Swine Dysentery: A Review

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Swine dysentery was first described by Whiting, Doyle, and Spray in Indiana in 1921.[32] It is a contagious infectious disease characterized by a mucohemorrhagic diarrhea. The disease has been reported from most parts of the world. Swine dysentery is also referred to as vibrionic dysentery, bloody scours, bloody dysentery, and mucohemorrhagic diarrhea.

The true incidence and economic significance of this disease is difficult to determine. Swine dysentery was the second most common swine disease diagnosed at the Iowa Veterinary Diagnostic Laboratory with 121 case submissions in 1969. In 1968, 271 herds were quarantined in Iowa involving 37,000 head of swine. In 1969, 159 herds were quarantined involving 22,000 head. However, it has been estimated that less than 50 percent of the cases ocurring in Iowa are reported for quarantine.[12] It is generally accepted that the disease is diagnosed and treated by the producer in many instances to avoid quarantine regulations.

Swine dysentery has a pronounced effect on rate of gain and feed conversion of affected pigs (Table 1).[11] Since the disease results in mortality in herds which do not respond to treatment and ocurs with a high morbidity in most cases, it definitely causes financial loses due to death of pigs, decrease in rate of gain, and expenses for chemotherapy.

The etiology of swine dysentery has not been adequately investigated nor definitely determined. Swine dysentery is considered to be caused by an infectious transmissible agent. The mucosa of the colon and cecum of acutely affected swine apparently contains the infectious organism(s). If this mucosa is scraped off, homogenized, mixed with buffered saline, and administered to susceptible pigs by oral inoculation (gut inoculum), clinical signs of the disease will appear in 5 to 12 days in 80 to 100 percent of the experimentally inoculated pigs. These signs appear sooner and in a higher percentage of animals if the pigs are starved for 12 to 18 hours prior to inoculation.

Some of the characteristics of the agent or agents responsible for the production of swine dysentery in pigs have been determined. The disease has been reproduced by administering filtrates of gut inoculum which passed a 0.8 micron pore diameter filter. This filtrate contained *Vibrio coli*, spirochete organisms, and many other bacteria. However, the disease could not be produced by inoculating pigs with filtrates which passed a 0.45 micron filter. This filtrate contained *V. coli*, *Escherichia coli* and a *Streptococus spp.*[31] Attempts to reproduce swine dysentery with filtrates of gut inoculum which passed a Berkefeld V filter (bacteria free) have been unsuccessful.[5]

It has been determined that the agent(s) is susceptible to heating at 60° C for 30 minutes but will withstand heating at 50° C for the same time interval. The agent(s) is resistant to penicillin but sensitive to streptomycin, neomycin sulphate, and tylosin tartrate as determined by treatment of gut inoculum with these compounds *in*

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vitro before administration to susceptible pigs. The agent(s) will not urvive storage at —20° C for 3 months.[28]

Although *V. coli* has been associated with swine dysentery for several years both from an etiologic and pathologic standpoint, its role in the disease process has not been substantiated. It has been reported that the feeding of pure cultures of *Vibrio spp.*, grown either in agar or broth and mixed with swine gastric mucin produced diarrhea in 50 out of 60 young pigs. The clinical signs and macroscopic lesions observed in the experimentally reproduced disease were indistinguishable from those seen in the naturally occurring disease.[14]

Other workers have reported the production of swine dysentery by experimental exposure of pigs to pure culture of *V. coli.* However, the experimental disease produced in these cases was often only a transient diarrhea and the number of swine affected was usually less than 50 percent of those exposed.[5, 20, 25, 29]

A recent report indicated an inability to reproduce the disease by exposing pigs to pure cultures of *V. coli.* grown in broth and mixed with swine gastric mucin. When a filtrate (0.45 micron) of gut inoculum was inoculated directly into embryonating chicken egg and incubated for 18 hours, death of the embryo resulted. *Vibrio coli* was present and other bacterial agents could not be isolated. Eight of 12 pigs inoculated with the egg fluids developed signs of swine dysentery. It was suggested that the failure of reproduction of the disease by some workers with pure cultures of *V. coli* resulted from the loss of pathogenicity of stock cultures of the organism.[31]

Several workers have reported on the failure of reproduction of swine dysentery with pure cultures of *V. coli.*[1, 2, 4, 7, 9, 22, 28] One study was very extensive in that it involved the use of 23 strains of *V. coli* isolated from pigs and experimental inoculation of both gnotobiotic and conventionally reared swine with various combinations of these strains grown in milk media. The organism was readily established in most of the pigs inoculated, but no clinical signs or macroscopic lesions of the disease were produced in either the gnotobiotic or the conventionally reared pigs.[1, 2]

During the past 4 years, research in several countries has revealed that another agent, a spirochete, may be associated with swine dysentery. King and Baeslack (1913) first observed spirochete (Order Spirochaetales) organisms associated with hog cholera.[15] However, since the isolation of hog cholera virus little significance has been attached to the organism when observed in diseases of swine.

