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INFLUENCE OF DIET ON EXPERIMENTAL SWINE DYSENTERY

1. EFFECTS OF A VITAMIN E AND SELENIUM DEFICIENT DIET SUPPLEMENTED WITH 6.8 % COD LIVER OIL

By

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TEIGE, JON jr., KNUT NORDSTOGA & JOHAN AURSJØ: *Influence of diet on experimental swine dysentery. 1. Effects of a vitamin E and selenium deficient diet supplemented with 6.8 % cod liver oil.* Acta vet. scand. 1977, *18*, 384—396. — Sixteen growing pigs were fed a vitamin E and selenium deficient diet; half of the animals (Group 2) were given a daily supply of vitamin E and selenium. After having been fed these diets for 53 days, the pigs were infected orally with minced colonic material from cases with typical swine dysentery. This exposure resulted in outbreaks of swine dysentery in both groups. The incubation times were, however, distinctly shorter and the clinical symptoms much more pronounced in Group 1 than in Group 2. The patho-morphological lesions in the colon also differed between the 2 groups. In the pigs of Group 1 evident pseudomembraneous lesions were observed in the spiral colon. In Group 2, the colonic alterations consisted predominantly of a catarrhal enteritis; pseudomembranes occurred in a minor part of colon in only 4 pigs. Both the clinical and the chemical observations and the pathological findings indicated a much better vitamin E and selenium balance in the pigs of Group 2. It is concluded that the treatment with vitamin E and selenium in Group 2 greatly increased resistance to swine dysentery.

swine dysentery; pseudomembraneous colitis; Treponema hyodysenteriae; vitamin E and selenium deficiency; diet.

Swine dysentery was originally reported by *Whiting et al.* in 1921. The disease was induced after inoculation with Vibrio coli (*Doyle et al.* 1944); however, the disease inductive with this organism has since proved difficult to repeat. Since 1970 a large spirochaete, Treponema hyodysenteriae, has been isolated from cases of swine dysentery (*Harris* 1974). The disease has, in recent years, been induced by inoculation with this microbe, often

administered in colonic material from diseased pigs (*Olson* 1974). Thus T. hyodysenteriae is now recognized by many workers as the primary cause of swine dysentery. Several authors have, however, pointed out that both environmental and dietary factors may play a role in this enteric disease (*Nordstoga et al.* 1968, *Eriksen & Andersen* 1970, *Glock* 1972).

It has been shown that pigs fed a diet deficient in vitamin E were susceptible to an experimental generalized Shwartzman reaction (*Teige et al.* 1973). It has also been suggested that the colonic lesions in swine dysentery may be the result of a local Shwartzman reaction (*Nordstoga et al., Nordstoga* 1973, *Espinasse* 1973), and that one possible factor could be a deficiency of vitamin E (*Nordstoga*).

In a preliminary experiment, 2 groups of pigs were inoculated with minced colon from cases with swine dysentery, and in a group fed a vitamin E deficient diet the inoculation resulted in diarrhoea and reduced appetite, whereas pigs in the other group, which were given vitamin E supplementation, did not show any clinical signs (unpublished observations). The present report presents the results of an experiment in which 2 groups of pigs were inoculated with minced colon from pigs with swine dysentery. Both groups were fed a diet deficient in both vitamin E and selenium and supplemented with cod liver oil, in order to enhance the requirement for these 2 factors. Half of the animals (Group 2) were given a daily supply of vitamin E and selenium.

MATERIALS AND METHODS

Experimental animals

Sixteen pigs with an average weight of 16 kg (13—18.5 kg) were used; the pigs were randomly divided into 2 equal groups (Groups 1 and 2) and given consecutive numbers (Nos. 1—16). All animals came from a herd with no history of outbreaks of swine dysentery. Except for 1 pig which had a transient diarrhoea for a few days on arrival at the experimental station, the pigs had no signs of intestinal disorders during the feeding period and also seemed to be in good health. The experiment was conducted in closed buildings. The pigs were kept in a pen with arrangements for individual feeding, and were handfed twice daily, given an adequate water supply and weighed individually every 2 weeks. The average weight gain per day during the feeding period was 410 and 460 g in Groups 1 and 2, respectively.

