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# CHRONIC ENTERITIS IN INDIANA FEEDER PIGS

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## SUMMARY

An unrecognised chronic enteritis syndrome which is not associated with any specific etiological agent, in Indiana feeder pigs is described. The subgross and microscopical lessions in the small intestine in relation to malabsorption syndrome in human are discussed.

## RANGKUMAN

Kejadian sindrome enteritis kronika pada anak-anak babi di negara bagian Indiana, Amerika Serikat, kelihatannya semakin meningkat setiap tahun. Penyakit ini belum diketahui agen penyebabnya dan tidak memberikan respon yang memuaskan terhadap segala macam pengobatan dengan obat kemoterapetika. Kelihatannya penyakit ini berhubungan dengan kelainan penurunan efisiensi terhadap konsumsi makanan, kelambanan penambahan berat badan dan disertai gejala mencret yang hilang timbul.

Penyakit ini hanya dapat dikenal berdasarkan pada anamnese dari peternakan yang bersangkutan dan pemeriksaan histologik dari usus halusnya.

Gejala yang pertama-tama timbul terjadi beberapa hari sesudah anak-anak babi disapih dan diberi makanan tambahan konsentrat. Derajat kemencretannya adalah sedang dan angka kematiannya sangat rendah. Di antara yang mencret, setelah 4 - 6 hari ada yang menjadi sembuh, sedangkan ada beberapa di antara sisanya tetap terus mencret dan akhirnya berubah menjadi apa yang disebut sindroma enteritis khronika.

Anak-anak babi yang sakit tidak menunjukkan gejala demam dan makan lebih banyak daripada yang sehat. Untuk penambahan berat badan 1 kg, pada babi sehat hanya dibutuhkan 5,38 kg makanan, sedangkan babi sakit untuk penambahan berat badan yang sama diperlukan makan 11,18 kg makanan. Babi sehat mencapai berat badan kira-kira 80 kg dalam waktu 5 - 6 bulan saja, sedangkan yang sakit membutuhkan 10 - 12 bulan.

Pada nekropsi, secara makroskopik tidak ditemukan kelainan. Secara subgross dengan menggunakan disecting microscope, pada jonjot vilus usus halus ditemukan kelainan atrofi dan perubahan bentuk vilus karena terjadi fusi dari beberapa vili menjadi satu seperti "daun", pada (club), membelit (convoluted) dan seperti bukit (ridge). Perubahan ini diteguh-

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kan dengan pemeriksaan mikroskopik. Kelainan ini mirip sekali dengan penyakit sindroma malabsorption pada manusia.

A chronic enteritis syndrome has been observed with increasing frequency in Indiana feeder pigs. This disease does not resemble any of the recognized enteric diseases of pigs, has no specific etiologic agent associated with it, does not respons to any currently available theraputic agents, and appears to be primarily a chronic enteritis with associated poor feed effeciency, intermittant low grade diarrhea, and very slow weight gains. The pattern of the disease is such that it can readily be recognized on the basis of herd history and histology examination of the small intestine. The report is based on experience in, and the records of, the Indiana Animal Disease Diagnostic Laboratory, visits to many Indiana veterinarians and farmers, and experimentation with 10 typically diseased pigs.

**Characteristics of the Disease.**

The disease has been recognized from all parts of Indiana. It is generally a problem in large intensified swine operations, but is seen occasionally in small operations having only a few sows. In almost all cases the pigs are farrowed in a central farrowing house. They thrive in the farrowing house and seldom develop any disease problems until moved to a weaning barn or shed at 4 to 6 weeks of age. Within a few days after being moved to weaning facilities, most of the pigs develop diarrhea shortly before or at the time the sows are removed. The diarrhea is moderate and death losses are very low. Response to treatment is variable, but usually the diarrhea regresses within 4 to 6 days. It is at this point that the chronic enteritis syndrome may first be recognized.

Following the initial diarrheal disease, a variable number of pigs will become unthrifty and continue to have a low grade intermittant diarrhea. The incidence in herds brought to our attention has been 5 per cent or greater with most herds having 10 to 50 per cent of the pigs affected. On large herd had an incidence of 100 per cent. Both the number of herds involved and the incidence of the disease seem to be increasing.