In 1968 a worker in the Netherlands precipitated immunoglobulins from the serum of swine chronically affected with swine dysentery. These immunoglobulins were conjugated with fluorescein isothiocynate and applied to smears of fece and digestive tract contents. Specific fluorescence could be found only in affected pigs and it was consistently associated with an organism resembling a spirochete. The conjugate did not react with feces from normal pigs nor did it react with any *Vibrio spp.* organisms. The spirochete organism could not be cultivated *in vitro.*[28]

Another worker, in Spain, has consistently associated spirochete organisms with clinical signs and lesions of swine dysentery by examining feces and colon contents from affected pigs by direct and electron microscopy. The organisms could definitely be differentiated from *Vibrio spp.* and were probably of the genus *Borrelia* on the basis of staining characteristics.[27] The spirochete organisms have been cultivated *in vitro* in commensal culture systems both in England[26, 30] and the United States.[10] These organisms have not been isolated in pure cultures and have not been inoculated into susceptible experimental swine.

Attempts to produce swine dysentery with other agents have been conducted on a limited scale. The disease cannot be reproduced with pure cultures of the following agents: *Streptococcus sp.*,[5] *Escherichia coli, Bacteriodes Sp.*, and *Trichomonad sp.*[28]

Since *Vibrio coli* and spirochete organisms are currently believed to be associated with the lesions of swine dysentery. The

characteristics of these two agents will be discussed.

Vibrio coli. This organism is a motile gram negative rod characterized by 1 polar flagella at each end of the cell. The organism requires a microaerophilic environment for growth at 37 C and growth is stimulated by 5 to 10 percent CO_2. It may be isolated on blood agar and is usually comma shaped initially.[23] Coccoid forms of the organism are seen after several passages in artificial medium.[21] Long spiral forms are often observed when the organism is grown on PPLO agar.[10] Biochemically, *V. coli* does not ferment carbohydrates but does reduce nitrates to nitrites.[23] Other types of *Vibrio spp.* may be isolated from the swine intestinal tract and therefore differentiation from these organisms is necessary (Table 2).

Vibrio coli may be isolated readily from intestines of both normal and swine dysentery affected pigs by a variety of methods: (1) A hot spatula may be used to singe the surface of the mucosa of the infected gut and inoculation of blood agar with the underlying material usually results in their isolation.[14] (2) A suspension of fecal material or gut mucosa prepared in buffered saline and passed through a 0.65 micron filter onto blood agar results in the elimination of many of the other nitestinal bacteria but allows for the passage of *V. coli.*[17] (3) Rectal swabs or fragments of mucosa may be placed in skimmed milk containing 25 IU of bacitracin, 20 IU of polymyxin, and 25 micrograms of novobiocin per ml and incubated at 37° C for 5 to 6 hours. A loopful of the sample is then inoculated onto blood agar for the isolation of the organism.[1]

Order Spijrochaetales. This order of bacteria contains two families, the Spirochaetaceae and the Treponemataceae. Pathogenic organisms are reported only to exist in the latter family. There are three genera within the Treponemataceae; *Borrelia*, *Treponema*, and *Leptospira*. The *Borrelia* and *Treponema* are anaerobic genera have been differentiated on the basis that *Borrelia* are stained readily with aniline dyes.[23] *Borrelia* seen in swine dysentery stain well with dilute carbol fuchsin.[27]

Morphologically, the *Borrelia* and *Treponema* are long spiral organisms and are motile. The organisms are not flagellated and their motility is believed to be due to the presence of axial fibrils located along the longitudinal axis of the protoplasmic cylinder.[19]

Recently, it has been reported that these two genera may be differentiated by electron microscopy. Based on this report, the *Treponema* have a protoplasmic cylinder diameter of 100–250 millimicrons and and either 1 or 2 axial fibrils originating at each end of the protoplasmic cylinder. The *Borrelia* have a protoplasmic cylinder diameter of 200–500 millimicrons and the number of axial fibrils originating near each end ranges from 3 to 20.[19] At the present time, classification of these organisms is quite difficult and may change drastically as more studies are conducted with them.

