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From the National Veterinary Institute, Oslo, Norway.

INFLUENCE OF DIET ON EXPERIMENTAL SWINE DYSENTERY

3. PATHOLOGICAL CHANGES

By

Jon Teige jr.

TEIGE, JON jr.: *Influence of diet on experimental swine dysentery. 3. Pathological changes.* Acta vet. scand. 1978, *19*, 506—519. — Three experiments have been carried out to elucidate the possible influence of the vitamin E and selenium content of the feed on experimental swine dysentery. In most of the pigs given a vitamin E and selenium deficient diet, large and diffuse pseudomembranes appeared in the spiral colon, which also usually displayed a distended appearance and prominent oedematous infiltrations in the mesentery. The histological examination revealed large fibrinous pseudomembranes attached to defects on the mucosal surface. There were also pseudomembranes containing necrotic mucosal tissue. Fibrinoid thrombi within minute vessels were readily observed in the latter lesions. The vitamin E supplemented pigs had colonic lesions very much like the deficient animals, while half of the selenium supplemented animals developed none or moderate inflammatory changes, the other half displayed, however, prominent pseudomembranes in the colon. Ten out of 26 pigs supplemented with both vitamin E and selenium were not affected by swine dysentery. In the remaining pigs catarrhal inflammatory lesions dominated in the colonic mucosa. In some of these animals pseudomembranes occurred, but they were usually small and of limited distribution. The vascular thromboses and tissue necrosis demonstrated within the colonic lesions are found to be compatible with a Shwartzman reaction. Erythrocytic "thrombi" and other phenomena associated with stasis are further believed to be of pathogenetic importance in this respect. It is emphasized that this report also illustrates the enhancing effect of a combined supplement of vitamin E and selenium on resistance to swine dysentery.

pig; swine dysentery; pseudomembraneous colitis; diet; vitamin E and/or selenium deficiency; Shwartzman reaction; erythrocytic "thrombi"; stasis.

The influence of vitamin E and selenium on the course of swine dysentery has been studied in 3 experiments previously reported (Exps. I—III, *Teige et al.* 1977, 1978). As the patho-

logical changes observed in the experimental pigs were only briefly mentioned, this report presents both the macroscopical and histological changes in the pigs of all 3 experiments. The colonic lesions are given special attention in order to facilitate a discussion on the pathogenetic mechanism in swine dysentery.

MATERIAL AND METHODS

An outline of the diets used in each group of pigs is given in Table 1. Further information regarding the animals, basic ration, supplements, laboratory tests, and inoculations, clinical and bacteriological procedures are given in the 2 preceding reports (*Teige et al.* 1977, 1978). The pigs are classified as vitamin E and selenium deficient pigs (Group 1, Exp. I; Groups 1 and 3, Exp. II and Group 1, Exp. III), vitamin E or selenium supplemented pigs (Groups 2 and 3, Exp. III) and vitamin E and selenium supplemented pigs (Group 2, Exp. I; Groups 2 and 4, Exp. II and Group 4, Exp. III), according to the diets. Necropsies were usually performed within an hour after sacrifice. Pieces of tissue from liver, kidney, heart and skeletal muscles, stomach, jejunum, colon, fatty tissue, lungs, spleen and adrenal gland were fixed in a 10 % formaldehyde solution, embedded in paraffin and sectioned at about 5 µ. All the sections were stained with haematoxylin and eosin (H & E). Colonic sections were also stained with Lendrum's acid picro-Mallory method, the Martius scarlet blue (MSB) method (*Lendrum et al.* 1962) and the Levaditi method. The Lendrum's acid picro-Mallory method was also used on sections from kidney and stomach (Exps. I and II), while fatty tissue sections were stained with long Ziehl-Neelsen.

RESULTS

Gastro-intestinal lesions

A survey of the mucosal lesions in the spiral colon is given in Table 1.

Vitamin E and selenium deficient pigs

Macroscopical lesions

One animal (No. 19) had no gastro-intestinal lesions; No. 41 had hyperaemic mucosa in the first half of the spiral colon and loose colonic contents. In all the other pigs, the changes were usually considerably more advanced. In 2 animals (Nos. 29 and

Table 1. The diets used in each group of pigs, and a survey of the lesions in the mucosa of the spiral colon.

