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INFLUENCE OF DIET ON EXPERIMENTAL SWINE DYSENTERY

2. EFFECTS OF A VITAMIN E AND SELENIUM DEFICIENT DIET SUPPLEMENTED WITH 3 % COD LIVER OIL, VITAMIN E OR SELENIUM

By

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TEIGE, JON jr., FINN SAXEGAARD and ARNE FRØSLIE: *Influence of diet on experimental swine dysentery. 2. Effects of a vitamin E and selenium deficient diet supplemented with 3 % cod liver oil, vitamin E or selenium.* Acta vet. scand. 1978, *19*, 133—146. — Two experiments (Exps. II and III) were performed with colonic material from swine dysentery as inoculum. The results of Exp. II showed that the inoculation produced less pronounced clinical signs and patho-morphological lesions and also affected fewer pigs in the groups supplemented with vitamin E and selenium than in the group not given a corresponding supplementation. It is therefore concluded that the daily supply of these 2 nutritional factors significantly increased the resistance to swine dysentery. The cod liver oil incorporated in the diet of 2 groups in Exp. II had also a positive effect in this respect. In Exp. III the inoculation of the pigs fed only the basic ration (Group 1) produced relatively moderate clinical signs and patho-morphological lesions in half of the animals, the results in the rest of the group are, however, in accordance with the observations in the corresponding groups of the 2 preceding experiments. The other results of Exp. III indicate an increased resistance to swine dysentery in the group with selenium as the only supplement. However, no equivalent condition could be demonstrated when a similar supplement of vitamin E was used. The pigs in Exp. III given both nutritional factors showed perhaps the best resistance to swine dysentery, especially when the patho-morphological lesions are taken into consideration.

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

In a previous experiment (Exp. I) supplementation of vitamin E and selenium was found to increase the resistance to swine dysentery in animals given a basic ration deficient in these factors (*Teige et al.* 1977). The pathological and clinical findings indicated deficiency of vitamin E or selenium in all the pigs in the experiment. The deficiency was, however, moderate in the

group with supplementation, and more pronounced in the pigs given only the basic ration.

The present experiments (Exps. II and III) were performed to study in more detail, the influence of diet on swine dysentery. The aims of Exp. II were primarily to test further the experimental model used previously in Exp. I, to induce more moderate lesions indicating deficiency in receiving diets without supplementation of vitamin E and selenium and to reach a better balance of these factors in pigs given supplementation. To achieve this purpose, the quantity of cod liver oil incorporated in the diet was reduced or omitted, while the daily supplement of selenium was doubled. An additional aim was to look for a dietetic influence of the cod liver oil disregarding its effect on the vitamin E requirement. No cod liver oil was incorporated in the diet used in Exp. III. The aim of this experiment was to test the effect of vitamin E and selenium given separately compared with none or a combined supply of the 2 nutritional factors.

MATERIALS AND METHODS

Experimental animals

In each of the 2 experiments, 24 pigs were used. They had average weights of 15.9 kg and 14.6 kg in Exps. II and III, respectively. The animals were randomly divided into 4 equal groups, (Groups 1, 2, 3 and 4) in both experiments and were given consecutive numbers (Exp. II: Nos. 17—40, Exp. III: Nos. 41—64). The pigs came from the same herd as those in Exp. I (*Teige et al.* 1977). The animals of Exp. II seemed to be in good health during the time from purchase to inoculation. In Exp. III a soft to watery faecal consistency for 2 days was seen in pigs Nos. 41 and 43, 14 and 38 days, respectively, after the start of the feeding period. In both cases the slight haemolytic variant of Treponema hyodysenteriae was isolated using the selective medium of *Songer et al.* (1976). In the same period a somewhat reduced appetite for 1 or more days was observed for 6 pigs (Nos. 41, 44, 45, 48, 59 and 60).

