EPIDEMIOLOGY, DIAGNOSIS AND CONTROL OF ENTERIC DISEASES IN GROWING-FINISHING PIGS

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Introduction

Enteric diseases of swine are common and important in swine veterinary medicine. This paper will look at the epidemiology, diagnosis and control of the main enteric conditions affecting growing-finishing pigs.

Swine Dysentery

When I graduated from the veterinary college, 26 years ago, swine dysentery (SD) was the pig disease I had to deal with most often in finishing units. Today, it has almost disappeared from our Canadian units and is only rarely identified in diagnostic laboratories. The exact reasons for this virtual elimination have not really been investigated, but I suspect that the following are among those which may have played a role:

1) The generalization of early weaning and all in/all out systems. Early weaning has likely reduced the number of piglets that would get out of the farrowing crates already infected, and all in/all out systems have reduced the likelihood that pigs would get infected because of the environment in which they are placed.

2) The increased popularity of segregated early weaning systems.

3) A reduction in the number of sources of pigs that are mixed, particularly in finishing units. In my early days as a practitioner we had small sow herds and relatively large finishing units. It was not uncommon to have pigs from 20 to 60 different sources introduced in the same finishing units. Today the mixing of different sources of pigs is avoided as much as possible, and when mixing does occur it usually involves only a few herds, and only rarely up to 10 sources. So monitoring and control of the health status of these sources is much easier than it used to be.

4) With sound biosecurity rules and introduction of only *Brachyspira hyodysenteriae* (BH)-free animals, it does not seem difficult to maintain a BH-free status, even in areas where swine production is quite intensified. While some organisms like PRRS virus and *Mycoplasma hyopneumoniae,* for example, seem to have different means, other than the introduction of infected pigs, by which they can infect swine herds, overall these indirect transmission means do not seem to play an important role in the epidemiology of SD. So it is usually not difficult to prevent introduction of this organism in swine barns.

5) Most breeding stock suppliers had BH-free animals to sell, so those that did not had to get rid of this organism or stop selling breeding animals.

6) There are efficacious eradication programs for BH.

There are no SD vaccines in Canada and prevention has to rely on something else. For herds that are not infected with BH, the idea is not to introduce it. As mentioned above, sound biosecurity rules and introducing only negative animals are usually enough to stay BH-free. If the organism is already present in a herd, early

weaning coupled with a strict all in/all out system in the farrowing, nursery and finishing sections, and a proper sanitation and rodent control program should be enough to significantly reduce the prevalence and importance of the condition. Feeding the pigs rations that contain effective anti-BH products after weaning may be useful in cases where other preventive strategies are not producing the desired results.

BH is reported sensitive to several disinfectants, including quaternary ammonium and phenolic compounds, sodium hypochlorite and organic iodines. The organism survived for 61 days in feces diluted in water at 5º C, and for 7 days in feces at 25º C.[1] In an experiment conducted by Boye et al[2] the survival time in swine feces was found to be 112 days at 10º C. So making sure that pigs do not have access to anything that could be contaminated with feces is crucial. It is possible for piglets to get infected with BH in the farrowing crates, but it is not considered a frequent event. Most of the contamination is thus likely to occur after weaning or after placement in the finishing unit. Pigs have been shown to shed BH for as long as 70 days after cessation of clinical signs.[3]

The organism has been found in farm mice and rats, and experimentally these species were shown to shed it for respectively 180 and 2 days.[4,5] An effective SD control strategy should thus include a rodent control program. In a recent study, it was found that a finishing site receiving piglets from two herds thought to be BH-free had experienced SD for at least 2 years.[6] The operation was run strictly all in/all out with cleaning and disinfection of pens between batches of pigs. Mice captured on the farm were found to be positive to BH, and comparison of the strains in pigs and mice by pulse field gel electrophoresis (PFGE) showed that both species were carriers of the same strain. The authors suggested that mice acted as reservoirs of BH in that herd.

The organism was also isolated from a dog that frequented pens containing pigs affected with SD.[7] Experimentally flies have been shown to carry BH up to 4 hours, and starlings for 8 hours.[1] BH has been recovered from naturally infected rheas in which it caused a necrotizing typhlo-colitis.[1] Recently the organism was also identified in both farm and wild mallard ducks.[8] However, while animal species other than pigs and even insects may have the potential to play a role in the epidemiology of SD, this role is not thought to be very significant since, as mentioned above, it seems fairly easy to remain free of this infection even in pig dense areas.

In a study conducted by Siba et al[9] it was shown that pigs fed a diet made of cooked rice and animal protein were protected against challenge with BH, while pigs consuming a commercial diet based on wheat and dehulled lupins were severely affected. However, in an experimental model Kirkwood et al[10] were unable to show any protection afforded by diets against SD, including diets formulated with either parboiled or cooked rice. Lindecrona et al[11] reported that fermented liquid feed had a preventing effect on the development of swine dysentery.