The affected pigs are afebrile and consume large amounts of feed. They grow very slowly and most require 10 to 12 months to reach market weight. Most of the unaffected pigs grow normally and reach market weight in 5 to 6 months. Within 3 to 4 weeks after the initial diarrheal outbreak the pigs can readily be separated into two groups on the basis of their size, weight and general appearance.

Affected pigs have been treated with dietary supplementation, antimicrobials in the feed and water, injectable vitamins and antibiotics and a combination of all of these. At the present time, no theraputic approach has been found to alter the course of the disease. The influence of treatment during the initial stages cannot be evaluated from the records available.

When presented for necropsy examination, the affected pigs appear very small for their age. Their hair coat is slightly coarse and rough. If presented at a time when diarrhea is present, they are slightly dehydrated. However, separated from the herd they would not be easily recognized as diseased animals unless an adequate history were available.

A moderate amount of parasitism, mycoplasmal pneumonie, and atrophic rhinitis may be present in many affected animals, but these findings are inconsistent and in many typically affected animals no gross lesions can be found. There are seldom any gross lesions of the

gastrointestional tract except for a slightly more fluid chyme and colon contents. The subgross and microscopic appearance of the small intestinal mucosa in described later.

Cultures of the small and large intestinal usually result in isolation of **E. coli** only. Occasionally **Proteus, Aerobacter** or **Pseudomonas** will also be isolated. Salmonella has been isolated from a typically affected animal only once.

**Case Report**

A swine farm at Greensburg, Indiana, experienced a typical chronic enteritis problem in 1966 and 1967. Pigs on this farm had a mild problem of chronic enteritis for 3 - 4 years, but it did not become economically important until the fall of 1966. During the fall farrowing in 1966 and in two farrowing in 1967, the incidence and severity of the disease increased through 3 separate farrowings. Management and sanitation were adequate and above standard for most Indiana farms, Veterinary service was available and used. All adult pigs remained healthy through out the period of time covered by this report.

Sows on this farm were farrowed in a central farrowing facility. When the pigs were 3 weeks old, they and the sows were moved to weaning facilities and the sows removed after several days. The pigs were vaccinated for both hog cholera and erysipelas at the time they were moved to the weaning facility. The feed was mixed at a local mill and contained approximately 12 per cent protein.

Approximately 150 pigs were farrowed during the last two week of April, 1967. The owner reported that these pigs did exceptionally well until about 1 week after being moved to the weaning facilities. At that time they developed a disease characterized by diarrhea and dehydration. The initial morbidity was over 90 per cent and the mortality in the first week of the disease was less than 2 per cent. Following antibiotic treatment about 65 per cent of the pigs recovered within 1 week. Approximately 35 per cent of the herd continued to have an intermittant scours problem and slow growth even through they were treated extensively with a number of antibacterials in both feed and water as well as with injectable vitamins and antibacterials.

When examined at 13 - 15 weeks of age, approximately one third of the herd were small, had slightly rough hair coats and many had soft stools. The remainder of the herd appeared to be healthly feeder pigs of normal size and weight for their age. The diseased pigs had been on various types of medication continuously for a period of 9 weeks.

**Experimental materials and methods**

Pigs — Ten pigs were selected at random from the diseased group of pigs referred to in the case report above. Five pigs were selected at random from the group of healthy pigs. These pigs were taken to the Purdue University research facilities and placed in isolation rooms. Feed and water were given **ad. lib.** Feed consumption and body weights were determined on a weekly basis for three weeks.

Glucose tolerance test — The pigs were fasted for 18 hours and then given orally 2.2. Gm. glucose, in a 33 per cent solution, per Kg. body weight. Blood samples were taken from the anterior vena cava immediately prior to and at hourly intervals for 3 hours following the administration of the glucose solution. Glucose levels in the serum were determined by the glucose oxidase method described by Guidotti **et al.** (1961) modified by using a 15 minute incubation period at room temperature. All samples were ran with standards and Lab-trol* as control.

* General Diagnostic Division, Warner - Chilcott Laboratories, Morris Plain New Jersey.