EXP. I			EXP. II			EXP. III		
Group[a]	Pig No.	Lesions in spiral colon[b]	Group[a]	Pig No.	Lesions in spiral colon[b]	Group[a]	Pig No.	Lesions in spiral colon[b]
1 (6.8 % cod liver oil)	1	×××××	1 (3 % cod liver oil)	17	×××××	1	41	×
	2	×××××		18	××××		42	×××
	3	×××××		19	—		43	××
	4	××××		20	×××		44	××
				21	×××		45	×××
	5	×××××		22	×××		46	××××
	6	×××××	2 (3 % cod liver oil + Vit. E + Se)	23	—	2 (Vit. E)	47	××××
				24	—		48	××××
	7	×××		25	—		49	××××
	8	××		26	—		50	××××
				27	—		51	××××
				28	—		52	×××
2 (6.8 % cod liver oil + Vit. E + Se)	9	××	3	29	×××××	3 (Se)	53	×××
	10	××		30	×××××		54	××××
	11	×		31	××××		55	××××
	12	××		32	××××		56	××
				33	×××××		57	×
				34	×××××		58	—
	13	××	4 (Vit. E + Se)	35	××	4 (Vit. E) + Se)	59	×××
	14	×		36	×××		60	×
				37	××		61	×
	15	×		38	×		62	××
	16	—		39	—		63	××
				40	—		64	—

[a] All the pigs received the basic ration; the nature of the supplements given to the groups of pigs are found in parentheses.

[b] 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.

34) a diffuse hyperaemia was observed in a greater area of the fundic part of the stomach; some other pigs displayed small hyperaemic areas in the fundic mucosa. No lesions were present in the jejunum. Caecum and colon were distended, except for

most of the pigs in Group 3, Exp. II, and the serosal surfaces were congested. Small grey nodules were seen on the serosal surfaces, and the caecal and, especially, the colonic walls were thickened. The contents of the large intestine were most commonly watery and had a greenish colour, although some pigs also displayed haemorrhagic or mucous contents in parts of the colon. The caecal mucosa was covered with diffuse fibrinous pseudomembranes in 8 pigs (Nos. 1—6, 29 and 33) and by scattered individual pseudomembranes in 8 other animals (Nos. 7, 8, 18, 30, 31, 42, 45 and 46). The pseudomembranes were usually loosely attached to the mucosa and quite thick, especially in the spiral colon where a thickness of 3—4 mm was not unusual (Fig. 1). Necrotic pseudomembranes were also demonstrated. They seemed to be most conspicuous in the pigs of Group 3, Exp. II. One of the animals (No. 34) had pseudomembranes consisting of merely necrotic tissue in the major part of the spiral colon. In 2 pigs (Nos. 33 and 34) minor scattered pseudomembranes were also recognized in colon descendens. The pseudomembranes in 2 pigs (Nos. 21 and 44) differed somewhat from the above description, as a very thin, white layer covered the colonic mucosa. There was further hyperaemia and sometimes haemorrhagic lesions in the mucosa of the large intestine; these lesions were, however, most evident in the cranial parts. The lymph nodes of the colon were enlarged and usually had a hyperaemic cut surface. Prominent oedematous lesions appeared in the mesentery of the spiral colon in most of the pigs; this phenomenon was especially pronounced in 3 animals (Nos. 29, 31 and 46) (Fig. 5).

