a previous paper (*Teige et al.)*. In addition, sections from kidneys, lungs and skin were stained by the Lendrums acid picro-Mallory method (*Lendrum et al.* 1962). Selenium analyses were performed on liver samples using a fluorometric method (*Ihnat* 1974)****.

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* Svinefór 3, produced by Møllesentralen, Oslo, Norway.

** Test performed by Vitaminlaboratoriet, Bergen, Norway.

*** Lipopoly-saccharide B obtained from E. coli type 026:B6, produced by the Difco laboratories, Detroit, Michigan, USA.

**** Tests performed by The National Veterinary Institute, Oslo, Norway.

Results and comments

The clinical-chemical examinations showed no pathological values. The patho-morphological changes in the kidneys and skin were similar to those described by *Teige et al.* as typical for GSR (Table 1).

Table 1. Weight of carcasses, dose of endotoxin, main necropsy findings and liver selenium values (wet weight).

Pig No.	Weight (kg)	Dose (mg)	Died or killed	Gross lesions* kidney	skin	Pathological* findings	Selenium (μg/g)
1	41	5	Died after 25 hrs.	+++	+	GSR	0.21
2	62	10	Killed after 50 hrs.	—	—	Fibrin thrombi in pulmonary vessels	0.19
3	55	20	„	+++	+++	GSR	0.24
4	53	20	„	+++	+	GSR	0.22
5	61	30	„	+	(+)	GSR	0.28

* The necropsy findings are evaluated according to the description given by *Teige et al.* (1973).

One of the most dominating types of microscopic lesions in the kidneys of the pigs with GSR was mural fibrinoid necrosis in afferent arterioles and interlobular arteries often accompanied by thrombosis. Thrombi were also found in pulmonary vessels of 3 pigs. These changes were especially prominent in pig No. 2. In the myocardium of pigs Nos. 1 and 5, small calcifying degenerative processes appeared. The doses of endotoxin used in the present experiment have not previously provoked the GSR in pigs fed commercial diets (*Teige et al.*). However, these authors have shown that pigs fed an experimental diet producing manifest vitamin E deficiency are predisposed for GSR. The pigs in the present experiment probably received insufficient amount of α-tocopherol as the diet contained only 8 mg per kg. The recommended concentration is between 25 and 30 mg per kg (*Nadai & Brubacher* 1970). The myocardial lesions described may support this assumption as corresponding lesions can develop as a result of vitamin E deficiency (*Nafstad & Tollersrud* 1970). The recorded values of liver selenium were within a range found in normal pigs (*Van Vleet et al.* 1970, *Simesen & Pedersen*

1975). However, the selenium balance of the experimental pigs, indicated by the liver values, seems unable to protect them from developing GSR after an endotoxin injection when the vitamin E supply is suboptimal.

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(Received February 22, 1977).

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