The animals were weighed individually every 2 weeks. The average weight gain per day during the feeding period was in Exp. II, Group 1: 567 g, Group 2: 571 g, Group 3: 523 g, Group 4: 561 g and in Exp. III, Group 1: 318 g, Group 2: 368 g, Group 3; 372 g, Group 4: 368 g.

Twelve days prior to inoculation in Exp. III rectal swabs were applied. The bacteriological examination of these swabs revealed strains of the slightly haemolytic variant of T. hyodysenteriae in 16 pigs (Nos. 41—44, 46, 47, 49, 50, 52—54, 56, 57, 59—61).

Experimental diets

The basic ration had the same composition as in Exp. I. The barley and oats were harvested on the same farm in all 3 experiments and the grain used in Exps. I and II was given from the same harvest. The basic ration was given to all the pigs, and the following supplements were given:

Exp. II
Group 1: 3 % cod liver oil.
Group 2: 3 % cod liver oil, 200 mg α-tocopherol and 0.2 mg selenium (given as sodium biselenite) per pig and day.
Group 3: None.
Group 4: 200 mg α-tocopherol, 0.2 mg selenium per pig and day.

Exp. III
Group 1: None.
Group 2: 200 mg α-tocopherol per pig and day.
Group 3: 0.2 mg selenium per pig and day.
Group 4: 200 mg α-tocopherol, 0.2 mg selenium per pig and day.

In Exp. II a sample of the basic ration collected at the start of the feeding period contained 10 mg α-tocopherol per kg. During the feeding period small samples from each feed bag with basic ration were collected and mixed together. In Exps. II and III this mixture contained only traces of and 29 mg α-tocopherol per kg, respectively*.

The cod liver oil** incorporated in the diet of Exp. II was subjected to the silica earth absorption method used previously. After this procedure the oil contained 275 μg α-tocopherol per g*.

The feeding procedures and the buildings used were the same as in Exp. I. At the time of inoculation the pigs had been fed the experimental diets for 70 and 44 days in Exps. II and III, respectively.

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* Analyses performed by Vitamininstituttet, Bergen, Norway.
** Produced by J. C. Martens and Co. Ltd., Bergen, Norway.

Laboratory tests

Blood samples were usually taken every 2 weeks during the feeding period. The tests performed and the procedures used were the same as in Exp. I.

Inoculation

In Exp. II the inoculum consisted of colon and caecum from 3 of the pigs in Exp. I (Nos. 1, 2 and 3) and small parts of colon from 3 other pigs suffering from swine dysentery. In Exp. III, caecum and colon from pigs Nos. 17 and 18 in Exp. II and a third one which had developed swine dysentery after being fed a culture of T. hyodysenteriae, were used as inoculum. Bacteriological examination of the colonic samples from the different pigs revealed no Salmonella sp. Strongly haemolytic strains of T. hyodysenteriae were isolated by the use of the selective medium of *Songer et al.* The material for inoculation had been stored for several weeks at —70°C before it was thawed, minced, mixed together and divided into portions for each pig. The inoculum was then mixed with some feed and water in the crib. The pigs ate most of this mixture within an hour.

Clinical procedures

After the inoculation, the pigs were inspected at least twice each day. The incubation period and the daily feed consumption were noted and the faecal appearance graded according to the description given in Table 2.

Killing

Approx. 72 hrs. after the pigs had developed diarrhoea they were killed by an intravenous injection of pentobarbital. Pigs with normal faecal appearance were observed for 30 days after the inoculation before they were slaughtered. Details from the necropsy will be given in a subsequent paper. Liver samples were analyzed for selenium according to the same method as in Exp. I.

Microbiological procedures

After necropsy a piece of colon was submitted to bacteriological examination. Phase-contrast microscopy and routine bacteriological investigations were performed with the intestinal mu-

cosa. For the isolation of T. hyodysenteriae the selective medium TSA-S400 of *Songer et al.* was used.