Microscopial lesions

There was hyperaemia, and moderate mononuclear cellular infiltrations in the mucosa of the stomach in some pigs. Many pseudomembranes in the colon consisted of a mixture of mucous, fibrinous material, inflammatory and epithelial cells, erythrocytes and frequently also debris of uncertain origin. These pseudomembranes were often adjacent to widespread mucosal defects, and sometimes to small separate defects on the mucosal surface (Figs. 7 and 8). There were also pseudomembranes containing necrotic mucosal tissue; the extension of these necrotic lesions varied considerably, the most pronounced lesions comprising the major part of the colonic mucosa (Pigs Nos. 33

and 34) (Fig. 9). The number of goblet cells was increased, causing an accumulation of mucous material in the glands of Lieberkühn. The lamina propria exhibited evident hyperaemic changes and also moderate infiltration of mononuclear cells. Small foci with polymorphonuclear leucocytes also appeared in the mucosa. An eosinophilic hyaline material, occluding dilated minute vessels, was frequently seen in areas with necrotic lesions. Solitary follicles were moderately enlarged, owing to cellular hyperplasia. In the most outlying part of submucosa a moderate number of mononuclear cells occurred, and there were often subserosal oedematous infiltrations.

In areas without pseudomembranes, clear spaces, interpreted as oedematous changes appeared regularly below the epithelial cells. The changes previously described in the lamina propria were more moderate in these areas.

In sections stained by the Levaditi method, many large spirochaetes were often found, they were usually observed within the glands of Lieberkühn, and never in intact epithelial cells nor in lamina propria. Several microbes of other morphological shapes were also observed. The hyaline thrombotic material frequently seen in vessels of necrotic mucosal tissue had, in sections, where the acid picro-Mallory and MSB methods had been employed, staining properties as fibrin (Fig. 3). Single, occlusive thrombi were found in intact tissue beneath pseudomembranes, although closely packed erythrocytes, stained red as fibrin, and fibrinoid material were frequently recognized in small vessels of these areas (Fig. 2). Similar observations were occasionally made in submucosal and subserosal vessels and also in minute vessels beneath intact epithelial cells on the mucosal surface.

Vitamin E or selenium supplemented pigs

No lesions appeared in the gastro-intestinal tract of pig No. 58 nor in the stomach or jejunum of the other animals. Three animals (Nos. 49, 50 and 55) had pseudomembranes in the caecal

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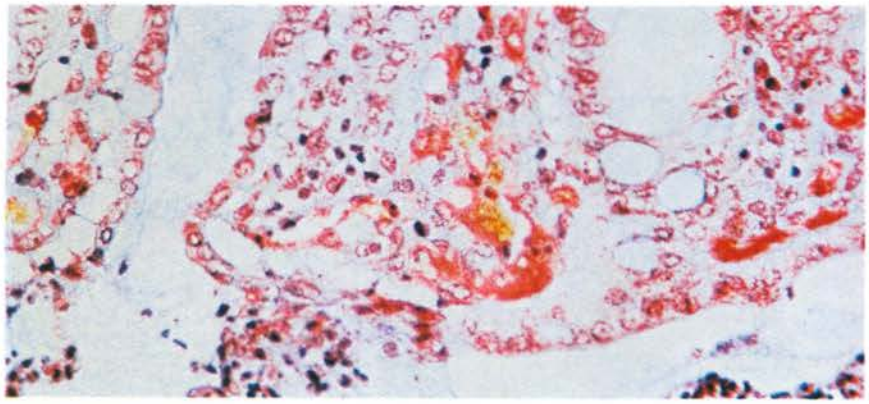
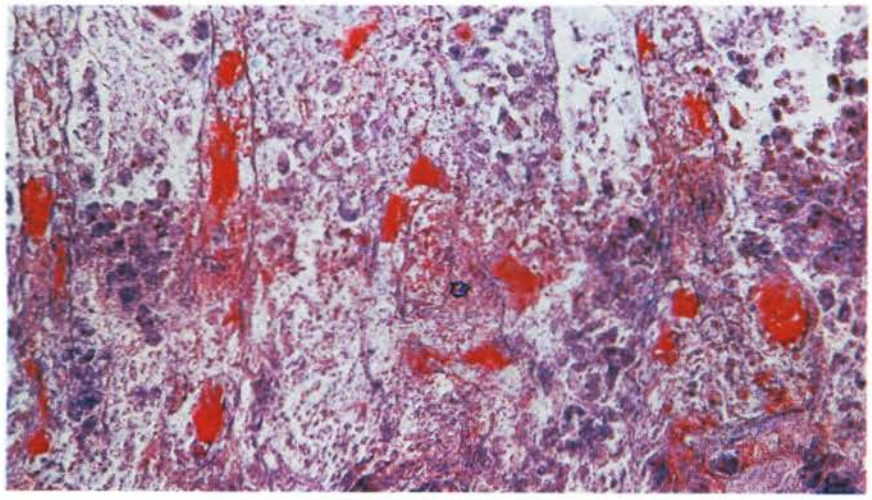
Figure 1. Diffuse fibrinous pseudomembranes on colonic mucosa. Pig No. 2.

