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## INTRODUCTION

Coccidiosis in neonatal swine is a clinical disease syndrome associated with infection of the obligate intracellular parasite *Isospora suis*.[1,2] *Isospora suis* infection, with or without concurrent infection of other porcine enteropathogens, is responsible for a significant portion of enteric disease in neonatal swine.[2-5] Although the pathogenicity associated with *I. suis* infection was described almost 50 years ago,[6] it is just recently being considered as a significant enteropathogen of swine.[3,7-9] This apparently growing problem has placed increased demands for methods of prevention and control on the practitioner.

## ETIOLOGY

Coccidia are common throughout the world and can be found in a wide variety of hosts.[10] Coccidia are host-specific and rarely cross-infect other species.[10] There are at least nine species of coccidia recognized in swine[11] and as many as thirteen have been suggested.[10] All but one, *Isospora suis,* are members of the genus *Eimeria* and occur most frequently in adult swine.[11] There seems to be some protection for the neonatal pig against infection with the *Eimeria* species that results from the transfer of maternal antibodies via the sow's colostrum.[3] *Eimeria debliecki* is the most common species found to infect adult swine and may affect as many as one-quarter to one-third of all adult swine.[10] On the other hand, the prevalence of *I. suis* has been found to be very low in older swine (0.5%),[12] but is relatively common in neonates.[3] There appears to be little or no transfer of immunity from the sow to protect the neonatal pig from infection by *Isospora suis*.[3]

*Isospora suis* is the primary pathogen that causes coccidiosis in neonatal swine.[1,2] *I. suis* can cause enteritis by itself or as a concurrent infection with any other enteropathogen that can infect neonatal swine such as enterotoxigenic *E. coli,* rotavirus, and transmissible gastroenteritis virus.[8] In most cases *I. suis* is the only etiologic agent found.[13] In one report, however, the authors found that concurrent infections with enterotoxigenic *E. coli* and/or transmissible gastroenteritis virus occurred in 79% of the cases of porcine neonatal coccidiosis.[14]

Coccidia are obligate intracellular parasites. They undergo both sexual (gamogony) and asexual (merogony) multiplication inside the host's cells. To understand how *I. suis* causes disease in neonatal swine, one needs to understand its life cycle.

*Isospora suis* infection begins when the newborn pig ingests sporulated oocysts. There is a discrepancy in current literature as to the source of these oocysts. Some researchers have been able to correlate *I. suis* oocyst shedding in sows with concurrent infections of *I. suis* in neonatal swine of infected herds.[1,9,15-17] Other researchers have been unable to do so and have suggested that the source of infection comes only from an environment that has been previously contaminated with oocysts from infected pigs.[18]

It has been suggested that oocyst shedding in sows was due to preparturient relaxation in immunity due to the stress of farrowing.[3] Roberts and Walker[16] were able to demonstrate, from fecal samples from sows in an infected herd, that these sows began to excrete oocysts or that oocyst excretion rose during a period from four to five days before farrowing to two to three days post-partum. Oocyst production varied from 100–10,000 oocysts per gram of feces.[16]

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Joyner believes that newborn pigs will normally consume ten to twenty grams of the sow's feces per day and that this is the most likely source of early acquired oocysts.[19] At this rate the newborn pig could ingest a dose of oocysts ranging from 10,000–200,000 oocysts per day.

The inability of coccidiostats fed to sows to completely eliminate coccidiosis in neonatal swine suggests that a previously contaminated environment is a significant source of infective oocysts. Presently it is known only that clinical disease occurs because of the buildup of lethal concentrations of oocysts under optimum sporulation conditions.[3] More experimental work needs to be done to actually pinpoint the source of the infective oocysts.

Before these oocysts can become infective, they must undergo development outside the pig, which is called sporogony. This takes approximately four days,[20] although it has been suggested that in the warm moist environment of a farrowing house, oocysts may become infective in less than four days.[19] The oocysts are very resistant and may remain infective for up to fifteen months in an ideal environment.[20] This would maintain a contaminated environment to serve as a source of infection to neonatal pigs for a very long time.