This medium consisted of trypticase soy agar (BBL) to which 5 % goat blood was added; 400 µg of spectinomycin per ml was added to inhibit growth of other faecal bacteria than T. hyodysenteriae. Inoculated plates were incubated anaerobically in GasPak jars (BBL), at 42°C for 2 to 4 days.

RESULTS

*Clinical-chemical findings**

The values for serum enzymes, haematocrit, haemoglobin and plasma α-tocopherol in the last blood sample taken before

Table 1. Concentration of serum enzymes and values of haematocrit, haemoglobin and plasma α-tocopherol before the inoculation*. Group mean ± s.

Exp.	Group	LDH (B-B units)	GOT (S-F units)	GPT (S-F units)	Haemato-crit (%)	Haemo-globin (g/100 ml)	Plasma α-tocopherol (µg/ml)
II	1	2035 ± 264	77 ± 73	79 ± 23	37 ± 0.8	10.4 ± 0.6	1.1 ± 0.3
	2	2117 ± 133	45 ± 7	83 ± 24	38 ± 2	10.8 ± 0.4	3.1 ± 0.4
	3	2100 ± 217	43 ± 21	75 ± 18	35 ± 2	9.5 ± 0.8	0.9 ± 0.7
	4	1950 ± 77	33 ± 12	81 ± 27	37 ± 2	10.4 ± 0.9	2.6 ± 1.3
III	1	1390 ± 225	28 ± 6	45 ± 9	39 ± 2	11.3 ± 0.8	0.7 ± 0.4
	2	1403 ± 173	25 ± 7	45 ± 8	36 ± 3	10.4 ± 1.3	2.8 ± 0.3
	3	1372 ± 159	22 ± 4	43 ± 10	37 ± 1	11.0 ± 0.4	1.2 ± 0.2
	4	1530 ± 112	25 ± 3	45 ± 29	37 ± 1	11.0 ± 0.5	3.1 ± 1.0

* The blood samples in Exp. II were taken the same day as the inoculation, in Exp. III 5 days prior to the inoculation.

* The serum enzyme, haematocrit and haemoglobin analyses were performed at the Research Station, Kjeller, and the plasma α-tocopherol analyses were performed by research fellow J. U. Skaare, Veterinary College of Norway, Oslo.

the inoculation are noted in Table 1 for both experiments. A GOT value of 215 was found in pig No. 18 at that time. This animal was the only one which had a marked elevated serum enzyme value. In pig No. 35 the plasma α-tocopherol was measured to be 2.2 μg/ml. In the other pigs of the same group the values varied between 0.3 and 0.7 μg/ml. The clinical-chemical values in blood samples taken during the feeding period revealed no marked differences between the groups of pigs.

Clinical findings

Exp. II. Four of the pigs in Group 3 had a reduced feed consumption the last 2—3 days before they died or were killed. In the other groups the feed consumption was normal or only slightly reduced during the course of the illness. The incubation period was 12.3, 12.7 and > 25 days in Groups 1, 3 and 4, respectively (Table 2) and showed the same pattern of variation in Groups 1 and 3. A bloody and watery faecal material was observed in the pigs of Groups 1 and 3, although more pigs were affected in the latter group (Table 2). In the pigs of Group 4, only soft or watery stools appeared which were also mixed with some mucus in 2 animals (pigs Nos. 35 and 36). At the time the pigs with swine dysentery were killed, or died, they had average weights which were approx. 6 kg below an estimated weight based on similar weight gain rates in the experimental and feeding periods. The reduced weight effect was somewhat more pronounced in Group 3 than in Groups 1 and 4. In the pigs in which the faeces had a normal appearance after the inoculation, the weight gains were normal. The pigs in Group 3 particularly, developed a gaunt appearance during the course of the illness. Some of them, including pigs Nos. 33 and 34 also became depressed after the onset of the diarrhoea. The affected pigs in the other groups remained alert or only mildly depressed. Pigs No. 38 developed no clinical signs indicating swine dysentery before slaughter. All the pigs suffered from a mild sarcoptic infestation.