Experimental diet

The basic ration given to the pigs of Group 1 had the following composition:

Toprina* 15 %, barley 69 %, oats 13 %, and minerals** and vitamins*** 3 %. The basic ration had the following calculated contents per kg: calcium 7.5 g, phosphorus 6.5 g, lysine 9.1 g, methionine and cystine 5.9 g, and digestible raw protein 133 g. In addition 6.8 % cod liver oil† was incorporated into the basic ration. This oil was subjected to a silica earth absorption treatment as this procedure is supported to reduce the vitamin E content. After this treatment the oil contained 130 mg α-tocopherol per kg. Samples of the basic ration without cod liver oil collected at the beginning of the feeding period contained 14 mg α-tocopherol†† per kg and 0.0065 p.p.m. selenium†††. In a sample of the same feed collected at the end of the experiment no measurable amounts of α-tocopherol were detected††.

The cod liver oil was added to the feed at each feeding. The pigs in Group 2 received the same diet as in Group 1 but, in addition, a daily supply of 200 mg α-tocopherol and 0.1 mg selenium, given as sodium biselenite. At the time of inoculation the pigs had been fed the experimental diets for 53 days.

Laboratory tests

Blood samples were taken from the anterior vena cava at 1, 3, 5, 7 and 8 weeks after the beginning of the feeding period. The following determinations were performed: The values of haematocrit and haemoglobin, and the serum concentrations of

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* Produced by British Petroleum.

** The following were added in g per 100 kg feed: Dicalcium phosphate 700, calcium carbonate 1350, sodium chloride 250, potassium chloride 400, magnesium carbonate 200, ferrous sulphate 60, manganese sulphate 10, copper sulphate 4, cobalt chloride 2, zinc oxide 8.

*** The following were added in mg per 100 kg feed: Ascorbic acid 500, nicotinic acid 1600, calcium pantothenate 1100, inositol 400, choline chloride 35,000, riboflavin 200, biotin 20, folic acid 70, pyridoxine chloride 130, thiamine hydrochloride 200, vitamin B_{12} 1; 200,000 i. u. vitamin A and 20,000 i. u. vitamin D were also added per 100 kg feed.

† Produced by J. C. Martens and Co. Ltd., Bergen, Norway.

†† Analyses performed by Vitamininstituttet, Bergen, Norway.

††† Analyses performed by Institutt for Atomenergi, Kjeller, Norway.

glutamate-oxalacetate transaminase (GOT)*, glutamate-pyruvate transaminase (GPT)* and lactate dehydrogenase (LDH)*. In the last blood sample the plasma α-tocopherol content** was also determined.

Inoculation

The inoculum consisted of colon and caecum from 3 pigs from a herd with persistent swine dysentery. In the 3 pigs, the pathological lesions were in accordance with the picture usually observed in swine dysentery (*Harris & Glock* 1972). By phase-contrast microscopy the large spirochaetes could be seen in relatively large numbers in colonic samples. Bacteriological examinations of the same samples revealed no growth of Salmonella spp.

The intestinal material from the 3 pigs had been stored at —20°C for 2, 3 and 17 days, respectively, when it was thawed, minced, mixed together and divided into equal portions for each pig. Feed and water had been withheld from the pigs for approx. 20 hrs. when this inoculum was mixed with some feed and water in the crib; 3 hrs. later all the pigs had eaten the mixture.

Clinical procedures

Both before and after the inoculation the faecal appearance was observed and scored as shown in Table 3. Post inoculation (p.i.), at least 2 observations on faecal appearance were made each day. The daily food intakes and the incubation times are given in Tables 2 and 3, respectively. Faecal samples were collected *a*) before inoculation, *b*) between inoculation and the onset of diarrhoea and *c*) when diarrhoea appeared. Giemsa's staining was used on smears from specimens collected at time *a*, while specimens collected at time *b* and *c* were examined by phase-contrast microscopy.

Killing

Except for 2 pigs, No. 6 which died and No. 16 which was sent to a slaughter-house, all the animals were killed 72 hrs.

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* Analytical procedures were according to Sigma Technical Bulletins (STB), for GOT and GPT: STB No. 505, 1964, Sigma Frankel (S-F) units, and for LDH: STB No. 500, 1960, Berger-Broida (B-B) units.

** Analytical procedure according to a method described by *Hashim & Schuttringer* (1966).

after they developed diarrhoea, with an intravenous injection of pentobarbital. Details of the necropsy procedures will be given in a subsequent paper. Liver samples were frozen and later analyzed for selenium*.

Microbiological procedures

The pseudomembranes in colonic samples were removed, homogenized and diluted in saline 1:5. Mucosal scrapings from pigs without pseudomembranes were treated similarly. The suspension of mucosal material was centrifuged at low speed for 5 min. and samples from the supernatant examined by phase-contrast microscopy. Dilutions to 1:200 were prepared, and from each dilution inoculation was made onto 5 % blood agar plates, which were incubated anaerobically in Gas Pak jars (BBL) at 37 °C for 4 days. Routine bacteriological examinations, including analyses for salmonella, were also performed.