There have also been reports in the past of the possible partial protection allowed by diets containing supplementary zinc, or supplementary Vit E, B2 and selenium. Some types of diet, or some elements of the diet could thus have a positive impact on the prevalence and severity of SD, but more work may be needed to reach a consensus on the cost effectiveness and practicality of their usage.

When SD was still a problem in Canada, nitroimidazoles, carbadox and tiamulin were the main products used to prevent and/or treat SD. Tylosin and arsenicals were not of much value, and the results with lincomycin were inconsistent. Nitroimidazoles and carbadox are not allowed anymore in my country, and valnemulin is not available, so we are mainly left with tiamulin for the few cases that are still diagnosed. Pigs clinically affected with SD should be treated parenterally and tiamulin usually worked

extremely well in our cases by injection. It also worked very well when administered in the water at 49 ppm. In the feed high dosages (178 ppm) were often needed to get a proper control. Strains of BH that are resistant to tiamulin have now been identified in different countries of Europe such as Poland, Hungary, Finland, Germany and the Czech Republic.[12]

One of the main problems we had in the treatment of SD was relapses. Although there could be others, the following reasons appear to be among those that could explain these relapses. First, even if the disease is controlled by medication, it does not mean that all animals have been cleared from the infection and don't carry and shed the organism anymore. They can thus serve as a source of re-infection once medication is removed. Second, the organism is resistant in the environment, so unless this environment is properly cleaned and disinfected before the treatment is stopped, treated animals can re-infect themselves because of the contaminated environment. Third, infected rodents present on the farm could also be the cause of re-infection. Fourth, when a SD problem occurs on a farm it does not necessarily affect all susceptible pigs at the same time, so those that were not affected before will still be susceptible. Finally, even if all susceptible pigs in the population came in contact with the organism at the same time, the use of an effective medication may still interfere with their immunization process.

Different successful eradication programs for SD have been described. These programs involve the use of an efficacious anti-BH product for a variable period of time, stringent cleaning and disinfection of the premises in which 'cleaned' pigs are placed or left, and a good rodent control program to make sure that rats and/or mice cannot serve as a source of re-infection. On a worldwide basis tiamulin is the product that has been used the most to eradicate BH from swine farms. For example, Blaha et al reported[13] an eradication program that involved the injection (10 mg/kg) of all pigs on the farm, except piglets under 3 weeks of age, for 5 consecutive days. On a large 2000 sow farm, farrow to finish, Janc et al[14] used tiamulin in the feed of all pigs at 8.8 mg/kg for 10 days, then at 5 mg/kg for 40 days. Suckling piglets received a feed containing 440 ppm of the antibiotic, and were orally administered a dose of 8.8 mg/kg individually for 4 days. Finally Jensen et al[15] reported the successful eradication of BH in 24 of 26 herds by using either medication in the feed (60 to 100 ppm for 14 days) or in the water (60 ppm for 7 days), and injection of suckling pigs (10 mg/kg on days 0, 7 and 14). Given the increasing resistance of BH to the various compounds available, including tiamulin, the choice of the product and dose to use should be made based on sensitivity results of the isolate(s) present on the farm, or, if not available, on field experiments that would evaluate the efficacy of the main products at various dosages. Similarly, because of the poorer survival rate of BH in warm and dry conditions, eradication attempts of BH should ideally be planned during the summer months.

MEW and SEW programs have been quite effective at producing BH-free pigs from BH-positive herds.[16]

Porcine Colonic Spirochetosis

We have in Quebec, the province in Canada where I live, a monitoring system that lists and briefly describes the main conditions, and the new ones, that are diagnosed in our different veterinary diagnostic laboratories every year. In the last seven years porcine colonic spirochetosis (PCS) was not mentioned even once. This does not mean that it is never diagnosed, but it does mean that its importance is very low. The condition does not seem to be a significant concern in the US either. In 2001, Schwartz[17] reported that of 3202 diagnoses of enteric diseases made at the Veterinary Diagnostic Laboratory of Iowa State University, 0.2% only were associated with

Brachyspira pilosicoli (BP). Concerning the prevalence of the condition in Spain, de Arriba et al[18] investigated the cause of post-weaning and growing pig diarrheas in 125 farms in 2001. PCS was found in only two of them.