Both Treponema- and Borrelia-like organisms have been observed in the intestinal contents of pigs affected with swine dysentery. The Treponema have been isolated in pure culture using the anaerobic technique of Hungate[13] (Figures 1 and 2).[10] The *Borrelia* have been cultured under anaerobic conditions on blood agar in the presence of other bacteria from the digestive tract (Figures 3 and 4).[10, 26, 30] There have been no reports of isolation of the *Borrelia* associated with swine dysentery in pure culture. It must be emphasized that while these organisms have been cultured from the digestive tract of pigs with swine dysentery, there is no evidence to
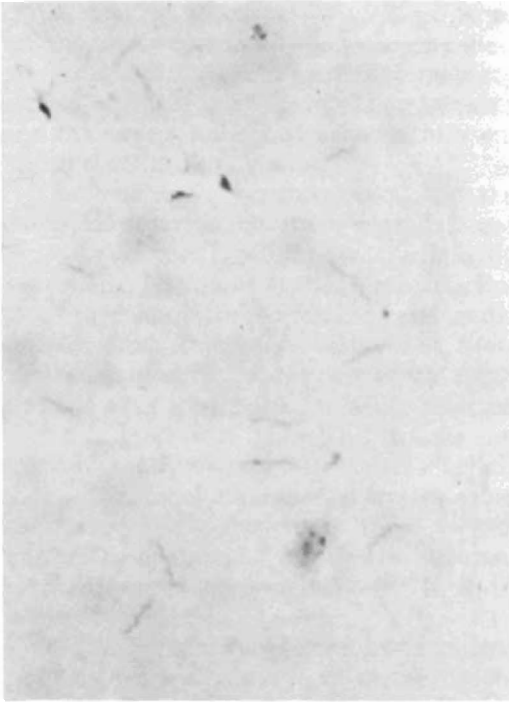
Figure 1. Treponema-like organism isolated from the colon of a pig affected with swine dysentery. The organism was grown in pre-reduced anaerobically sterilized media. Carbol Fuchsin stain; x1400.[10]

Figure 2. Electron micrograph of Treponema-like organism pictured in Figure 1. Axial fibrils (arrow) are in the "2–4–2" type arrangement. Phosphotungstic Aicd stain; x20,000.[10]

prove that they are the etiology of the disease.

Although the course of swine dysentery may vary from peracute to chronic, the various forms are quite similar except in time sequence. The overall appearance of affected animals is one of dehydration and loss of condition with a gaunt contracted abdomen. The perineum is usually stained with fecal material as a result of diarrhea.

Lesions are confined to the digestive tract and typically involve the large intestine. The small intestine is usually quite empty but appears normal upon gross and microscopic examination. Necrotic debris containing inflammatory cells is often observed in the lumen of the terminal ileum but is probably the result of regurgitation from the cecum.[31]

The characteristic lesions of swine dysentery are observed in the cecum, colon, and rectum although they may be confined to the colon. The serosal surface of the large intestine often presents a dry, granular appearance probably as a result of dehydration and submucosal inflammation; however, in acute cases the mesentery and intestinal wall are often edematous.[20] The gut is congested and is frequently dilated and flaccid. The contents of the colon are uniformly soft and contain blood, mucus, and fibrin. The mucosal surface is inflamed and has varying amounts of mucofibrinous exudate adherent to it.

The pathogenesis of swine dysentery has not been described, but the sequential pattern of lesion development is as follows. Early lesions include congestion of vessels near the lumen with edema in the lamina propria. Copious amounts of mucus are secreted from hyperactive cells causing dilatation of crypts and submucosal glands. Exhaustion of mucus-producing cells ocurs and sloughing of epithelial cells lining the lumen soon follows. Increased amounts of fibrin accumulate and mix with mucus to form an adherent pseudomembrane which contains numerous

sloughed epithelial cells, inflammatory cells, erythrocytes, and bacteria. There is an accumulation of variable numbers of inflammatory cells in the lamina propria. Hemorrhage probably results from exposure of capillaries due to erosion of the mucosal surface. Necrosis continues until most of the lumenal surface of the mucosa is involved, but the deeper portions remain relatively unaffected.[8]