Figure 2. Almost intact epithelial layer of colon; oedema below the epithelial cells and fibrinoid material and erythrocytes stained red as fibrin in minute vessels. M S B, × 100.

Figure 3. Necrotic mucosal tissue in colon; many fibrinoid thrombi in dilated vessels. M S B, × 100.





mucosa, in pig No. 55 the lesions had a diffuse distribution, whereas the changes were restricted to a small area in the 2 other pigs. The macro- and microscopical lesions in other parts of the large intestine were in 9 pigs (Nos. 47—55) in accordance with the description for the deficient animals, whereas 2 animals (Nos. 56 and 57) displayed colonic lesions similar to those occurring in most of the vitamin E and selenium supplemented pigs (vide infra).

Vitamin E and selenium supplemented pigs

Macroscopical lesions

Ten pigs (Nos. 16, 23—28, 39, 40 and 64) had no patho-morphological lesions in the gastro-intestinal tract. The following description includes 13 of the other animals (Nos. 9—15, 35, 37, 38, 60, 61 and 63). There were no significant lesions in the stomach nor in the jejunum. These animals had, compared with the deficient pigs, a minor enlargement of the lymph nodes of the colon, a more moderate serosal hyperaemia, a relatively smaller distension of the large intestine and a less pronounced thickening of the intestinal wall. The same pigs had no haemorrhagic contents in the colon, but there was often more mucous material in the colon than demonstrated in the deficient pigs (Fig. 4). Seven out of the 13 animals displayed pseudomembranes (Table 1), which were usually situated in the most cranial parts of colon. These pseudomembranes were smaller and considerably less numerous than seen in the deficient pigs. The colonic lesions of the remaining supplemented pigs (Nos. 36, 59 and 62) were more in accordance with the description for the deficient animals except that only a slight oedema appeared in the mesentery of the spiral colon.

Microscopical lesions

There was often a mucous cover containing numerous necrotic epithelial cells on an intact mucosal surface (Fig. 6). The mucosal lesions or findings were otherwise in accordance with the description for the deficient pigs, but less pronounced. This situation was conspicuous regarding the pseudomembranes as they had a limited size, were attached to small mucosal defects and contained minor parts of necrotic tissue in only 1 pig (No. 59). The sections stained with the acid picro-Mallory and MSB methods revealed few erythrocytes stained red as fibrin and a small amount of fibrinoid material in some mucosal vessels.