Fat tolerance test — Soybean oil was given orally at a level of 2.2 ml per Kg. body weight after an 18 hour fast. Blood samples were taken from the anterior vena cava prior to and at hourly intervals for 5 hours following the administration of the soybean oil. Serum turbidity was determined as optical density at a wave length of 640 mu. using distilled water as a blank.

Transit time of food residues and fecal fat determinations — After a 5 hour fast the pigs were exposed for 2½ hours to a feed containing 5 per cent carbon granules which had an average diameter of 1 mm. At the end of the 2½ hour period, the unconsumed feed was removed and a standard diet fed. Feces were collected at 8, 10, 12 and 14 hours. Beginning at 16 hours feces were collected at half-hour intervals until carbon granules appeared in the feces. Thereafter fecal samples were collected at 4 hour intervals for a 6 day period. A morning and afternoon fecal sample from each pig were mixed and total fecal fatty acid content determined by the method of Van De Kramer, et al. (1949).

Necropsy and tissue examination — Following completion of all the above studies, the pigs were electrocuted and necropsied. All pigs were fasted overnight and then given 2.2 ml. soybean oil per Kg. body weight 2 hours before they were killed. Blood samples were collected at necropsy for hematology examination. All tissues except the central nervous system were examined. The pH of the stomach contents, duodenum, jejunum and ileum was determined with a pH paper strip *. Sections of the three areas of the stomach, 3 equidistance points along the small intestine, ileoceco-valve, apex of the cecum, apex of the spiral colon, mesenteric lymph nodes, spleen, liver, lung, kidneys and adrenal glands were fixed in 10 per cent neutral buffered formalin. Tissue from the three equidistant points along the small intestine was frozen, sectioned and stained with oil-red-O. Formalin fixed tissues was sectioned and stained with hematoxylin and eosin. Selected tissues were stained with aqueous periodic acid schift (PAS).

Fluids and mucosa from the jejunum were cultured in selenite mediun and subcultured to brilliant green agar. The spleen and liver were cultured aerobically on conventional blood agar and brilliant green agar.

Transmission — Jejunum from one typically diseased pig was made into a 20 per cent suspension in saline. A portion of the suspension was filtered through an HA Millipore ** filter. Ten micrograms Streptomycin and 100 unit penicillin per ml were added to half of the filtrate and incubated for 30 minutes at room temperature. Six three week old specific pathogen free pigs were placed in individual isolation units and fed cows milk. Two pigs were given 10 ml each of the per cent intestinal suspension orally. Two pigs were given 2.5 ml each of non-antibiotic treated filtrate and two were given 2.5 ml each of the antibiotic treated filtrate orally. These pigs were then observed for a period of 6 weeks.

**Experimental Results**

At the time the pigs were purchased and placed in isolation rooms, the smallest diseased pig weighed 32 pounds and the largest control pig weighted 116 pounds. The general appearance of these pigs is shown in figure 1.

During the three weeks the pigs were held in isolation units, the control pigs averaged 1.65 lbs. daily gain on 2.69 lbs. feed per pound gain. The diseased pigs gained 0.6 lbs. daily and required 5.59 lbs. feed per pound of gain. The mean and range of weight gains in the two groups are depicted in figure 2.

* Micro Essential Laboratories, Brooklyn, New York.
** Millipore Filter Corporation, Bedford, Mass.

The serum glucose levels are given in table 1. More variation was found in the post-prandial serum glucose levels of diseased pigs than in normals. The mean level of serum glucose in diseased pigs did not increase as rapidly or reach as high a level in the diseased pigs as in the controls.

Postcibum serum turbidity reached its maximum in 2 hours in diseased pigs and in 4 hours in controls. There appeared to be little total difference between the two groups, but the earlier rise and slower decrease in diseased pigs suggested some difference in the metabolism of orally administered soybean oil. In the small intestinal epithelial cells of the control pigs the fat appeared in very find droplets having a dust-like appearance in the apical cytoplasm of all cells along the villus. In the diseased pigs the fat in the epithelial cells had the same distribution, but was present in large irregular droplets or as single globules. The mean total fatty acid content in feces of pigs fed a diet containing 3.04 per cent fatty acids was 2.52 per cent in the control and 2.36 per cent in the diseased group.