Once the sporulated oocysts are ingested, they undergo excystation and release their sporozoites in the intestinal lumen. This release most likely occurs through the action of trypsin in the gut.[3] These sporozoites then invade the superficial intestinal epithelial cells of the small intestine.[21] This is the beginning of the endogenous stages of the *I. suis* life cycle.

The endogenous stages can be found throughout the epithelium of the lower small intestine.[22] The highest concentration can be found in the mid-jejunal region, but some stages may be seen in the ileum and even the colon if the infection is severe.[22] The endogenous stages are usually found in the upper one-third of the villous epithelium[22]; however, in cases with severe villous atrophy, they can be found in the crypt epithelium.[22] Extraintestinal stages do not seem to occur.[23]

After the sporozoites penetrate the intestinal epithelial cells, they round up to form trophozoites.[21] These trophozoites can be seen in mucosal smears from the jejunum and ileum as early as 36 hours post-infection.[22] Through asexual multiplication, meronts are formed.[21] These undergo a variable number of consecutive divisions in the same host cell to produce a

total of two to fourteen merozoites.[22] This first generation of asexual stages can be seen in histological sections starting about three days post-infection.[22] The merozoites are released, destroying the villous epithelial cells.[24] They then penetrate other, non-infected intestinal epithelial cells and the process starts all over again producing a second generation of meronts and merozoites.[21]

The merozoites formed from the second generation are released destroying more villous epithelial cells. These merozoites again invade new intestinal epithelial cells, but now they begin to form sexual stages. The sexual multiplication is called gamogony and the sexual stages are called macrogametes and microgametocytes.[21] Macrogametes are more numerous and larger than the microgametocytes, but only one macrogamete is found per cell.[22] The microgametocytes are less numerous and contain highly motile, flagellated microgametes[22] which correspond to the spermatozoa of higher animals. The cell containing the microgametocytes eventually ruptures, releasing the microgametes.[21] The microgametes penetrate the intestinal cells that contain the macrogametes and fertilization occurs, forming zygotes which then develop walls and form oocysts.[21] The intestinal cell ruptures and releases the oocysts into the intestinal lumen to be passed out with the feces.[21] No sexual states are seen in histological sections until four days post-infection.[22]

The prepatent period for *I. suis* has been determined to be five days while the patent period is from five to eight days.[22,25]

The host's cells are destroyed by merogony, gametogony and the releasing of the oocysts. Each oocyst ingested has the potential to destroy many cells. The number of asexual stages and the number of merozoites produced by a species of coccidia remain relatively constant which means that each species has an intrinsic ability to destroy a certain number of cells. Coccidial infections are thus said to be "self limiting" because of their nature of reproduction.[21]

In summary, a neonatal pig picks up a dose of infective oocysts. This infection results in destruction of a certain number of epithelial cells based on the dose of infective oocysts received. The animal sheds oocysts for five to eight days, then is cleared of the infection. Infection with *I. suis* seems to be immunogenic as piglets given a second challenge two to four weeks after the first inoculation fail to pass

oocysts.[5] A relaxation in immunity of sows may occur due to the stress associated with farrowing, thereby precipitating shedding of oocysts.

## CLINICAL FINDINGS

Coccidia in general cause a severe enteritis.[21] In the past, coccidial infection in swine was considered to be of little importance.[3,8,11] There are, however, a growing number of reports which have described the presence of coccidia associated with, or causing, diarrhea in neonatal pigs. These reports have come mostly from the United States[9,11,26-28] with some from Canada[8,14] and one from Scotland.[15] Reports from Canada[14] and the United States[13] have indicated that approximately 12-24% of all cases of neonatal pig diarrhea are diagnosed as coccidiosis.