The intensity of the reaction is not constant. As a result, the amounts of blood, mucus, and fibrin in the lumen are variable. Occasionally only a portion of the spiral colon may be involved but in most cases the entire colon and at least a portion of the cecum are affected. Lesions in the rectum are less severe and may not progress beyond catarrhal inflammation.[8]

It is common, especially in advanced cases, to observe pale, slightly raised nodules scattered over the serosal surface of the spiral colon. These are greatly dilated submucosal glands which extend nearly to the serosal surface and contain mucus, cellular debris, bacteria, and a few inflammatory cells.[8]

Inflammation and thrombosis of the gastric wall and mild hepatic degeneration may be observed in cases of swine dysentery but these lesions are observed in various other conditions and are non-specific.

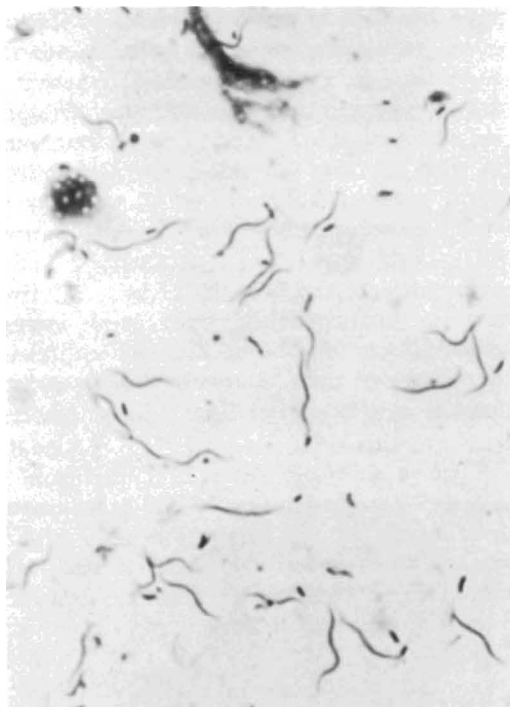Large numbers of bacteria are present in the crypts and near the lumen surface

Figure 3. Borrelia-like organism isolated from the colon of a pig affected with swine dysentery. The organism was grown in mixed culture in blood agar under anaerobic conditions. Carbol Fuchsin stain; x1400.[10]

TABLE II

Differentiation of Common Vibrio species[a] Isolated from Domestic Animals

	V. fetus var. venerealis	V. fetus var. intestinalis	V. bubulus	V. coli
Catalase	+	+	−	+
H₂S TSI	−	−	+	+
Growth in 3.5% salt	−	−	+	−
Growth in 1% Glycine	−	+	+	+
Growth in 1% Bile	+	+	−	+

[a] All four species reduce nitrate and do not ferment carbohydrates; all are microaerophilic.

early in the disease and throughout its course. The organisms in the lumen are of various types but those found in the crypts are more restricted in variety. One type commonly present is represented by short, comma shaped rods which resemble V. coli. The other organisms which are consistently present are longer spiral shaped forms (Figure 5). These are identified as spirochetes on the basis of their morphology as observed by electron microscopy.[3, 8, 26, 27] Their diameter is 0.25 to 0.35 microns and they usually have approximately 12 axial fibrils which suggests classification as Borrelia. These spirochetes have been observed in degenerating epithelial cells (Figure 6)[8]. There is no evidence available to determine whether they produce cellular degeneration by invasion or are merely invading previously damaged cells.

Balantidium coli are frequently present in large numbers in the lumen, mucosal

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crypts and necrotic areas, but there is no inflammatory response accompanying their presence and it is assumed that they are merely opportunists. They feed on bacteria and the high numbers of bacteria may enhance their multiplication.

The methods generally used in the diagnosis of swine dysentery have changed little since the condition was originally described. The bases of diagnosis are clinical observation, gross lesions, and some support from laboratory tests. The most consistent finding is diarrhea with rapid shrinkage of the flank area in affected pigs. A moderate, transient, febrile response usually precedes the onset of diarrhea. The incubation period varies from one to two or occasionally three weeks. The disease spreads rapidly in infected herds. It is most frequently seen in 8–14 week-old pigs.[8, 20]

Soft, unformed feces containing mucus and blood are suggestive of swine dysentery. The blod may be bright red and only