RESULTS

*Clinical-chemical findings***

Five weeks after the start of the feeding period the first elevation of the serum enzyme values was recorded for pig No. 6 which developed a GOT value of 121. Two weeks later the following pigs in Group 1 showed elevations in some of the serum enzyme values: No. 1: LDH = 11,500, GOT = 260 and GPT = 205, No. 3: GOT = 181, No. 6: LDH = 6900, GOT = 257 and GPT = 144, and No. 8: GOT = 224. In Group 2, 1 single pig, No. 9, showed the following elevation: GOT = 86. The remaining animals all had normal values. The results of the next blood sampling (2 days p.i.) are recorded in Table 1, together with the plasma α-tocopherol determinations***.

Clinical findings after inoculation

Observations on the feed consumption for the first 10 days p.i. are given in Table 2. The pigs surviving beyond this period ate their daily ration.

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* Analytical procedures according to a fluorometric method described by *Ihnat* (1974).

** The serum enzyme tests were performed at the Research Station, Kjeller, Norway.

*** Analyses performed by research fellow J. U. Skaare, the Veterinary College of Norway, Oslo.

Table 1. Concentrations of serum enzymes and values of haematocrit, haemoglobin and plasma α-tocopherol 2 days after the inoculation.

Group	Pig No.	LDH (B-B units)	GPT (S-F units)	GOT (S-F units)	Haematocrit (%)	Haemoglobin (g/100 ml)	Plasma α-tocopherol (μg/ml)
1	1	9900	235	138	34	11.9	—
	2	1730	52	39	37	11.2	1.60
	3	2200	60	70	32	9.3	0.40
	4	1830	35	26	36	10.5	0.75
	5	5800	62	48	34	10.5	1.60
	6	6500	200	250	36	10.8	1.35
	7	1550	44	42	43	11.9	1.35
	8	2700	84	99	30	9.0	1.05
	mean ± s	4026 ± 3049	97 ± 76.6	89 ± 76.8	35 ± 3.9	10.6 ± 1.1	1.16* ± 0.45
2	9	3250	46	41	36	10.1	—
	10	1510	44	67	36	10.5	1.90
	11	2500	61	68	40	11.6	1.90
	12	2500	46	36	35	9.8	2.30
	13	1780	42	31	35	10.7	3.25
	14	1400	61	44	40	11.0	2.90
	15	1380	46	24	32	9.9	1.35
	16	2050	50	40	34	10.1	1.90
	mean ± s	2046 ± 663	50 ± 7.4	44 ± 15.9	36 ± 2.8	10.5 ± 0.6	2.21 ± 0.66

* Statistically different from group 2 ($P \leqq 0.01$).

The mean incubation time for the pigs in Group 1 was 8.5 days and 13.4 days in Group 2 for the 7 pigs developing diarrhoea (Table 3) (statistically different, $P \leqq 0.01$). The last pig (No. 16) in the group was killed 25 days p.i. as no symptoms indicating swine dysentery had been observed. In 5 of the pigs in Group 1 the stools had a watery and bloody character (Table 3). The

Table 2. Daily feed consumption after the inoculation. The figures refer to the number of pigs not eating their feed the day after inoculation. The figure 1 recorded from day 6 to 9 in Group 2 refers to pig No. 9.

Days after inoculation	1	2	3	4	5	6	7	8	9	10
Group 1	0	0	3	2	3	2	3	1	5	7
Group 2	0	0	4	0	0	1	1	1	1	0

enteric disorder seemed, according to the appearance of the excrements, to get worse during the 72 hrs. of observation; pigs Nos. 1 to 6 had a gaunt appearance also. In Group 2 the pigs had, with a single exception (No. 13), either entirely normal or soft faeces on the day of killing; in most of the same pigs the faecal appearance seemed gradually to become normal, and the pigs were otherwise in good health. All the pigs suffered from a sarcoptic infestation which caused only a moderate itching during the feeding period; this itching became more pronounced for a couple of days when the first signs of diarrhoea appeared. Phase-contrast microscopy of faeces collected 3 days p.i. revealed no spirochaetes, but 2 days later large spirochaetes were detected in faecal samples, and after the onset of diarrhoea, large spirochaetes were consistantly found by phase-contrast microscopy.