Exp. III. Reduced feed consumption was noted in 11 pigs (42, 45—48, 50, 54, 56, 57, 59 and 62) the last 2 days before killing. These animals were at the same time somewhat depressed and a few of them also developed a moderate gaunt appearance. The following mean values were noted for the incubation period: 7.3, 9.8, > 14 and > 13 in Groups 1, 2, 3 and 4, respec-

Table 2. Weights, clinical observations, necropsy findings and liver selenium values (wet weight) for Experiment II.

Group	Pig No.	Weight[1] (kg)	Weight[2] (kg)	Incubation period (days)	Faecal appearance[3] 1	2	3	4	Lesions in the spiral colon[4]	Isolation of T. hyodysenteriae	Lesions indicating vit. E/selenium deficiency[5]	Liver selenium (µg/g)
	17	13.0	48.0	6	††	†††	†††	††	*****	+	SMD, MD	0.03
1	18	14.4	52.5	8	††	††	††	†††	****	+	SMD, MD	0.05
	19	16.6	56.0	9	†	n	n	n	—	+	SMD, MD	0.02
(cod	20	15.5	58.0	13	†	†††	†††	†††	***	+	SMD, MD	0.02
liver	21	18.5	59.0	17	†	††	††	†	***	+	SMD, —	0.04
oil)	22	18.0	60.5	21	†	†	†	n	***	+	— —	0.04
	mean	16.0	55.7	12.3								0.03
2	23	14.0	57.0	>30					—	—	SMD	0.36
(cod	24	15.0	54.0	>30					—	+	—	0.33
liver	25	15.0	54.0	>30					—	—	—	0.29
oil +	26	15.5	58.0	>30					—	+	—	0.44
vit. E	27	19.0	60.0	>30					—	—	—	0.41
+ Se)	28	16.5	52.0	>30					—	—	—	0.36
	mean	15.8	55.8	>30								0.37
	29	13.5	45.5	5	†	†††	††	†††	*****	+	SMD, MD	0.03
	30	14.0	48.0	7	†	††	††	†††	*****	+	SMD, MD	0.04
3	31	16.5	56.0	10	†	††	†††	††	****	+	SMD, —	0.04
	32	15.5	55.0	15	††	††	††	†††	****	+	SMD, MD	0.03
	33	17.5	54.0	19	††	††	†††	D[6]	*****	+	SMD, MD	0.04
	34	18.0	56.0	20	††	†††	D[6]		*****	+	SMD, —	0.05
	mean	15.8	52.4	12.7								0.04
	35	15.5	52.0	15	†	††	††	††	**	+	—	0.33
4	36	15.0	51.0	22	†	††	††	††	***	+	—	0.32
	37	16.5	58.5	23	†	†	†	n	**	+	—	0.32
(vit.	38	16.0	57.5	30	—				*	+	—	0.42
E +	39	18.0	51.0	>30					—	—	—	0.42
Se)	40	14.5	55.0	>30					—	+	—	0.44
	mean	15.9	55.2	>25								0.38

[1] Weights of the pigs at the beginning of the feeding period.
[2] Weights of the pigs at the end of the feeding period.
[3] Observations on faecal appearance the first day the pigs had diarrhoea are noted in column 1, the same observation 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
[4] 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.
[5] SMD: Skeletal muscle degeneration. MD: myocardial degeneration. Yellow fat disease or hepatosis dietetica were not demonstrated.
[6] D: Died, pigs Nos. 33 and 34 were found dead 3 and 2 days, respectively, after the onset of diarrhoea.

tively (Table 3). Watery and bloody faeces were seen in 8 pigs, 4 in Group 2, and 2 in each of Groups 3 and 4 (Table 3). In the other pigs developing swine dysentery the faeces were soft or watery. Reduced weight was also observed in most of the pigs with swine dysentery in this experiment at the time they were killed. This reduction was between 4 and 5 kg in pigs with watery and bloody faeces and more moderate in the other animals. Pigs Nos. 58 and 64 showed no clinical signs after the inoculation.