The epidemiology of colonic spirochetosis has not been studied in detail and is not very well known. Apart from pigs, BP has been isolated in dogs, rats, different kinds of birds and in primates, including humans. It has also been identified in water samples, so water could potentially serve as a source of infection.[19] In one experiment the organism survived up to 210 days in feces at 10ºC, so it is resistant in the environment.[2] In an epidemiological study conducted in two different Australian herds, a number of isolates were subjected to multilocus enzyme electrophoresis and to pulse field gel electrophoresis. The six isolates recovered from the first herd were all of the same type, but the ten from the second herd were considered genetically heterogeneous, being divided into 6 different electrophoretic types and 7 pulse field gel electrophoresis types.[19] In another study, this time conducted in Finland, 131 BP isolates from 49 herds were studied by pulse field gel electrophoresis.[20] Most farms had distinct genotypes and common genotypes among herds were rare. Such diversity does complicate the picture when comes time to identify by what means, other than infected pigs, herds could become infected.

Overall it is thought that the same principles and products used to control SD can be used to control PCS. In the Spanish study reported above, a rapid clinical response was obtained with 125 ppm of tiamulin in the feed in one farm with PCS problems.[18] Karlsson et al[21] looked at the sensitivity of Swedish field isolates of BP to different antibiotics. The MICs at which 90% of the isolates were inhibited by tylosin, erythromycin, clindamycin (for lincomycin), virginiamycin, tiamulin and carbadox were >256, >256, >4, 4, 2 and 0.125 microg/ml. The efficacy of four types of disinfectants on field isolates from Scotland was recently evaluated by Corona-Barrera et al.[22] Quaternary ammonium compounds and tar organic acids were the most efficacious while peroxygen was the least efficacious.

The influence of a diet based on cooked rice on colonization of pigs by BP was compared to a standard diet based on wheat and lupins.[23] Pigs were experimentally infected with BP. One pig receiving the standard diet had acute diarrhea and a severe erosive colitis with end-on attachment of spirochetes to the colonic epithelium. Overall however the challenge only produced loose feces on one or two sampling days, and there were no weight differences. Consumption of the rice-based diet delayed and significantly reduced the onset of BP fecal excretion. The influence of five different diets on the experimental infection of pigs with BP was also evaluated in Denmark.[24] The clinical signs of BP infection varied from loose stools to watery, mucoid diarrhea. The group fed the diet based on cooked rice excreted BP for a significantly shorter period than the group fed the standard diet based on wheat and barley, and fewer of them excreted the organism. All the pigs fed the pelleted standard diet excreted BP in their feces and significantly more of them showed clinical signs of disease than the pigs fed the non-pelleted standard diet. A fermented liquid feed and one containing lactic acid had no significant impact on excretion or on the number of pigs with clinical signs.

Fossi et al[25] have reported a successful eradication program for BP where tiamulin was used at 200 ppm for a period of 18 to 30 days, depending on the age group. The piggery unit was emptied, cleaned, disinfected and dried, and all worn surfaces were repaired. The animals were removed to temporary sheds situated 0-100 m from the piggery unit. Only the sows and the boars returned to the piggery unit, all other pigs were sold from the sheds within 3 months after the eradication. Post weaning diarrhea had been a severe problem in this herd for years, and BP, as well as *Brachyspira innocens* had been isolated repeatedly from the herd. Immediately after

the eradication program the post-weaning diarrhea disappeared, and samples obtained after the program remained negative to BP.

Salmonellosis

Another disease that was a significant concern after my graduation from the veterinary college was septicemic salmonellosis associated with *Salmonella choleraesuis* (SC). If not treated properly losses associated with it were sometimes serious. Interestingly, this pathogen has also virtually disappeared from Quebec, to the point that in the last ten years I did not have to deal with a single case. Although this is mainly speculation and there could be other factors, I suspect that the following ones may have played a role in the apparent elimination of this organism from our herds: earlier weaning; all in/all out practices in farrowing, nursery and finishing sections; no market for herds selling infected animals (feeder pigs or breeding stock) so they had to get rid of SC, or stop producing pigs; less mixing of animals from different sources in the nursery and finishing units; an organism that is rarely isolated from animals other than pigs and, finally, indirect transmission (associated with something other than introduction of infected pigs) that does not seem to be as difficult to prevent as with other pathogens like PRRS virus, for example.

The peak of SC problems in Quebec appeared to be in the late seventies early eighties. At that time *Salmonella typhimurium* (ST) was rarely involved in pig problems and when it did show its head, it was usually not very bad. Today ST is regularly diagnosed in Quebec herds as a cause of diarrhea, wasting and mortality, mainly in nursery and finishing pigs, and losses associated with it can be serious. While most ST strains are still sensitive to ceftiofur, one of our last resort drugs for particularly resistant pig pathogens, there are situations where an increasing number of them are not anymore. For example Dr. Claude Tremblay, a fellow practitioner from Quebec, has recently looked at the sensitivity pattern of 29 isolates of this bacterium in his practice and found that 5 (17%) of them were not sensitive to ceftiofur (2 resistant, 3 limited, 24 sensitive). While this could still be considered as quite good, the increasing number of strains that are not sensitive to this important antibacterial agent, as well as to many of the other ones that can be used in swine, is a second concern that we should have in relation to ST. Since antibiotics have to be used to control acute outbreaks of the condition caused by ST, at least with the known therapeutic tools currently available, a reduction in their efficacy cannot be good news.