Figure 5. Borrelia-like organisms (arrow) in crypt of colon from a pig affected with swine dysentery. Organisms with a morphology resembling *Vibrio spp.* are also present. Goodpasture stain; x1400.[8]
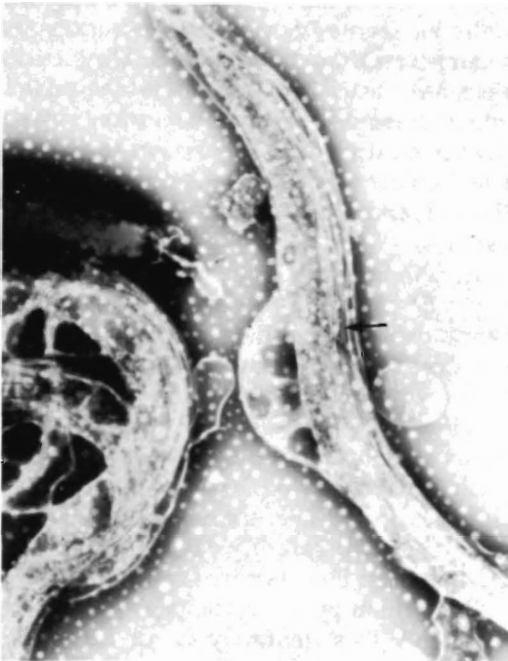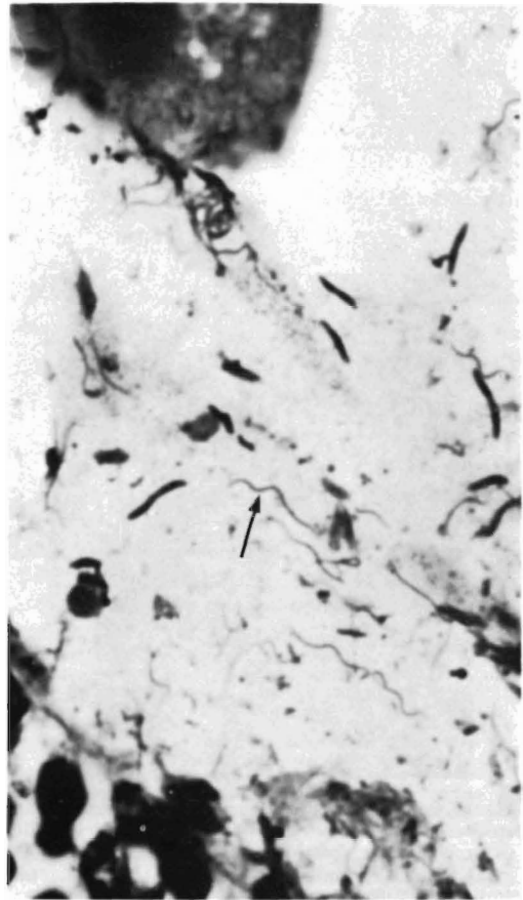
Figure 4. Electron micrograph of Borrelia-like organism pictured in Figure 3. Approximately 12 axial fibrils (arrow) are present. Phosphotungstic Acid stain; x20,000.[10]

partially mixed with mucoid fecal material or may be thoroughly mixed giving the feces a dark red color. Necropsy reveals gross lesions confined to the large intestine as previously described.

Phase contrast or dark field microscopy of tissues collected soon after death may reveal large numbers of motile *Vibrio spp.* and spirochetes, which helps to substantiate the diagnosis. This, however, is only a presumptive test as it is conceivable that a similar flora could exist in other conditions. A good laboratory diagnostic test awaits the revelation of a specific etiologic agent which can be definitively identified.