Pathological findings

A survey of the macroscopical lesions in colon, and the lesions indicating vitamin E and selenium deficiency are recorded in Tables 2 and 3. Further details on the pathological findings will be described in a subsequent paper.

Liver selenium

The contents of selenium in the livers are recorded in Tables 2 and 3*.

Microbiological findings

Routine diagnostic procedures revealed growth of a flora predominated aerobically by non-haemolytic Escherichia coli. On the TSA-S400 medium slight to heavy growth of strongly haemolytic T. hyodysenteriae appeared in pure culture in most cases (Tables 2 and 3). The growth of T. hyodysenteriae, partly as a film on the agar surface and partly in the depth of the agar, was confirmed by phase-contrast microscopy.

DISCUSSION

The experimental model used in this investigation is similar to that used previously (*Teige et al.* 1977). The inoculation resulted in outbreaks of swine dysentery according to the criteria given by *Harris et al.* (1976).

In Exp. II, the most pronounced clinical signs and pathomorphological lesions were observed in the pigs given the basic ration (Group 3). These animals had watery and bloody faeces,

* The analyses were performed with financial support from the Agricultural Research Council of Norway.

Table 3. Weights, clinical observations, necropsy findings and liver selenium values (wet weight) for Experiment III.

Group	Pig No.	Weight[1] (kg)	Weight[2] (kg)	Incubation period (days)	Faecal appearance[3] 1	2	3	4	Lesions in the spiral colon[4]	Isolation of T. hyodysenteriae	Lesions indicating vit. E/selenium deficiency[5]	Liver selenium (µg/g)
	41	15.5	29.0	3	†	†	†	n	*	+	SMD, MD	0.03
	42	16.5	28.0	3	†	†	†	††	***	+	—, MD	0.03
1	43	13.0	28.0	4	†	†	†	†	**	+	—, MD	0.05
	44	14.0	29.0	8	††	†	†	†	**	+	—, —	0.03
	45	12.5	28.0	11	††	††	††	††	***	+	SMD, MD	0.04
	46	16.0	29.5	15	†	†	†	††	****	+	SMD, MD	0.03
	mean	14.6	28.6	7.3								0.04
	47	14.5	31.5	8	††	††	†††	†††	****	+	SMD, —	0.04
	48	13.5	28.0	8	†	††	†††	†††	****	+	—, —	0.03
2	49	16.5	34.5	9	†	n	††	††	****	+	—, —	0.05
(vit.	50	14.5	30.0	10	††	††	†††	††	****	+	—, —	0.03
E)	51	12.0	28.5	11	†	††	†††	†††	****	+	—, —	0.03
	52	16.5	32.0	13	†	†	††	††	***	+	—, —	0.04
	mean	14.6	30.8	9.8								0.04
	53	12.0	29.0	8	††	††	††	††	***	+	—, —	0.38
	54	17.0	33.5	8	††	††	†††	†††	****	+	—, —	0.39
3	55	14.0	28.5	10	††	†	†††	††	****	+	SMD, —	0.38
(Se)	56	12.0	31.0	10	†	n	††	†	**	+	—, —	0.33
	57	15.0	31.0	17	†	††	††	††	*	+	—, —	0.31
	58	17.5	32.5	>30					—	—	—, —	0.53
	mean	14.6	31.0	>14								0.39
	59	12.0	29.5	8	††	††	†††	†††	***	+	—, —	0.38
	60	14.5	31.0	9	†	†	†	†	*	+	—, —	0.36
4	61	16.0	33.5	9	†	††	††	††	*	+	—, —	0.36
(vit. E	62	14.5	31.5	10	††	††	†††	†††	**	+	—, —	0.41
+ Se)	63	14.5	31.0	13	†	††	††	††	**	+	—, —	0.36
	64	16.0	33.0	>30					—	—	—, —	0.39
	mean	14.6	31.6	>13								0.38

[1], [2], [3], [4] and [5]. See Table 2.