Pathological findings

A survey of the macroscopical lesions in colon is given in Table 3, together with the occurrence of lesions attributed to vitamin E and selenium deficiency, i.e. yellow fat disease, hepatosis dietetica, skeletal muscle degeneration and myocardial degeneration. A detailed description of the pathological findings will be published in a subsequent paper.

Liver selenium

The content of selenium in the livers is recorded in Table 3*.

Microbiological findings

Phase-contrast microscopy of the suspension from the colonic mucosa revealed numerous large spirochaetes in all the pigs of Group 1; spirochaetes were also recovered from 5 of these pigs (Nos. 1, 2, 3, 4 and 6), by cultivation. These organisms showed morphology, motility and growth characteristics which were in accordance with the description of T. hyodysenteriae (*Harris et al.* 1972). In Group 2, large spirochaetes were found by phase-contrast microscopy in 7 of the pigs. (Nos. 9 to 15); the number of spirochaetes, however, was moderate in these animals and by

* Analyses performed at the Department of Toxicology, National Veterinary Institute, Oslo.

Table 3. Weights of the pigs, clinical observations, necropsy findings and liver selenium values.

Group	Pig No.	Weight[1] (kg)	Incubation period (days)	Faecal appearance[2] 1	2	3	4	Lesions in the spiral colon[3]	Lesions indicating vit. E/selenium deficiency[4]	Liver selenium (μg/g)
	1	49	7	††	††	††	††	*****	YF, —, MD, HD.	0.03
	2	46	7	††	††	†††	†††	*****	YF, SMD, —, —.	0.02
	3	48	7	†	††	††	†††	*****	YF, SMD, MD, —.	0.02
1	4	44	8	†	††	††	†††	****	YF, —, MD, —.	0.02
	5	46	8	††	††	†††	†††	*****	YF, SMD, MD, —.	0.02
	6	41	8	††	††	†††	D[5]	*****	YF, SMD, MD, HD.	≦ 0.02
	7	37	11	†	†	†	†	***	YF, —, —, —.	0.02
	8	47	12	†	n	n	†	**	YF, SMD, —, —.	≦ 0.02
	mean	45	8.5	—	—	—	—	—		0.02
	9	42	8	†	†	††	†	**	—	0.16
	10	51	10	††	††	††	†	**	YF	0.12
	11	49	11	†	†	†	†	*	YF, SMD	0.13
	12	43	12	†	††	†	†	**	YF	0.11
2	13	37	13	†	††	††	††	**	YF	0.13
	14	50	14	†	†	††	n	*	YF, SMD	0.12
	15	55	14	†	†	†	n	*	YF	0.10
	16	55	> 25	—	—	—	—	0	YF	0.17
	mean	48	13.4	—	—	—	—	—		0.13

[1] Weight of the pigs at the end of the feeding period.

[2] Observations on the faecal appearance the first day the pigs had diarrhoea are noted in column 1, the same observations on the next 3 days are noted in columns 2, 3 and 4, respectively.
Grading of the faecal appearance: n: normal
†: soft
††: watery
†††: watery and bloody

[3] Grading of the lesions in the mucosa of the spiral colon:
*: A catarrhal inflammation.
**: Few pseudomembranes in the first half.
***: Numerous small pseudomembranes in the first half.
****: Diffuse pseudomembranes in the first half and many small pseudomembranes in the second half.
*****: Diffuse pseudomembranes in all parts.

[4] YF: yellow fat disease. HD: hepatosis dietetica. SMD: skeletal muscle degeneration and MD: myocardial degeneration.

[5] D: died, pig No. 6 was found dead 3 days after the onset of the diarrhoea, the necropsy findings included pronounced hepatosis dietetica.

cultivation the spirochaete was demonstrated in only 1 of the pigs (No. 9).

Salmonella spp. or other pathogenic enterobacteria were not isolated from any of the pigs.

DISCUSSION

In the present experiment it is demonstrated that oral inoculation with minced colon from cases of swine dysentery results in both clinical signs and patho-morphological changes accepted as typical for this disease (*Harris & Glock* 1972). As a large spirochaete was readily observed in colonic samples from the same pigs, the diagnosis of swine dysentery can definitely be established (*Harris* 1974). Our observations indicate, however, that the pigs in the 2 groups reacted rather differently to the inoculation, as the incubation time was shorter in Group 1 than in Group 2, and the clinical signs were also considerably more pronounced in the pigs of Group 1 as the animals had a bloody, stained stool before killing. They also developed an unhealthy appearance and reduced appetite during the course of the illness, whereas the diarrhoea in Group 2 was mild and seemed to decline, and the pigs looked otherwise healthy. The clinical difference between Group 1 and 2 was also in good agreement with the patho-morphological lesions observed.