Swine dysentery must be differentiated from other diarrheal diseases of pigs. Salmonellosis is usually diagnosed by isola-
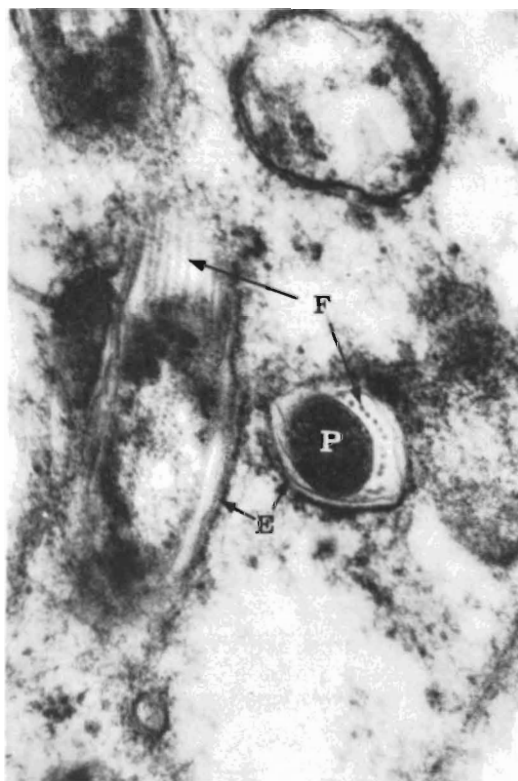
Figure 6. Borrelia-like organisms in degenerating epithelial cell from the colon of a pig affected with swine dysentery showing protoplasmic cylinder (P), axial bbrils (F), and envelope (E). Uranyl Acetate and Lead Citrate stain; x50,000.[8]

tion and identification of *Salmonella spp.* This disease is characterized by hemorrhage and necrosis in both the small and large intestine. Enteric colibacillosis occurs commonly in the weanling pig and is characterized by a watery diarrhea which does not contain blood or mucus. The isolation and identification of a high number of enteropathogenic strains of *E. coli* from the anterior small intestine of affected pigs is confirmatory for colibacillosis.[24]

The wide variety of agents which has been used in the treatment of swine dysentery attests to the unfortunate fact that none are uniformly successful. Arsenicals were among the first sucessful treatments and are still widely used, especially sodium arsanilate which has a relatively low toxicity. The effectiveness of arsenicals appears to have diminished somewhat in the past few years. This may be due to re-

sistance developed by the etiologic agent(s) as a result of wide usage of arsenicals.

Streptomycin, bacitracin, and neomycin are moderately effective. Penicillin and tetracyclines have been used but wtih less satisfactory results. Tylosin has beneficial effects in many cases of swine dysentery and is used not only as oral medication but also via parenteral routes.

Sulfonamides are of some benefit and are often incorporated with other medications such as arsenicals. Nitrofurazone has been used but with a relatively low degree of success. An excellent review of the chemotherapeutic compounds used to treat swine dysentery has been reported.[18]

Two drugs have been used experimentally with very promising results. One of these is Carbadox, [methyl 3-2-quinoxalinylmethylene carbazate N^1, N^4 dioxide].[6, 16] The other is gentamicin, an antibiotic.[11] Neither is currently available but both are expected to be cleared for field use in the near future.

A detailed comparison of the many drugs used to treat swine dysentery would serve little purpose as none are uniformly effective. Remission of symptoms may initially be seen with various treatments but recurrence often follows. The clinician is therefore faced with a continuing problem which depends on experimentation and repeated treatments. The reason for diminished efficacy by chemotherapy may lie in the induction of resistance by the etiologic agent(s).

At the present time, the only reliable method of experimental production of swine dysentery is the inoculation of susceptible pigs with gut material collected from swine affected with the disease. No specific etiologic agent has been consistently shown to cause the disease when inoculated into pigs.

The primary agent which causes swine dysentery is apparently capable of passing a 0.8 micron filter but incapable of passing a filter which prevents the passage of most bacteria. This definitely is an indication that the primary agent is not protozoan (*Balantidium* or *Trichomonad spp.*) because these agents are eliminated by 0.8 micron filters. The possibility of a viral

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agent being responsible as a primary inciting cause of the disease has not been eliminated. Although the 0.45 micron and the Berkefeld V filters allow passage of most viral agents, the failure of these filtrates to elicit lesions of swine dysentery could be due to the absence of essential secondary pathogens.

The fact that the agent(s) is susceptible to 60° C heating for 30 minutes does eliminate spore forming bacteria as significant agents. For the above reasons, the non-spore forming bacteria which are capable of passing a 0.8 micron filter are prime suspects as the main etiologic agent(s) of swine dysentery. *Vibrio spp.* and spirochetes (non-spore formers) are present in filtrates of gut inoculum which produce swine dysentery when inoculated into pigs. These two agents have been directly associated with the lesions of the disease. Whether or not the spirochete organism is capable of invading the gut epithelium without pre-conditioning by other microbial factors is subject to conjecture. It has not been determined if the presence of both agents is necessary for the production of the lesions of the disease. It must be recognized that other agents in the colon of affected pigs could produce toxic products that may be responsible for the pathological manifestations of the disease.

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