reduced feed consumption and a gaunt appearance and diffuse pseudomembranes in the colon. Two of the pigs in this group probably died from swine dysentery. The clinical signs and the colonic lesions observed in the other pigs given a deficient diet in Exp. II were also typical for swine dysentery but less pronounced than in Group 3. Only 4 of the pigs in Exp. II, which

were fed a diet supplemented with α-tocopherol and selenium, developed swine dysentery. The mean incubation period in these pigs was significantly longer than in the other animals in the experiment. Watery or soft faeces was the only clinical sign and the pseudomembranes were small and of limited distribution.

The analyses of liver selenium and plasma α-tocopherol in Exp. II were in good accordance with the diets in the different groups. On the basis of the clinical and pathological findings, it may be concluded that the supplementation of α-tocopherol and selenium greatly increased resistance to swine dysentery in this experiment. An equivalent response to a similar supplementation was seen in Exp. I (*Teige et al.*). Some of the dietary components were changed from Exp. I to Exp. II. These arrangements seem to have improved the resistance to swine dysentery in Groups 2 and 4 in Exp. II, compared with the corresponding group in Exp. I; this is, however, most clearly demonstrated in the pigs of Group 2.

The deficient and the supplemented groups in Exps. I and II correspond to Groups 1 and 4 in Exp. III, respectively. The mean incubation period was distinctly shorter in Group 1 than in Group 4. The results also show that pseudomembranes were more extensive in Group 1 and that 1 pig of Group 4 did not develop swine dysentery. Regarding the clinical observations, no marked differences between the same groups were observed except for faecal appearance, as 2 pigs in Group 4, and none in Group 1 had a bloody diarrhoea. The results for Groups 1 and 4, Exp. III, are, however, similar to the observations in the preceding experiments, although some pigs of Group 1 had more moderate clinical signs and colonic lesions than the corresponding groups of Exps. I and II.

Three of the pigs in Group 1, Exp. III, had a shorter incubation period than the pigs in Exps. I and II and other experiments using colonic material as inoculum (*Olson* 1974). The clinical signs of swine dysentery have, therefore, probably occurred very early in the development of the disease. The moderate colonic lesions demonstrated at the time of killing may be a result of this situation. Two of the 3 pigs with the short incubation period had diarrhoea during the feeding period. These observations can indicate a labile intestinal state thus forming the basis for the early clinical reaction seen after the inoculation

in the 2 animals. The group of pigs supplemented only with vitamin E, developed the most pronounced clinical signs and colonic lesions after the inoculation in Exp. III. The results in the group were, however, in most respects similar to those seen in the deficient groups in Exps. I and II. This unexpected observation seems to indicate that the vitamin E supplementation used in Group 2, Exp. III, gave no demonstrable protection against inoculation with material from swine dysentery. The results in the same group further support the assumption that the moderate clinical signs and pathological lesions in some pigs of Group 1, Exp. III, are due to unknown factors.

The results of the inoculation in Group 3, Exp. III, supplemented only with selenium, showed relatively large variations with regard to both the clinical signs and the colonic lesions. The findings are, however, less pronounced than those in the group of pigs receiving only vitamin E supplementation. Comparing the results of the selenium supplemented group with the group given both vitamin E and selenium, no great differences are found, except for a more widespread distribution of pseudomembranes in some pigs of the former group.

In Exp. III, the supposed apathogenic haemolytic variant of T. hyodysenteriae was isolated from more than half of the pigs at the end of the feeding period. The results of the inoculation reveal no particular differences between these and the other pigs of the experiment, an observation indicating no apparent influence of the bacteria mentioned in this connection.

Both the liver selenium and plasma α-tocopherol values recorded in Exp. III were in good agreement with the diets in the different groups. Therefore, the results for Groups 2, 3 and 4 indicate that selenium gave a better protection against the inoculation than vitamin E. The results may further indicate that a combined supplementation of vitamin E and selenium seems to have given the best protection against the inoculation. Further experiments will be necessary before a definite answer can be given concerning the role of each of these nutritional factors.