The mean time taken for carbon granules to appear in the feces of the control pigs was 18.9 hours while it was 22.7 hours in the diseased pigs. The mean time of terminal excretion of the carbon granules was 88.0 hours in the control pigs and 95.8 hours in the diseased animals.

The mean hemoglobin level of the control pigs was 12.4 Gm. per 100 ml in the control pigs and 11.6 Gm. in the diseased group. The mean white blood cell count in the control pigs was 16,000 per cu. mm. with 34.8 per cent neutrophils and 61.6 per cent lymphocytes. For the diseased pigs the mean white blood cell count was 25,600 per cu. mm. with 50.6 per cent neutrophils and 44.5 per cent lymphosytes.

No significant gross lesions were found in either the control or diseased pigs. All had adult ascarids in the small intestine, but the diseased group had a few more than did the controls. The maximum number of ascarids in any pig was 50. Six of the diseased pigs had greyish pale areas of atelestasis in the apical lobes. No turbinate atrophy was found in any of the pigs. There was no significant difference in the pH of the gastrointestinal contets.

Subgross examination of the small intestine revealed the mucosa of the control pigs to consist of relatively long villi which were free of adhesions. A few leaf-form villi were observed in most of the control pigs. In the diseased pigs the mucosa contained few individual villi. Most were adhered together to give the leaf-form appearance. Ridges and convolutions of the mucosa were common and in those areas free villi were not present. All villi, leaf-forms, ridges and convolutions appeared shorter than did the free villi in the control pigs.

Histologic examination of the small intestine revealed considerable variation in the length of villi and depth of crypts in the three sections examined. The longest villi were found in the jejunum of the control pigs and the appearance of this tissue is shown in figure 3. The lesions in the disease pigs were most marked in the duodenum and jejunum. The short villi (figure 4) and villous fusin (figure 5) were present in all of the diseased pigs. The relative villous-crypt rations are given in table 3. A model to illustrate a combination of the subgross and histologic appearance of the convoluted mucosa was prepared from serial sections and is shown in figure 6.

The surface epithelial cells of the small intestine varied from tall columnar to cuboidal in the diseased pigs while in the control pigs the cells were univormly columnar. Extrusion zones were more apparent in the diseased pigs and mitotic activity in the crypts was greater than in the controls. There Was extensive mononuclear infiltration, primarily of lymphocytic type cells, in the lamina propria of the diseased intestines. A most striking feature was the extensive migration of mononuclear cells through the surface epithelium of the diseased

intestines. Globule leukocytes and eosinophils were found uniformly in both groups. In the disease pigs clumps of inflammatory cells were found occasionally in the crypts lumens in the ileum and ileocecal valve regions, but there was no associated increase in the inflammatory response of the lamina propria.

The mesenteric lymph nodes had a generalized lymphoid hyperplasia. The sinusoids were extensively infiltrated with eosinophils and globule leukocytes were randomly distributed in both lymphoid and sinusoidal areas. The lung lesion were typical of those associated with mycoplasmal pneumonia. No other histologic lesions of significance were observed.

**E. coli** and **Proteus** were isolated from the small intestinal contents of all pigs. Cultures of liver and spleen uniformly failed to produce bacterial growth.

None of the 6 pigs receiving intestinal material prepared from a diseased pig developed any signs of illness during the 6-week observation period and the growth rate of the pigs was normal.

**Discussion**

A specific disease syndrome of young weanling and feeder pigs has been observed for several years in Indiana. This syndrome has identifiable characteristics allowing it to be separated from the commonly recognized swine disease. While no etiologic agent has been identified, the development of the disease as a herd problem, the clinical manifestation of the disease in the individual animal and the morphologic features of the small intestine of the diseased animal suggest a common cause and pathogenesis.

The economic importance of the disease can be very great to the individual swine producer. During the course of these investigations several farmers were contacted who had sold all of their pigs because of the severe economic loss associated with persistence of the disease on their farms. Other reported that the only way they could stay in the swine business was to take the diseased pigs to the local sale barn at the first sign of chronic enteritis. Prolonged uneffective medication and extremely poor feed conversion are the major costs to the producer.