Although this should be enough to worry about there is a third concern, potentially much more damaging to our industry, that we need to be aware of. *Salmonella* is the second most important zoonotic agent in people, behind *Campylobacter*. Furthermore, of the more than 2400 Salmonella serotypes that have been identified so far, ST is usually number one or two in importance for people, in front or behind *Salmonella enteritidis*, a serotype that they usually get from the consumption of eggs or egg products. So if one had to imagine a particularly bad scenario it would be a severe and large outbreak of human salmonellosis caused by an ST strain that is very resistant to antibiotics, very virulent and seriously affects the health of people, and for which it is proven that the source of infection was pig products.

ST also seems to be a significant pathogen in Spain. In a study conducted by Vidal et al[26] in 84 herds with problems of diarrhea, fecal samples collected in these herds revealed the presence of *Salmonella* in 26 (31 %) of the herds. *Salmonella* was the only identified agent in 13 of them (50%). Serotyping was conducted on 29 Salmonella isolates found in 21 herds. ST was the most frequently identified serovar (24.1%). The authors concluded that *Salmonella* was frequently isolated from diarrheic

outbreaks in Spanish swine herds, and that the serovars isolated were among the most commonly recovered from cases of human salmonellosis in this country.

Realizing the importance of ST for both pigs and people, what can we do to control the problems that can be associated with it in pigs? If a herd is free of ST, the idea is naturally to remain free, but knowing whether a herd is free or not of this organism is not as simple as for others to start with. There is no commercial test in Canada that specifically detects antibodies against ST. We have different tests that can detect antibodies against organisms of the genus *Salmonella*, but not against ST per se. This means that a positive serological reaction could be related to antibodies against *Salmonella derby*, he*idelberg* or many other serotypes of *Salmonell*a, and not necessarily to ST. Thus serology is not at this time a technique that allows us to accurately detect the presence of ST in a herd, even though herds that have a high number of positive reactions are often found to be infected with it. Confirmation of contamination is for now usually relying on isolation and characterization of the bacterium. When there are no clinical signs, pools of feces can be submitted to the diagnostic laboratory for that purpose, but one needs to take multiple samples on a more or less regular basis to have some confidence that the herd is truly negative. If clinical signs are present, the organism can normally be isolated easily from diarrheic feces, the intestines and sometimes from other tissues of affected pigs as well.

If a herd is ST-free, the first thing to do is to make sure that animals introduced in that herd are also ST-free. Obtaining a good health status history from the supplying herd's veterinarian is always a good idea when comes time to decide where to buy pigs. Has ST been identified in the past in the supplying herd, or in herds that have bought animals from it? Have bacteriological tests been conducted on pools of feces to determine if the organism was present or not and if so, has it been done recently? Was it done only once or is there a regular monitoring program? Obtaining a good history from the seller's veterinarian is particularly important for pathogens like ST for which simple diagnostic techniques like serology are either not available, or not totally reliable. Having a closed herd where no animals from the outside are introduced is evidently even safer, but this is not possible everywhere and is not without some disadvantages that need to be considered and weighed before going that route. If animals have to be introduced, the fewer sources the better. In a recent study conducted in Canada, high seroprevalence to *Salmonella* in finishing was 25 times more likely to occur in farms obtaining animals from more than one source, compared to those that had only one supply source of pigs.[27] While as mentioned above seroprevalence here could be associated to many different *Salmonella* serotypes, and not to ST specifically, the principle for prevention remains the same.

Another difficulty or hurdle with ST is that most animal species, including horses, cattle, dogs and cats, as well as birds can be carriers of the bacterium, and can thus serve as a source of infection for pigs. The same in fact is true for people, so infection is not only possible from pigs or pig products to people, but can go the other way around. In the case of pig farms, one of the main potential dangers coming from non porcine hosts is rodents, so a good pest control should be in place to reduce the risks associated with these undesired guests.

As for many other swine pathogens, the epidemiology of infection and disease associated with ST is not totally understood, and there are farms that become infected without an obvious source of infection being detected. Since ST has been found on flies caught in farms that had clinical problems of salmonellosis, insects could potentially serve as a source of infection between farms, but this has not been proven. Aerosol transmission of *Salmonella enteritidis* in poultry, and more recently *Salmonella agona* in pigs has been demonstrated experimentally over short distances, but the role that this transmission means can play between farms is not believed by thought leaders to be a significant threat. Of course if the neighbor has an ST-infected herd and

manure from this farm is spread close to a negative herd, there is certainly a potential risk there, whether related to insects or aerosol. ST can remain viable in manure for extensive periods of time (months), so anything that has been in contact with manure from infected pigs should not find its way into a negative herd. In a study by Letellier et al[28] the level of environmental contamination in farms that had shown clinical salmonellosis associated with ST was impressive. The organism could be detected from samples of fecal material from pens, water (tap), feedstuff (in the troughs), floors, doors, ventilation units, dust, dead animals, rodents, flies, boots, shovel and exterior soil near dead animals (Quessy S, personal communication, 2005). So biosecurity rules should be such that direct (pigs) and indirect (transport, fomites, people, other animals or birds, etc.) contamination can be prevented.