Coccidiosis is most often associated with diarrhea in young pigs between the ages of five to fourteen days.[8,16] It also seems to be a fairly predictable disease, with the majority of cases in an infected herd breaking with a diarrhea at eight to ten days of age.[8,29] The disease is characterized by a variable morbidity and a variable mortality. It tends to be a chronic herd problem which persists for several months as repeated outbreaks of diarrhea in neonatal pigs.[8,16] Only a portion of the litters are usually affected at one time,[16] and morbidity within a litter can vary from just one pig to all the pigs in that litter.[16] The lack of response to common antimicrobial therapy and/or vaccination of sows for *E. coli* is often a common finding.[8] Coccidiosis is commonly associated with intensive confinement rearing,[8] but can cause serious problems in any type of management situation.[26] There appears to be a seasonal incidence with the greatest number of cases being reported in late summer and early fall.[3,11,26]

The severity of signs is variable, depending on the dose of sporulated oocysts ingested, and the age of the pig.[2] The severity of the disease may range from a poor hair coat and unthriftiness to a severe diarrhea that may result in dehydration, emaciation, coma, and finally death. Not all pigs will get diarrhea, but all pigs that become infected will have some growth retardation.

Outbreaks of porcine neonatal coccidiosis are most often marked by the onset of a profuse watery diarrhea in eight to ten-day-old pigs which, up to this time, had been growing well.[29] The diarrhea is usually a yellow liquid at first and can often be found staining the perineal area.[26] It may become grey and pasty after two to three days.[13] The diarrhea generally lasts between one and five days.[16] Unlike coccidial infection in other species, the diarrhea rarely contains blood.[3,30] Vomiting is not characteristic, but may occasionally occur; the sows of the infected litters are not usually affected and will appear to be healthy.[8,11]

The diarrhea is caused by the destruction of the intestinal epithelial cells, which is caused by the developmental stages of *I. suis*.[3] This results in an intestinal malabsorption diarrhea in much the same way as with transmissible gastroenteritis. This destruction of epithelial cells results in a diarrhea that begins at about three to four days post-infection[2] and if uncomplicated by other diseases, will run its course in five to eight days.[2-5] If the infection is mild, there is regeneration of the intestinal epithelial cells, and the pig will survive. If the coccidial infection is severe, secondary bacterial infection may occur, leading to necrotic enteritis and death.[31] The bacteria most often involved are those from the pig's own natural intestinal flora or *E. coli*.[27]

## LESIONS

The gross lesions that accompany porcine neonatal coccidiosis can vary from none to very severe. In the past, a gold colored fibrinonecrotic pseudomembrane found in the region of the jejunum and ileum of the small intestine was considered to be characteristic of *I. suis* infection.[2,24,25] Recently, it has been suggested that the development of a fibrinonecrotic membrane is common in only severely affected pigs.[2] Other recent studies have indicated that only 7-20% of the pigs with coccidiosis will show a fibrinonecrotic pseudomembrane.[7,8] The number of pigs that will show this pseudomembrane at necropsy is dependent upon the number of sporulated oocysts ingested,[2,25] the age of the pig,[2] and the presence of concurrent enteric infection by other organisms.[32]

Other gross lesions which are often observed include thin-walled intestines filled with watery intestinal contents, the lack of chyle in the lacteals and a catarrhal enteritis with or without fibrin strands. Many of the pigs may, however, show no gross lesions at all.[8]

The predominant histopathologic lesion is villous atrophy[32] caused by the necrosis and sloughing of intestinal epithelial cells, which is

caused by the multiplication and subsequent release of coccidial forms.[32] The segmental villous atrophy occurs mainly in the jejunum and ileum[32] and may be mild to severe with crypt hyperplasia.[32] In some cases, a severe diffuse mucosal necrosis and fibrinous cellular exudate occurs.[8] Bacterial invasion of the necrotic areas will result in necrotic enteritis.[32]

Numerous coccidial forms can be found within the intestinal epithelial cells or loose in the intestinal lumen. Coccidial forms are the most numerous when the intestinal lesions are mild[32] and are harder to find in cases that have severe intestinal lesions.[32] The stages seen depend on the number of days since the pig was first infected. In general, however, meronts and merozoites are the most common stages encountered at the onset of diarrhea.[8]

## DIAGNOSIS

The diagnosis of porcine neonatal coccidiosis can be made from clinical findings, stained impression smears, smears of mucosal scrapings, the presence of occysts in feces or colonic contents, histopathology, and occasionally from gross lesions.