A wide variety of terms have been attached to this syndrome by different veterinarians and diagnosticans. These include chronic enteritis, non-specific enteritis, coliform enteritis, sprue, sprue-like syndrome, malabsorption syndrome, and wasting disease. Many times the final disposition of these cases has been simply - no diagnosis.

Malabsorption syndromes are common in human medicine and may be caused by a wide variety of agents and factors. To be meaningful the term must be modified by both qualitative and quantitative description. Total absorption from the digestive tract cannot be determined by simply subtracting the volume of the fecal material from the volume of food and fluid ingested because digestion and assimilation of ingesta involves exsorption as well as absorption. Total volume of exsorbed material in the monogastric animal is several times the volume of the ingesta so that the total absorptive load placed on the intestinal tract is quite large. Because of the difficulties in measuring total absorption, studies on malabsorption usually involve a single dietary component and the rate of its absorption determined by measuring its concentration in the circulating fluids. This method by-passes the problem of exsorption as well as cell loss from the mucosa and bacterial activity in the digestive tract.

The degree and rate of glucose absorption in these pigs indicated a moderate carbohydrate malabsorption in the diseased pigs. However, the use of glucose to measure carbohydrate absorption has certain shortcomings since the rate of absorption is influenced by dosage and by stomach emptying time (Cuningham, 1959; Dollar **et al,** 1957; Hanawalt &

Sampson, 1947). Furthermore, glucose is not a normal component of the diet and its use fails to measure the enzyme activity necessary from digestion of disaccharides and polysaccharides. Thus a carbohydrate malabsorption syndrome cannot always be detected or its severity measured by use of the oral glucose tolerance test.

The results of the fat absorption tests indicated that an abnormal pattern of lipid absorption and utilization existed in the diseases pigs. While there was little difference in total fat absorption as determined by serum turbidity and fecal fat content, there was an apparent difference between the fat metabolism of the diseased and the control pigs. Rapid stomach emptying might have been responsible for the earlier rise in serum lipids of diseased pigs, but this was not apparent in the glucose tolerance tests. Abnormal assimilation of the lipid in the mucosal epithelial cells in suggested by the observarions made on frozen sections stained with oil-red-O. The marked difference in the size and morphology of the lipid droplets in the epithelial cells of the small intestine suggests that there might be a deficiancy of lipoprotein synthesis which is necessary for the formation of the chylomicron (Isselbacher, 1967). The lack of steatorrhea in the diseased pigs suggests that resterification of the lipid components occured since, in nontropical sprue where esterification is reduced the disease is characterized by steatorrhea (Brice **et al.**, 1965). Abnormal metabolism of absorbed fat is suggested by the prolonged time required to clear lipids from the blood of diseased pigs as compared to a relative rapid clearance from the blood of the controls.

The only significant lesions were those in the small intestine. The normal mucosal pattern appears to vary with age. Hooper and Haelterman (1966) reported that the normal villuscrypt ratio in baby pigs was 7 : 1. In older pigs the same finger-like mucosal pattern is maintained (Sloss, 1954; Titkemeyer & Calhoun, 1955) but the villi of older pigs were reported to be somewhat shorter (Titkemeyer & Calhoun, 1955). Other than the report of a few leaf-form villi in the small intestines of pigs recovering from transmissible gastroenteritis (TGE) (Teir & Rytomaa, 1966), leaf-form villi have not been reported in pigs. Subgross examination was necessary to evaluate the nature and extent of these abnormal villous forms since histological examination of stained sections can be very misleading (McCarthy **et al.**, 1964).