As can be seen remaining negative to Salmonella and to ST specifically can be a challenge. Yet many farms do show negative results time and time again so it is not as if it were impossible to achieve. In fact if Sweden was able to remain almost free of *Salmonella* organisms on a country basis, it must be feasible on a farm basis.

The treatment of affected pigs is best done, at least at this time, by injecting them with an antibiotic to which the strain is sensitive. For this strategy to be of value, pigs need to be injected early in the course of the disease, with the right product, the right dose and as long as the animals show clinical signs. Although some of the indications available suggest that ST may become more resistant to antibiotics most strains, at least in North America, are still susceptible to ceftiofur and potentiated sulfas (a sulfonamide with trimethoprim, for example). It is sometimes possible to limit the progression of the disease in a barn by also injecting all the other pigs in the pens of pigs showing clinical signs. Measures should also be taken to make sure that personnel that get into pens to inject pigs or to clean the pens will not carry the organism throughout the barn with contaminated boots, clothes or any other indirect transmission means (e.g. material, instruments). In cases where the disease progression justifies it, it may be necessary to add an antibiotic in the feed or in the water. However the tendency at this time being to reduce the use of antibiotics in swine production, the long term use of a strategy based on antibiotics should not be favored.

There are no commercially available vaccines in North America that are specific to ST. We have however modified live vaccines produced from attenuated strains of SC. These vaccines, that are administered in the water or intranasally, provide excellent protection against SC, but also offer cross protection against ST. The level of that cross protection in the field appears to vary since the results are sometimes excellent, and sometimes somewhat inconclusive. In a recent experimental study Neubauer et al[29] showed that the mortality in vaccinated animals was 4 times less than for unvaccinated pigs (4.8 vs 19.0 %). In addition vaccinated pigs were 4.4 lbs heavier than unvaccinated pigs 14 days post challenge. These live vaccines have also been shown in different studies to reduce the carrier rate and seroprevalence to *Salmonella* sp. of pigs at slaughter. A reduction in the carrier rate of pigs at slaughter is especially beneficial from a food safety point of view since there is a correlation between the carrier rate of pigs shipped to slaughter and the contamination of pork products.

The type of feed used is another factor to consider. If there are problems associated with ST and if the feed used is pelleted, a switch to mash (meal) feed may not only help to prevent the occurrence of these problems, but as for vaccines it may also reduce the carrier rate and seroprevalence in slaughter pigs. Quessy[27] reported that in a study involving 300 Canadian herds, those using mash feed were 30 times less likely to have a high *Salmonella* seroprevalence when compared to a standard herd. A physiologic reason for this impact may have been provided by Mikkelsen et al[30], in a study where they looked at the effects of feed grinding and processing. Pigs fed the coarse, non pelleted diet showed increased in vitro death rate of ST DT12 in

content from the stomach ($p < 0.001$). Pigs fed this diet had a significantly higher concentration of undissociated lactic acid in gastric content than pigs fed the other diets ($p < 0.001$). A strong correlation between the concentration of undissociated lactic acid and the death rate of ST DT12 was found. It was concluded that feeding a coarsely ground meal feed to pigs changes the physicochemical and microbial properties of content of the stomach, which decreases the survival of *Salmonella* during passage through the stomach.

When dealing with ST problems producers must pay a particular attention to hygiene because, as mentioned above, the level of environmental contamination can be very high. A thorough disinfection program must thus be undertaken to insure that pigs cannot have access to anything contaminated with the organism. In Quebec trials, a disinfectant based on glutaraldehyde and a quaternary ammonium offered a good quality/price ratio. As is the case for PRRS, allowing the surfaces to dry before pigs have access to them is of paramount importance and should not be overlooked. Because it is not easy to properly disinfect the environment in farms that are having ST problems, it is recommended to verify that the program is efficacious by testing the disinfected surfaces for the presence of the organism. This can be done by scrubbing disinfected and dried surfaces with gauzes, using a specific procedure, and sending them to the diagnostic laboratory where isolation of *Salmonella* is attempted. If it is isolated it means that the disinfection program is not efficacious and must be reevaluated. If it is not, it should indicate that the surfaces have been properly disinfected and are less likely to constitute an important source of infection.