An evident deficiency of selenium developed in the pigs given no supplement, as their liver selenium content was below the level reported in pigs dying from mulberry heart disease and hepatosis dietetica (*Van Vleet et al.* 1970, *Simesen & Pedersen* 1975). The selenium concentrations in the livers of the pigs in Exp. I, Group 2, which were given 0.1 mg selenium daily,

were at the upper limit of values characterized as deficient (*Van Vleet et al., Simesen & Pedersen*). Degeneration in the skeletal muscles was observed in 2 of the pigs in the same group, and the low selenium balance could probably be a predisposing factor for the muscular lesions. It may also be assumed that the cod liver oil in the diet had some effect in this respect, as the pigs developed yellow fat in spite of the α-tocopherol supplementation. Doubling the selenium supplement from Exp. I to Exp. II gave an evident increase in selenium retention in the liver. This observation indicates, according to investigations by *Groce et al.* (1973), that the physiological stores of selenium in Group 2, Exp. I, were not filled. The selenium values in Groups 2 and 4, Exp. II, were, however, on a level considered to be normal in pigs (*Rasmussen* 1974, *Simesen & Pedersen, Underwood* 1977).

The mean incubation period was distinctly shorter in the deficient groups than in those supplemented with vitamin E and selenium in the 3 experiments. The incubation periods were, however, of longer duration in Exp. II than in the corresponding groups of Exps. I and III. The pigs of Exp. II were, on the other hand, 3 to 4 weeks older than in the 2 other experiments when the inoculation took place. This difference may be of importance as the incubation period in swine dysentery seems to increase as the pigs become older (*Olson*).

The observations in Exp. II indicate a less pronounced response to the inoculation in the groups given cod liver oil. Initially, it was thought that the higher levels of plasma α-tocopherol in the pigs of Groups 1 and 2, compared with Groups 3 and 4, respectively, could account for this difference. The results obtained in Exp. III, however, seem to exclude this possibility. It appears, therefore, that cod liver oil is another factor which may enhance resistance to experimental swine dysentery. The results of the present experiments indicate, however, that selenium seems to be the most important factor in this respect, an assumption which is supported by observations in ruminants (*Oksanen* 1965, *Andrews et al.* 1968).

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SAMMENDRAG

Diettens innflytelse ved eksperimentell svinedysenteri.
2. Betydningen av en vitamin E- og selen-fattig diett med tillskudd av 3 % tran, vitamin E eller selen.

To forsøk (Exp. II and III) ble utført med griser podet med kolonmateriale fra tilfelle av svinedysenteri. Resultatene i Exp. II viser at podningen forårsaket mindre uttalte symptomer og patomorfologiske forandringer og også omfattet et mindre antall griser i de grupper som ble gitt tillskudd av vitamin E og selen enn i gruppen som ikke fikk tilsvarende tillskudd. Det er derfor konkludert med at den tilførsel som ble gitt av disse to næringsfaktorene, øket modstandskraften overfor svinedysenteri. Tranen som var iblandet fôret til 2 av gruppene i Exp. II, øket også motstandskraften hos grisene. I Exp. III medførte podningen av dyr fôret med bare grunnfôr (gruppe 1) relativt moderate symptomer og patomorfologiske forandringer hos halvparten av dyrene, resultatene i gruppen for øvrig var imidlertid i samsvar med tilsvarende grupper i de forutgående forsøkene. De

andre resultatene i Exp. III indikerer økt mostandskraft overfor svinedysenteri i den gruppen som hadde fått selen som eneste daglige tilskudd. Derimot kunne man ikke påvise noen slik effekt etter et tilsvarende vitamin E-tilskudd. De grisene i Exp. III som ble gitt begge faktorene, viste kanskje størst motstandskraft overfor svinedysenteri, særlig når en ser på de patomorfologiske forandringene.

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