Clinical findings can aid in the presumptive diagnosis of porcine neonatal coccidiosis until laboratory confirmation can be made. The clinical findings that would support a diagnosis of porcine neonatal coccidiosis would include the following:

1. Repeated outbreaks of diarrhea in 5–14 day-old pigs which had been previously doing well.
2. The lack of response to routine antimicrobial-antidiarrheal therapy or to vaccination of the sows for *E. coli* and/or TGE.
3. The rest of the herd appearing to be healthy, including the sows with the affected litters.
4. Not all of the litters in the farrowing house are affected.

Stained impression smears[7] and smears of mucosal scrapings[33] are probably the most useful tools for the diagnosis of porcine neonatal coccidiosis and can be made quickly and easily in most any practice. This results in a rapid diagnosis and allows immediate implementation of control and preventative measures. The pigs selected for examination should be freshly dead or killed as coccidial forms are harder to identify if the pig has been dead long.[7] Not all

the pigs in an affected herd will be positive, so examination of several pigs is best.[7] Several areas of the jejunum and ileum should be sampled, especially those areas that show any gross lesions.[7] After staining, various developmental stages of coccidia can be visualized. The most common forms seen are merozoites which are dark staining and crescent to comma shaped. Impression smears have been proven to be as reliable as histopathology.[7]

Looking for oocysts in feces of affected pigs or colonic contents is perhaps one of the easiest things to do, but it is not a reliable way to diagnose porcine neonatal coccidiosis. Oocysts are generally passed in the feces two to three days after the diarrhea begins.[33] However, they may only be seen in the feces of about 50% of the infected pigs.[5] Pigs heavily infected may die before oocyst production occurs.[25]

Histopathology is very reliable but is also time consuming and expensive. The presence of endogenous stages of coccidia associated with villous atrophy and necrotic enteritis is characteristic. Histopathology can also be used to evaluate concurrent diseases as well as determine lesion severity.[13]

Gross lesions are only occasionally of diagnostic value. However, a fibrinonecrotic membrane found in the jejunum or ileum is very suggestive.

Other diseases causing enteritis in 5–14 day-old pigs that should be considered in a differential diagnosis include enterotoxigenic *E. coli*, *Clostridium perfringens* type C, rotavirus, and transmissible gastroenteritis virus.

## PREVENTION AND CONTROL

Treatment of porcine neonatal coccidiosis is usually of little benefit since permanent irreversible damage to the intestinal mucosa has already occurred by the time the diarrhea occurs. Control and prevention is the best way to manage *Isospora suis* infection in neonatal pigs.

Control of porcine neonatal coccidiosis is based on the prevention of clinical signs by reducing the number of infective organisms. The two main sources of infective organisms to the neonatal pig are the oocyst-shedding sow[16] and the contaminated farrowing units.[18] Prevention and control is attempted through strict sanitation and the use of coccidiostats.

Thorough disinfection and sanitation of the farrowing area is of the utmost importance for control of porcine neonatal coccidiosis. Before entering the farrowing area, the sow should be

scrubbed clean of all fecal matter and dirt. The farrowing crates or stalls should be thoroughly cleaned and disinfected between each use. Coccidial oocysts are very resistant to many of the common disinfectants,[34] so a thorough mechanical cleaning of the area is probably the most important. When the sows and pigs are in the crates or stalls, all the manure should be removed daily. Manure, the source of infective oocysts, should not be allowed to build up. There are no short cuts to good sanitation.