Animals in both groups were fed a basic ration, which, according to other investigations (*Van Vleet et al.* 1970, *Trapp et al.* 1970), had an insufficient content of vitamin E and selenium. The feeding procedure, which in Group 2 included a daily supply of the 2 deficient factors, resulted in both higher liver selenium and plasma α-tocopherol in this group than in Group 1. The necropsy findings also indicated a higher degree of vitamin E and selenium deficiency in Group 1 than in Group 2, as the patho-morphological lesions considered as typical for deficiency of these factors (*Nafstad & Tollersrud* 1970, *Jenkins & Hidiroglou* 1972) were much more pronounced in the pigs of Group 1.

A preliminary investigation indicated that treatment with vitamin E reduced the susceptibility to oral exposure of colonic material from cases of swine dysentery (unpublished observations). The observations in the present experiment indicate that a mixed supply of vitamin E and selenium has a similar effect. An influence of selenium on the course of enteritis has occasional-

ly been mentioned in the literature. *Andrews et al.* (1968) thus reported the occurrence of profuse diarrhoea in cows with selenium deficiency. It was further shown that administration of selenium could prevent and cure the diarrhoea (*Jolly* 1960). *Oksanen* (1965) observed that diarrhoea was a common clinical sign in all age groups of cattle with nutritional muscular degeneration and that inflammatory changes were present in the digestive tract on post-mortem examination of these animals.

An influence of vitamin E on enteritic disorders does not seem to have been investigated, but a positive effect of supplementation with vitamin E has recently been observed on certain infectious disorders outside the alimentary tract. *Tvedten et al.* (1973) thus showed that rats supplemented with both vitamins E and A were more resistant than rats given rations deficient in either one or both vitamins. In small experimental animals, both vitamin E and selenium administrations have been shown to increase the immune response (*Tengerdy et al.* 1972, 1973, *Spallholz et al.* 1973). *Heinzerling et al.* (1974) showed that vitamin E caused an increased protection against an Escherichia coli infection in chickens, and that this effect had, at least in part, an immunological background. In pigs, *Ellis & Vorhies* (1976) found that vitamin E supply gave an increased serologic response to an E. coli bacterin.

Immunological factors are probably also involved in swine dysentery (*Olson* 1974, *Glock et al.* 1976, *Schwartz & Glock* 1976). Seen in connection with the above information, it seems possible that there may have been a difference in the immune response between the pigs in the 2 groups in this experiment. This assumption seems to be supported by the observation that a more moderate anaphylactic reaction was provoked in pigs fed a vitamin E deficient ration than in pigs given feed supplemented with vitamin E (*Teige & Nordstoga* 1977). Microbes other than T. hyodysenteriae may, however, also be involved in the development of swine dysentery (*Hamdy & Glenn* 1974, *Griffin et al.* 1976, *Alexander et al.* 1976) and the participation of such microbes may also have been influenced by immunological factors.

Other possible effects of selenium and vitamin E deficiency which may have played a pathogenetic role in this experiment will be discussed in subsequent reports.

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SAMMENDRAG

Diettens innflytelse ved eksperimentell svinedysentery. 1. Betydningen av en vitamin E- og selen-fattig diett med tilskudd av 6.8 % tran.

Seksten griser fikk et vitamin E- og selen-fattig fôr, hvorav den ene halvparten (gruppe 2) fikk et daglig tilskudd av de to manglende næringsfaktorene. Etter at grisene hadde blitt fôret med disse diettene i 53 dager, ble de podet med oppmalt colon fra tilfelle av svinedysenteri. Denne podning resulterte i utbrudd av svinedysenteri i begge grupper. Imidlertid var inkubasjonstiden kortere og de kliniske symptomer mer uttalte i gruppe 1 enn i gruppe 2. De patologiske forandringer varierte også mellom gruppene. Hos grisene i gruppe 1 ble tydelige pseudomembranøse forandringer sett i colonspiralen. I gruppe 2 fantes hovedsakelig en katarrhalsk betennelsestilstand; pseudomembraner såes bare i en avgrenset del av colon hos 4 av grisene. Både de analysene som er utført og de patologiske forandringer som er funnet indikerer en bedre vitamin E og selen status hos grisene i gruppe 2. Det blir derfor konkludert med at tilskuddet av vitamin E og selen i stor grad økte motstandskraften overfor svinedysenteri.

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