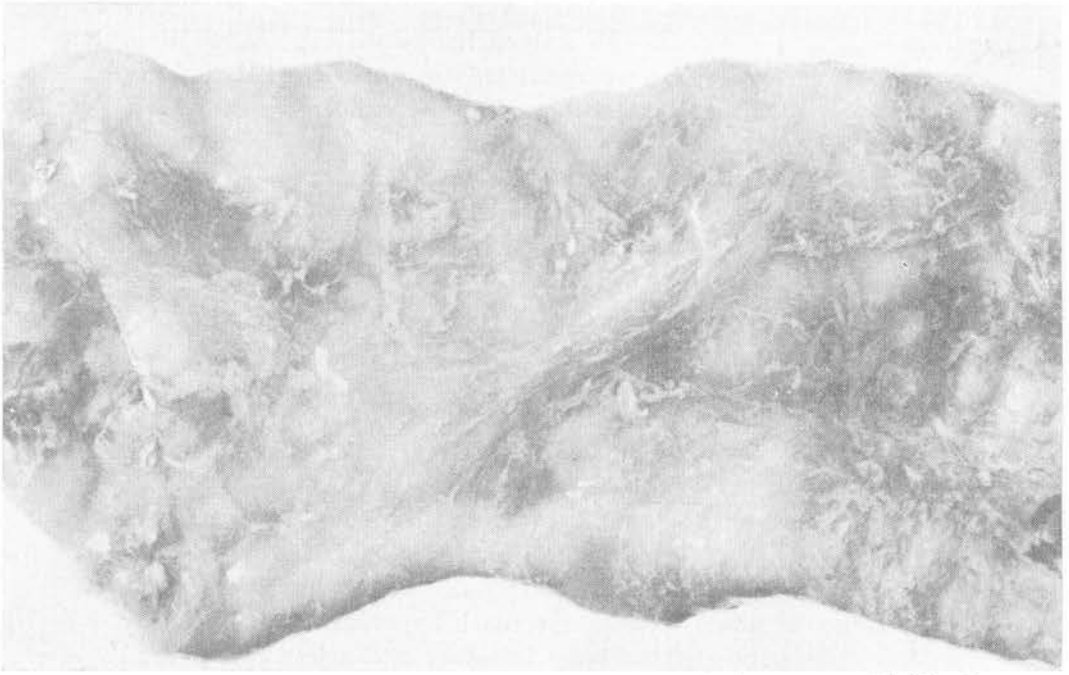


Figure 4. Mucous material covering the colonic mucosa. Pig No. 9.

Figure 5. Extensive oedema in the mesentery of the spiral colon. One of the colonic loops is slitted and exhibits a mucosa covered by pseudomembranes. Pig No. 29.