Since contaminated manure is the main initial source of infection, everything that can be in contact with it must be considered as a potential source of infection, and boots are high

in the list of fomites that can play a role in diffusion of the organism. Although there are studies in which ST has been found in the feces of nursing piglets, there seems to be many herds where piglets are found negative at weaning. In studies conducted in Quebec, it was shown that pigs rarely become infected with *Salmonella* before 10-12 days of age. This finding was actually used to produce *Salmonella* negative pigs from *Salmonella* positive farms (Quessy S, personal communication, 2004). The idea here was to wean pigs at a maximum of 10-12 days of age and move them to sites that were not contaminated with *Salmonella*.

A plethora of other strategies and products have been reported in different publications to have a potential beneficial impact in the control of *Salmonella* sp. problems. These include but are not limited to the following: addition of organic acids in the water or feed, bambermycin in the feed, the use of liquid fermented feeds or whey, antibodies in egg yolk powder from immunized hens, inclusion of high fiber grain in the ration, all in/all out pig flow, probiotics, fructooligosaccharides and sodium chlorate. While the results obtained with some of these alternatives were quite interesting, for others more work is needed before their true value can be assessed.

Ileitis

Porcine proliferative enteropathy, or ileitis, is caused by an obligate intracellular bacterium, *Lawsonia intracellularis* (LI). Although we do have both the chronic and hemorrhagic (PHE) form of the disease in Quebec, the latter is much more frequent and important.

The epidemiology of ileitis is both interesting and complex. Of course introduction of infected animals is an important means by which swine farms become infected, but there are clearly situations where this is not the case, and for which the

origin of infection remains a mystery. For example, many farms that have been populated by hysterectomy, or with LI-free animals, eventually became infected and we know that it was not associated with the introduction of infected pigs. It thus seems that indirect transmission of this organism is possible, even in farms with strict biosecurity measures.

LI has so far been recovered from a multitude of animal species: mouse, rat, guinea pig, hamster, rabbit, hedge hog, dog, wolf, fox, ferret, horse, calf, deer, giraffe, ostrich, emu and monkeys. Reproduction of proliferative intestinal lesions with LI has now been demonstrated in pigs, hamsters, mice and foals. Abshier[31] et al reported spontaneous proliferative typhlocolitis associated with LI-like bacteria in mice bred for zoo animal feeding. It is also known that porcine LI isolates can produce the disease in hamsters. The role that other animal species, particularly rodents, may play in contamination cases that are not involving pigs will thus have to be investigated. Insects should not be totally disregarded either, whether it is as hosts that could be infected themselves, or simply as potential mechanical carriers of the organism.

Lavritsen et a[32] have investigated the ability of sows to transmit infectious organisms to their piglets during a lactation period of 21 days. Five sows from a herd infected with different organisms, including LI, were moved to uncontaminated premises. Their piglets were weaned to a different clean site. LI was detected by PCR on feces from a piglet as early as 10 days of age, indicating that sows could be a source of infection for their piglets. However, it is thought that most pigs become infected after weaning, after exposure to piglets shedding LI or to contaminated premises.

In a recent study conducted by Stege et al[33] in 5 farms, 100 pigs (20 per farm) were followed serologically and by PCR starting at weaning, and every two weeks up to slaughter. Clinical disease was not reported but infection was present in all herds, and 75% of the pigs were found infected by PCR at one point in time or another. Only one pig in one herd was PCR positive at weaning (about 4 weeks of age). Most infected pigs were shedding LI at 10-12 weeks of age, and shed for 2 to 6 successive weeks. After 18 weeks of age all shedding had ceased and reinfection at PCR detectable level was not seen. Relative to bacterial shedding, seroconversion was delayed by about two weeks, and once sero-converted, most pigs remained seropositive up to slaughter. In another study this time with experimentally infected pigs, the longest duration of shedding detected was 12 weeks for a pathogenic strain and 9 weeks for a vaccine strain.[34] The organism has been shown to survive for 2 weeks in feces kept between 5 and 15º C.[35]

Chouet et al[36] looked at the time pigs became seropositive in 33 French farms and 29 Spanish farms. Four farms in France and 3 in Spain remained free of clinical signs and were found to be seronegative to LI. The postweaning pigs on all of the remaining French farms and on 20 of the 26 remaining Spanish farms had a pattern of infection characterized by seroconversion in the grower period, generally between eight and 16 weeks of age, and at least 15% of the breeding females tested were seropositive. These farms were farrow to finish operations on one site. On the six remaining Spanish farms a multiple-site system was used, and on three of them the seroconversion was delayed, at between 16 and 20 weeks of age, and none of the breeding females tested were seropositive. The percentage of pigs found infected at different ages appears to vary significantly from one study to the other, so care should taken before extrapolating the results of one specific study to other situations. The type of production system and the medication programs used can, among others, have an impact on the first time pigs become infected and carriers. Variations in results obtained could also be due to differences in the sensitivity and specificity of the tests used.