The mucosal lesions found in the diseased pigs is similar to that reported in dogs with a malabsorption syndrome (Kaneko **et al.**, 1965) and in humans with both nontropical sprue (Padykula **et al.**, 1961) and tropical sprue (Swanson & Thomassen, 1965). The range of agents and conditions that have been associated with mucosal damage and villous atrophy are extremely numerous and these have been reviewed by Collins (1965). In view of the excellent regenerative powers of the intestinal mucosa (McMinn & Mitchel, 1954; Stephens **et al.**, 1954) it seems unlikely that this intestinal lesion could be explained on the basis of residual damage remaining from the initial diarrheal disease experienced by most of the pigs shortly affter weaning. While the intestine appears to serve as the "graveyard" for the white blood cells of the body (Teir & Rytomaa, 1966), the presence of extremely large numbers of these in the epithelial border of the intestinal mucosa of the diseased pigs would suggest that an active inflammatory process was present. While these generally appeared to be lymphocytic cells. Teir and Rytomaa (1966), reported that neutrophile in the epithelial border also resemble lymphocytes. The absolute neutrophilia and relative lymphoneia observed in the diseased pigs as well as the presence of inflammatory cells of the ileal crypts also sugges that the intestinal lesions should be considered to represent an active inflammatory process. The absence of any excessive mucus accumulation or goblet cell hyperplasia precludes calling this a catarrhal enteritis.

The bacterial isolation procedures carried out on these experimental pigs as well as on diagnostic cases have failed to incriminate a bacterial agent in this disease. With the restricted approach to isolation and identification of the bacterial flora which has been used there is certainly the possibility that more extensive bacteriologic examination would prove fruitful. However, the orgnism isolated to date were present in control as well as diseased animals and are considered to be normal flora in the digestive tract of the pig.

The limited transmission attempts reported here failed to produce detectable disease in supposedly suspectable pigs. However, in typically affected herds most of the pigs usually remain healthy which would suggest that if an infections agent is responsible that its infectivity is less than that expected with most swine pathogens. The fact that the disease does appear to begin in a significant number of pigs at the same time suggests that some commun environmental, nutritional, or infectious agent is responsible. The complete lack of response to available theraputic agents reduces the possibility that a bacterial organism is responsible. Nutritional factors may play a part as in the gluten induced enteropathy of non-tropical sprue. We have not attempted to isolate viral agents from these diseased pigs, but at the present time viral agents have not been reported to produce similar lesions with this degree of chronicity in any species.

Table 1. Fasting and Postprandial Levels of Serum Glucose * in Pigs Given 1 Gm. Glucose per pound body weight after an 18 hour fast

| Time | 5 Control Pigs | | 10 Diseased Pigs | |
|---|---|---|---|---|
| | Mean | Range | Mean | Range |
| Fasting | 84.5 | 75.0— 98.5 | 91.6 | 74.5—109.0 |
| 1 hour | 169.9 | 158.6—182.0 | 116.6 | 76.5—214.0 |
| 2 hours | 136.3 | 112.0—165.0 | 127.1 | 106.0—156.5 |
| 3 hours | 99.3 | 76.0—118.0 | 105.3 | 90.5—127.0 |

* mg. glucose per 100 ml serum.

Table 2. Optical density * of fasting and postcibun serum of control and diseased pigs given 1 ml soybean oil per pound body weight after an 18 hour fast

| Time | 5 Control Pigs | | 10 Diseased Pigs | |
|---|---|---|---|---|
| | Mean | Range | Mean | Range |
| Fasting | 0.076 | 0.070—0.080 | 0.074 | 0.054—0.114 |
| 1 hour | 0.080 | 0.076—0.081 | 0.079 | 0.050—0.127 |
| 2 hours | 0.094 | 0.082—0.120 | 0.153 | 0.006—0.369 |
| 3 hours | 0.139 | 0.096—0.181 | 0.148 | 0.070—0.278 |
| 4 hours | 0.145 | 0.106—0.182 | 0.136 | 0.065—0.240 |
| 5 hours | 0.086 | 0.066—0.100 | 0.107 | 0.054—0.151 |

* Optical density at 640 mu wave length against a distilled water blank.

Table 3. Measurements of villi and crypts * in the small intestines of control and diseased pigs

| Region of small intestine | Control Pigs | | Diseased Pigs | |
|---|---|---|---|---|
| | Villus/Crypt * | V : C Ratio | Villus/Crypt | V : C Ratio |
| Duedenom | 590/457 | 1.3 : 1 | 387/421 | 0.9 : 1 |
| Jejunum | 647/233 | 2.8 : 1 | 437/306 | 1.4 : 1 |
| Ileum ** | 464/240 | 1.9 : 1 | 356/233 | 1.5 : 1 |

* Mean villous length and crypt depth in microns.