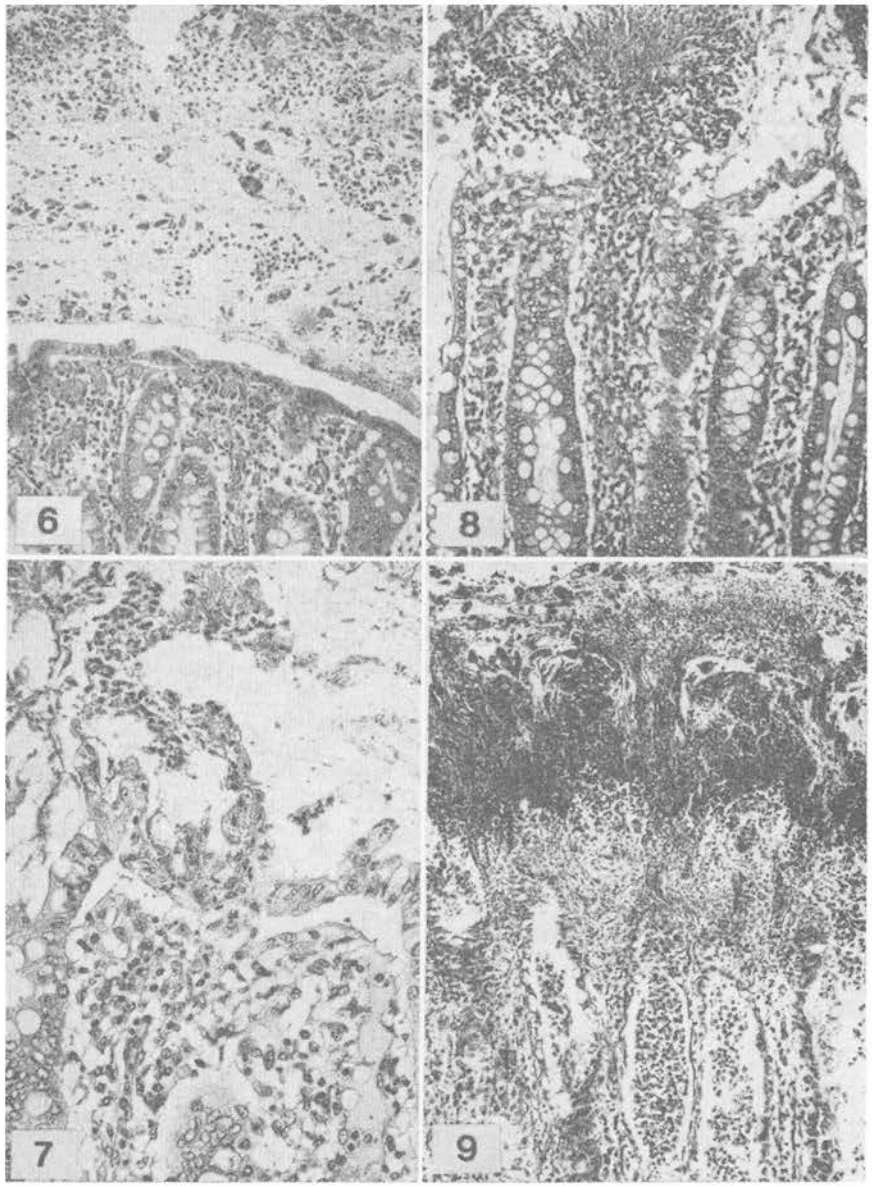


Figure 6. Mucous masses containing necrotic epithelial cells and other cellular debris on the mucosal surface in colon; a slight oedema below the epithelial layer. H & E, × 40.

Figure 7. An early pseudomembraneous lesion in colon. A very thin pseudomembrane is found adjacent to a mucosal defect; extensive oedema below the epithelial layer and moderate cellular infiltrations in the lamina propria. H & E, × 100.

Figure 8. A small pseudomembrane adjacent to a limited mucosal defect in colon. Oedema and cellular infiltrations in the lamina propria, goblet cell proliferation. H & E, × 40.

Figure 9. Advanced colonic lesion. A large part of the mucosa exhibits necrotic changes. Cellular debris in the glands of Lieberkühn. Oedema and cellular infiltrations in the lamina propria. M S B, × 40.

Lesions associated with vitamin E and selenium deficiency

The abbreviations used and the lesions found in the animals of Exps. I, II and III are given in Table 3 (*Teige et al.* 1977) and in Tables 2 and 3 (*Teige et al.* 1978). SMD, HD and YF refers to the histological demonstration of hyaline degenerative lesions in skeletal muscles, massive hepatic necrosis and the occurrence of lipofuscin granules, respectively. MD is used when degenerative changes in myocardial fibres, areas of fibrosis and inflammatory cellular infiltrations were seen. These changes occurred predominantly in the atrial walls. Changes indicating YF were evident in 4 pigs (Nos. 1, 4, 5 and 7). In the other animals with this diagnosis only few lipofuscin granules appeared.

One pig (No. 6) had HD-lesions in all parts of the liver, in another animal (No. 1) only few necrotic lobules were seen. The SMD-lesions were very pronounced or distinct in 5 pigs (Nos. 5, 6, 32, 34 and 46). The other animals with SMD had only moderate or just visible histological changes. Regarding MD, distinct changes extending to many parts of the atrial walls appeared in 7 pigs (Nos. 3, 4, 6, 29, 30, 32 and 43). Only minor or moderate myocardial lesions were observed in the other animals where this diagnosis was made.

Other lesions

Experiment I. Four animals (Nos. 6, 8, 13 and 15) had been the subject of an operation before inoculation in order to obtain biopsies of the colon. Around the surgical wounds in the peritoneum fibrotic adhesions had developed. These lesions, which were more pronounced in pigs Nos. 6 and 8 than in the other 2, caused no narrowing of the large intestine.

Experiment II. Four pigs (Nos. 33, 34, 35 and 40) had been the subject of the same surgical procedure as in Exp. I. The operation caused large fibrotic adhesions in the peritoneum of pig No. 34. Similar but less pronounced lesions were demonstrated in pig No. 33. Only minor peritoneal lesions appeared in the 2 other pigs.

Experiment III. Pig No. 41 showed diffuse fibrotic adhesions in the pericardial sac. In pars oesophagea of the stomach distinct ulcerative and erosive lesions were observed in 3 pigs (Nos. 43, 45 and 55) and 2 pigs (Nos. 41 and 42), respectively.