Collins et al[37] showed that pigs were protected (immune) after experimental infection followed by challenge 7 weeks after detection of fecal shedding of LI had ceased. Love et al[38] found that in a herd where two successive outbreaks occurred about 2 months apart, animals involved in the first outbreak did not show clinical signs during the second outbreak. Furthermore, clinical cases occurred only in animals recently introduced into the breeding population. It thus seems that initial contact does result in development of immunity, but at this time it is not known if this immunity lasts for months or for years. Results obtained following vaccination however are suggesting that immunity to ileitis could be of long duration, possibly for the productive life of the animal. It should be noted that pigs may not be properly immunized if they are exposed to the organism whilst protected by an effective medication.

Collins et al[37] have specifically addressed this question. They found three types of responses following challenge of susceptible 4-week-old pigs with LI, in the presence of various medications and dosages. In this study, pigs were medicated in the feed starting 4 days before they were challenged, and up to an unspecified period of time afterwards. Oxytetracycline at 300 and 600 ppm, and chlortetracycline at 400 ppm completely protected pigs against the first dose of LI, but these pigs were fully susceptible when challenged again after cessation of medication.

Tylosin at 50 ppm and oxytetracycline at 50 and 100 ppm did not protect the pigs against the first experimental dose of LI and all became infected with LI. These pigs shed LI in their feces and developed a serological response to it. The clinical signs were less severe than non medicated pigs, and all pigs were immune to re-infection with LI. Tylosin at 100 ppm did not prevent every pig from becoming infected with LI. Fecal shedding and the development of an immune response were delayed in the pigs that had become infected, compared with non medicated pigs. Pigs that had become infected with LI following primary inoculation were immune to re-infection. Those that had been protected by the medication post primary inoculation (i.e had not shed LI in their feces) were not immune to LI, and thus susceptible to infection following the second inoculation.

This tends to indicate that if a medication prevents infection with LI to the point that shedding of the organism is prevented, the animal is likely to be susceptible if re-exposed. With this in mind, the authors suggested that the most successful strategies to induce the development of immunity to LI were those that allowed subclinical infection of pigs that were continuously medicated with low levels of antibiotics. They reminded that the level of antibiotic medication necessary to allow subclinical infection and immunity may depend on the level of exposure of pigs to LI.

Ileitis appears to be a dose-dependent condition. Collins et al[37] infected pigs with 2×10^3, 2×10^5, 2×10^7 or 2×10^{10} organisms. Another group was not infected and served as control. Pigs that received the two lowest doses did not show any clinical signs, and had no reduction in growth rate. Those that received 10^7 organisms showed mild clinical signs, but no reduction in growth rate. Finally, the pigs that received the highest dose showed severe clinical signs, a marked reduction in growth rate and had to be treated. Thus the infectious dose appears to have an impact on the severity of clinical signs and reduction in weight gain observed.

Pozo et al[39] infected piglets from three different sows, at different times. Sows 1 and 2

came from a herd considered to be endemically infected with LI, but were seronegative to an IFA test on day 0 of the experiment. Sow 3 was from a herd with no previous history of proliferative enteropathy, but had a strong positive result on the IFA test on day 0. It was thought that this sow had recently been infected with LI. Piglets from all three sows that were weaned at the time of infection showed clinical signs.

Piglets from sows 1 and 2 that were still suckling at the time of infection developed relatively mild clinical signs of the disease and did shed organisms in their feces. The piglets of sow 3 that were still suckling showed no signs of infection. The ingestion of milk from sow 3 appeared to prevent LI infection in her unweaned, inoculated piglets. However, when these pigs from sow 3 were re-inoculated 4 weeks after ingestion of milk had ceased, they were susceptible to infection. Although this is a very small experiment, it does suggest that protection against LI in young pigs could be mediated primarily by IgA antibodies in sows' milk, rather than antibodies derived from colostrum.

In another experiment that looked at maternal immunity and its possible interference with vaccination, Kroll et al[40] reported that pigs born from sows that had been vaccinated three times before farrowing were partially protected, when challenged at 6 weeks of age, compared to pigs from non vaccinated sows. This would seem to indicate that maternal immunity may not be limited to antibodies present in the milk. In the same experiment, pigs born from vaccinated sows, vaccinated at 3 weeks of age and challenged at 6 weeks of age were protected. The protection was not as good numerically as in pigs from non vaccinated sows, but there were no statistical differences. Although this is an area where more work may be needed, the experiment suggests that young vaccinated pigs born from presumably immune sows were protected against challenge. It should be noted here that the pigs were vaccinated after weaning, so not while they were consuming milk from their mothers.