A wide variety of coccidiostats have been used in an attempt to control porcine neonatal coccidiosis. By interrupting the coccidial life cycle, coccidiostats can reduce oocyst production as well as clinical disease. One must remember that these drugs only retard coccidial growth and do not remove all the coccidia from the gut.[21,25] They help reduce the problem but can not eliminate it. There are no coccidiostats approved for use in prevention and control of porcine neonatal coccidiosis. However, some of the therapeutic agents that have been used as coccidiostats in swine include several of the sulfonamides, amprolium, decoquinate, monensin, nitrofurazone, and a chlortetracycline combination. These therapeutic agents have been used in sows to reduce oocyst shedding and/or in neonatal pigs to reduce or prevent clinical disease.[1,8,9,13,16,17,21,26,28,35-37]

Sulfonamides have been used to control coccidiosis in other species for a number of years. They interfere with folic acid synthesis and exert their maximum anticoccidial effect against second generation meronts.[38] Sulfonamides that have been shown to be effective against coccidiosis in other species include sulfaquinoxaline,[35] sulfadimethoxine,[29] and sulfamethazine.[39] Sulfonamides can be used to control oocyst shedding in sows. When used this way therapy should begin approximately 10-14 days before farrowing and be continued 7-10 days post-farrowing. The daily oral dose should be in the range of 50-100 mg/kg.[36] These drugs are probably not a very effective treatment for affected baby pigs in an outbreak of porcine neonatal coccidiosis. Since they act on the second generation meronts, serious damage has already been done by the time they begin to work. A suggested daily oral dose for piglets should be 130-200 mg/kg.[29]

Amprolium has its peak anticoccidial effect very early in the coccidial cycle and thus shows promise for use in controlling outbreaks in neonatal pigs. It is a thiamine antagonist and thus inhibits folic acid synthysis.[38] Although similar in this respect to the sulfonamides, its peak activity is reported to be on the first generation meronts.[38] Its activity can be enhanced by mixing with ethopabate, sulfaquinoxaline and other agents that also inhibit the synthesis of folic acid at different steps.[38] To control oocyst shedding in the sow, amprolium should be fed at a daily dose rate of 20 mg/kg[29] for 10-14 days before farrowing and continued until 7-10 days after farrowing. Amprolium can also be used in neonatal pigs to help control outbreaks of porcine neonatal coccidiosis. Because it acts very early in the coccidial life cycle it may reduce the severity of clinical disease. If used in neonatal pigs it should be given three to four days before the expected outbreak of diarrhea occurs and continued for several days. In most herds this will mean orally dosing each pig with approximately 50 mg/kg of amprolium solution when they are four, five and six days old.

The coccidiostat decoquinate acts very early in the coccidial cycle preventing the development of the first generation meronts.[38] Feeding it after exposure probably does little good.[38] This drug also has a greater tendency to allow drug-resistant strains to develop.[38] It probably has little use in control of swine coccidial infections. If used it should be fed at a rate of 0.5 mg/kg daily in the same manner as previously discussed for the other coccidiostats.

Monensin's anticoccidial effect is confined to the first two days of the coccidian life cycle. It works by inhibiting the mitochondrial function of the trophozoites and first generation meronts.[38] It can be fed in the sow ration at a rate of 100 grams/ton of feed for the same time period as the other coccidiostats previously mentioned.

Nitrofurazone exerts its anticoccidial effect on the middle stages of the coccidial life cycle.[21] A suggested dose is 400-600 grams/ton of sow feed which is then fed in the same manner as the other coccidiostats.

Onawunmi and Todd showed that the combination of chlortetracycline, sulfamethazine and penicillin[a] can also act as a coccidiostat in swine.[37] The sulfamethazine is probably the active ingredient that is exerting an anticoccidial action. It was fed at a rate of 375 mg of active ingredients in the feed daily. It too should be given to sows 10-14 days before farrowing and

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[a]Aureo S-P-250, American Cyanamid, Princeton, NJ.

until 7–10 days post-farrowing for control of oocyst shedding.

Many different drugs and drug regimens have been used to try to control coccidiosis, but none have been successful in all herds all the time. It appears that keeping the newborn pigs' environment as clean as possible and free of fecal contamination is the most important step in control and prevention of porcine neonatal coccidiosis.

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