DISCUSSION

The large intestine in the great majority of the vitamin E and selenium deficient pigs revealed a pronounced fibrinous inflammation and also necrotic lesions. In the vitamin E and selenium supplemented groups fewer animals were affected by swine dysentery and in those developing the disease the colonic changes consisted predominantly of catharrhal inflammatory lesions. Pseudomembranes were certainly observed in some of these pigs, but the membranes were usually small and of scattered distribution. There was no obvious difference in the colonic lesions between the vitamin E supplemented and the deficient pigs. The colonic lesions in animals supplemented with only selenium were variable, as half of the pigs developed moderate or no mucosal changes and the other half displayed prominent fibrinous membranes.

It should be emphasized that the pseudomembraneous lesions in the deficient pigs also were pronounced when compared with observations during natural outbreaks of swine dysentery (*Lussier* 1962). The apical region of the spiral colon is described as the most prevalent site of lesions in swine dysentery (*Hughes et al.* 1975). In the present study the pseudomembranes were often situated in the most cranial part of colon in pigs with a limited fibrinous inflammation in the mucosa. In pigs with more extensive lesions the first half of the spiral colon was often uniformly affected. The second half was also the site of a fibrinous inflammation, but usually to a lesser degree than in the more cranial parts.

Olson (1974) found that the caecal lesions in swine dysentery first appeared in pigs euthanized on the fourth day of diarrhoea. This observation could probably explain the relatively moderate inflammatory changes in caecum of many pigs in the present experiments as our animals were killed 72 hrs. after the beginning of the diarrhoea. Advanced caecal lesions were, however, often present in the same animals as those which showed the most pronounced alterations in the colon.

Oedema in the mesentery of the spiral colon was recognized almost exclusively in pigs given diets deficient in vitamin E and/or selenium. The oedema is probably mostly a secondary phenomenon to the extensive colonic lesions in these pigs, but it must be considered, whether vitamin E or selenium deficiencies have been a contributing factor in this respect, as vascular

lesions and also oedema are features in the colon of these disorders (*Grant* 1961, *Van Vleet et al.* 1970).

Gastric lesions, which often accompany swine dysentery (*Lussier*), were not conspicuous in the great majority of pigs in this investigation, neither were lesions observed in the colon descendens. The 2 pigs with pseudomembranes in this caudal part of the colon had, however, a very pronounced inflammation in the spiral colon.

The histological examination revealed necrotic mucosal tissue within the pseudomembranes. Fibrinoid thrombi were readily demonstrated in this necrotic tissue, which was almost exclusively observed in pigs fed a vitamin E and/or selenium deficient diet. Similar necrotic and vascular lesions have also been described previously in swine dysentery (*Lussier, Nordstoga et al.* 1968, *Hughes et al.*). Vascular thrombosis and tissue necrosis are further essential features in the Shwartzman reaction. It has been shown that the intestinal mucosa respond to the Sanarelli-Shwartzman phenomenon (*Sanarelli* 1924, *Goldgraber & Kirsner* 1959, *Nordstoga & Fjølstad* 1970) and that pigs fed relatively high amounts of unsaturated fat are predisposed for the experimental generalized Shwartzman reaction (GSR) (*Teige et al.* 1973). It was therefore not surprising that some pigs in the present experiments developed lesions in the colonic mucosa compatible with the Shwartzman reaction. In intact tissue underlying the pseudomembranes, only single occlusive thrombi appeared although conglutinated erythrocytes and a fibrinoid material were frequently present in small vessels. The fibrinoid thrombi observed in the necrotic tissue showed a hyaline appearance and had otherwise almost the same staining characteristic as erythrocytes in H & E stained sections. These observations may be seen in connection with a study by *Nordstoga* (1974) who used porcine salmonellosis as an experimental model of GSR. This author demonstrated that the hyaline and fibrinoid deposits in glomerular capillaries and minor skin vessels were derived predominantly from disintegrated erythrocytes. Severe stasis with excessive vasodilation was found to precede the erythrocytic damage. These findings correspond closely to the phenomenon known as true stasis, as described by *Kreyberg* (1963).