** Villi and crypts were those not over lymphoid tissue.

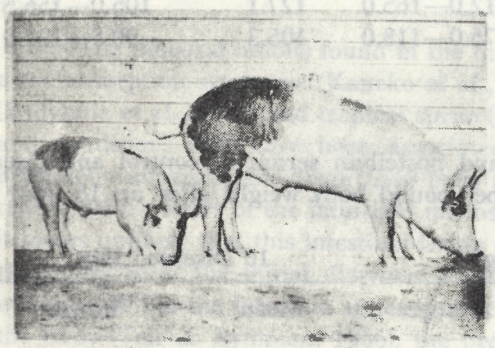

Fig. 1. Control and diseased pigs farrowed at the same time and raised together. These pigs are approximately 14 weeks old.

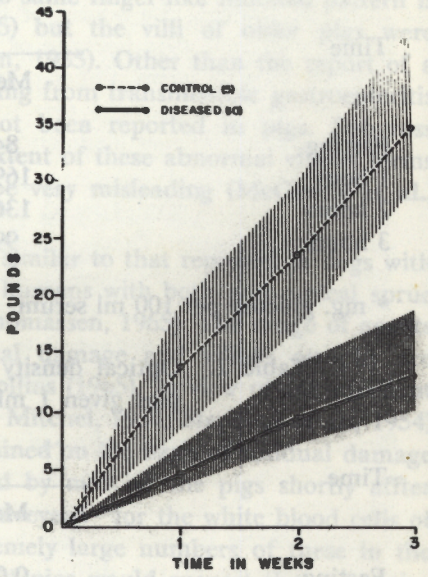


Fig. 2. Mean and range of weight gain in control and diseased pigs over a three period.

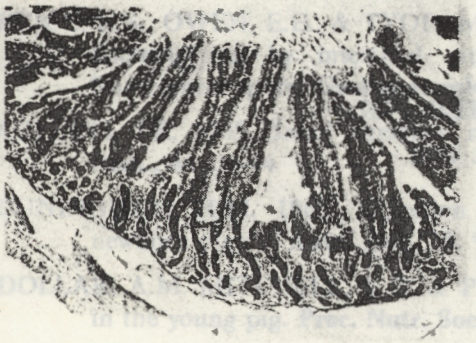

Fig. 3. Normal jejunal mucosa of a control pig. The ling villi and shallow crypts give a villouscrypt ratio 3:1. 80 x magnification.

Fig. 4. Jenunal mucosa of a diseased pig. Extremely short villi and deep crypts give a villouscrypt ratio of less than 1:1. 200 x magnification. H & E stain.

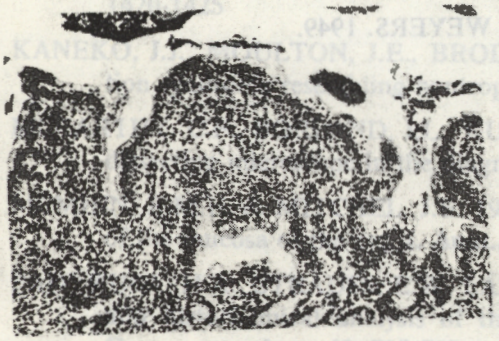

Fig. 5. Villous fusion in the jejunum of a diseased pig. This type of mucosa appears convoluted when examined with a dissecting microscope. Villouscrypt ratio is approximately 1 : 3. 200 x magnification. H & E stain.

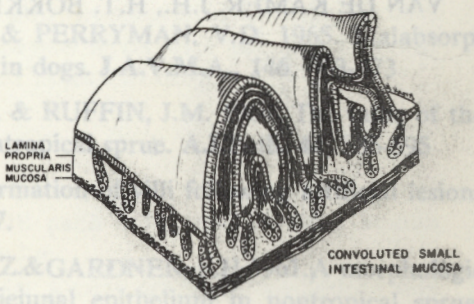


Fig. 6. Three dimensional appearance of convoluted small intestional mucosa. Drawing based on clay model reconstruction of serial sections from jejunal mucosa of a diseased pig.

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