A modified live virus vaccine has been available in North America for a few years, and is presently being launched in several European countries. The vaccine is administered orally, usually in the water. Since it is a live vaccine there are precautions that have to be taken when using it. For example a period of at least 7 days without antibiotics, and if possible more, is recommended when it is used. So far my personal experience with this vaccine has been very positive.

Different products used in swine have been reported to have efficacy against LI. These include tiamulin, lincomycin, oxytetracycline, chlortetracycline and tylosin. In Canada, only tylosin has so far been licensed as an aid in the prevention of problems associated with LI. Because the PHE form of the disease can kill animals so quickly, affected animals should be treated with effective injectable antibiotics immediately at the onset of clinical signs and water medication should, at least initially, be favoured over feed medication in severe cases where a group treatment appears justified. In Canada, it is becoming increasingly difficult to get consistent results in the treatment of PHE. Levels of certain antibiotics like tylosin and lincomycin that appeared to be consistently effective years ago do not always provide adequate protection today.

In a study conducted by Stege et al[41] coarse ground non pelleted feed was shown to reduce the prevalence of LI. More recently Johansen et al[42] reported that home mixed feed reduced the number of antibiotic treatments for diarrhea thought to be associated with LI and the number of days with diarrhea in the grower-finisher unit. However, home mixed feed also had a negative impact on performance, mainly on feed conversion, when compared to pelleted feed. Boesen et al[43] have reported the influence of diet on LI colonization in pigs upon experimental challenge. Five diets were evaluated: a standard diet (fine ground and pelleted), the standard diet fed as fermented liquid feed, the standard diet with 1.8% formic acid added, the standard diet with 2.4% lactic acid added and the standard diet fed coarse ground. The fermented liquid diet delayed the excretion of LI. Pigs fed the diet supplemented with lactic acid had limited pathological lesions when the intestines were examined four weeks after inoculation. Feeding coarse ground non pelleted feed, which mimicked a home mixed diet, did not reduce the infection with LI, compared with the fine ground and pelleted diet. Again it seems difficult to reach a consensus on the benefits of certain diets on this disease.

There are a few reports of small herds where eradication programs have seemingly and at least temporarily succeeded, so it appears that it could be possible to achieve.[44,45] Eradication of LI was attempted in two small herds of 35 sows with a history of medication, diarrhea and poor growth rate in weaned and growing pigs[44]. A program somewhat similar to the one used for eradication of *Mycoplasma hyopneumoniae* was implemented. Except for suckling piglets, all animals less than 10 months old were removed from the farm, and the sow herd was medicated with tiamulin in the drinking water for three weeks at a dosage of 60 ppm. Suckling piglets born during the medication period were treated three times with injectable tiamulin, at a dosage of 15 mg/kg. Fecal samples, which were positive before the eradication program, were negative afterwards. No clinical signs were noted in the two herds for 20 months following institution of the program, and no anti diarrheic antibiotics were used. Eradication of LI was attempted in a newly established Danish herd with no farrowings yet.[45] This herd was populated from another herd that just had a verified case of PHE (6 weeks before). A 14 day medication program with tylosin in the water, coupled with a disinfection program, was implemented. No clinical signs were noted afterwards and PCR tests on feces were negative. However, in a larger study where it was attempted in 9 herds, 7 of them were infected again by 15 to 22 months after the eradication program.[46] Recently Nielsen[47] described a program where three farms were depopulated, cleaned, disinfected, left empty for 2-3 months and repopulated with animals coming from LI positive herds. All new animals entering the farm were medicated with tiamulin for two periods of 14 days, separated by movement and washing of the animals. The antibiotic was added to the feed and the dosage was respectively of 8 mg/kg and 4 mg/kg for the first and second period respectively. The program was successful in the three herds. Because many herds with an excellent biosecurity program become infected with this organism by means that are not identified, and because the re-infection rate in herds that have attempted an eradication program can be high, eradication of this organism from infected herds should be considered with caution. This would be true at least until we better understand the causes of these unexplained infections and re-infections.

Other conditions

There are other enteric conditions, or conditions with an enteric component, that can have a negative impact on the performances of growing-finishing pigs and/or their survival. These include colibacillosis, porcine circovirus associated disease (PCVAD), porcine intestinal distension syndrome (also called hemorrhagic bowel syndrome), intestinal torsion or volvulus, transmissible gastroenteritis, non specific colitis and parasites like *Ascaris suum* and *Trichuris suis*. These conditions will only be briefly discussed during the presentation.

Conclusion

The ultimate control program for any disease is not to have it. For some conditions like septicemic salmonellosis and swine dysentery, our experience in Canada suggests that it is feasible to virtually eliminate them from our list of concerns. For others, we will likely have to learn more about their epidemiology and all the means by which they actually get transmitted between farms, before we can reach that goal.

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