Lesions associated with vitamin E and selenium deficiency were present in several of the experimental animals. The lesions

of hepatosis dietetica in pig No. 6 had a distribution equivalent to that seen in pigs dying from this disease. Other pathological lesions attributable to vitamin E and selenium deficiency were probably of considerably less importance in the animals as they had a limited distribution, and did not cause any clinical signs (*Teige et al.* 1977, 1978). The surgical procedure performed in 4 of the pigs in each of Exps. I and II caused peritoneal fibrotic adhesions which were most pronounced in the pigs given a vitamin E and selenium deficient diet. The number of animals is too small for final conclusions, as to a possible influence of these factors on the development of the post-operative changes. It should, however, be noted that vitamin E has been tested as an anti-inflammatory agent and found to be effective (*Stuyvesant & Jolley* 1967).

Hughes et al. (1976) proposed the following sequential pattern of lesion development in swine dysentery; hyperaemia of superficial venules, excessive production of mucous, inflammatory oedema, necrosis of the epithelium with erosions, exposure of vessels causing haemorrhages and invasion of spirochaetes and opportunist organisms which potentiate the tissue necrosis and produce the more advanced fibrino-necrotic membrane seen in the later stage of the disease. Also *Harris & Glock* (1972) suggested that almost the same sequence of events occurs in swine dysentery. *Nordstoga et al.* and *Espinasse* (1973) seem, however, to be the only authors calling attention to a possible pathogenetic role of the thrombotic vascular lesions.

As a conclusion it may be emphasized that the observations in the present experiments parallel those described by *Hughes et al.* and *Harris & Glock,* although it is suggested in this work that the formation of erythrocytic "thrombi" and other phenomena associated with stasis, such as disturbances in the permeability of minute vessels, may be factors contributing to the development of the necrotic lesions. The pathogenetic mechanism will be further discussed in a subsequent report, in which the electron-microscopic changes are described (*Teige & Nordstoga,* in preparation). Another implication from this common presentation of the patho-morphological manifestations in the pigs of the 3 experiments developing swine dysentery may be that it supports the conclusions submitted in the 2 previous reports regarding the enhancing effect of a combined supplement of vitamin E and selenium on resistance to this disorder.

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SAMMENDRAG

Diettens innflytelse ved eksperimentell svinedysenteri.
3. Patologiske forandringer.

Tre forsøk er blitt utført for å belyse en mulig virkning av innholdet av vitamin E og selen i fôret på eksperimentell svinedysenteri. Hos de fleste grisene som fikk en vitamin E- og selen-fattig diett såes tykke og diffuse pseudomembraner i kolonspiralen. Denne hadde vanligvis også et dilatert utseende og tydelige ødemer i krøset. Den histologiske undersøkelse viste fibrinmembraner festet til defekter på slimhinneoverflaten. Det var også pseudomembraner som inneholdt nekrotisk vev. Fibrinoide tromber var lette å se i små kar i de sistnevnte lesjonene. Griser med bare vitamin E-tilskudd hadde kolonforandringer svært like forandringene som ble sett hos mangelgrisene, mens halvparten av dyra med selen-tilskudd utviklet ingen eller moderate betennelsesaktige forandringer, den andre halvparten hadde imidlertid tydelige pseudomembraner i kolon. Ti av 26 griser med tilskudd av både vitamin E og selen var ikke angrepet av svinedysenteri. Hos de øvrige grisene dominerte katarrhalske betennelsesforandringer i kolonslimhinnen. Hos endel av disse dyrene så man pseudomembraner, men de var vanligvis små og de hadde en begrenset utbredelse. Tromboseringene og vevsnekrosene som ble påvist i lesjonene i kolon er funnet å være i samsvar med en Shwartzman reaksjon. „Erythrocytt-tromber" og andre forhold satt i forbindelse med stase er videre ment å være av patogenetisk betydning i denne forbindelse. Denne rapporten illustrerer også at et kombinert tilskudd av vitamin E og selen øker motstandskraften overfor svinedysenteri.

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