CONTROLLING E. COLI IN THE WEANED PIG

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ABSTRACT

Post weaning colibacillosis is a bacterial disease caused by certain types of Escherichia coli and primarily affects pigs in the period shortly after weaning. The diarrhea causes metabolic acidosis, electrolyte imbalance and dehydration which leads to increased death loss and reduced production efficiency.

Economic losses are attributed to mortality and treatment costs of individual animals. Mortality may reach as high as 7%. A balanced approach to control has involved the simultaneous implementation of multiple interventions. There is strong scientific evidence for the use of antimicrobials. There is however little scientific evidence to support other best management practices. Control strategies have relied on empirical observation in the field.

ETIOLOGY

Escherichia coli (E. coli) are a group of gram negative flagellated bacteria that normally reside and multiply in the intestinal tract of all animals. E. coli normally increase in numbers during passage from the stomach through the duodenum, jejunum and ileum of the small intestine with the greatest numbers in the ileum. Some strains are hemolytic. Most virulence factors including hemolysins, toxins and adhesins are plasmid mediated. These plasmids contain genetic coding for virulence factors and can be transferred from one E. coli to another. This transfer of genetics via plasmids allows for more rapid change in the virulence of strains of E. coli. Gene probes are used to determine the genetic makeup of E. coli with respect to virulence factors.

The Enterotoxigenic E. coli (ETEC) are strains of E. coli that have the genetic ability to produce one or more of three different types of enterotoxins. Enterotoxins are "exotoxins", which are secreted by the E. coli and work external to the E. coli by causing a combination of deleterious effects on the function of intestinal cells. STa enterotoxins are small, heat stable, non immunogenic toxins. STa is less active in pigs older than two weeks of age. STb enterotoxins are heat stable, poorly immunogenic toxins. STb toxin is relatively common and is found in 70% of ETEC isolates. LT enterotoxins are heat labile toxins. Serotyping of individual strains of E. coli is based on specific characteristics that relate to several categories of antigens that the E. coli possess. Serotyping is one of the best ways to characterize strains of E. coli with respect to their virulence traits. Only the disease causing strains are generally typeable. "O" antigens are the somatic or cell body antigens. "K" antigens are the capsular

or micro capsular antigens. "H" antigens are the flagellar antigens. "F" antigens are the fimbrial or pilus adhesin antigens. Many E. coli require colonization of mucus membranes to cause disease and as such these extra cellular proteinaceous appendages can be specific markers which can indicate disease causing ability. Examples of "F" antigen types involved in colibacillosis of pigs include the following: F4 (also called K88), F5 (also called K99), F6 (also called 987P), and F41. Three subgroups of K88 (K88ab, K88ac and K88ad) exist . An example of a problematic serotype of E. coli is O141:K91, which is F4 (K88) positive. This is a bacteria that possesses somatic antigen 141, capsular antigen 91 and fimbrial type 4 adhesive factor.

EPIDEMIOLOGY

Post weaning colibacillosis is a disease that may occur sporadically within an individual nursery. Sites, barns and rooms that are managed with either all-in all-out pig flow or continuous flow may be affected. Historically, F4 (K88) E. coli have been a common cause of preweaning piglet diarrhea. Vaccination of sows prior to farrowing has provided very effective passive lactational immunity to suckling pigs. Removal of the piglet from the sow at the time of weaning, removes this rich source of protective antibodies in milk. The pig very quickly becomes dependent on its' own active immunity for protection. An apparent increase in the number of cases of post weaning colibacillosis was noted in the fall/winter of 1997 in Ontario. A search of the files from the Animal Health Laboratory, University of Guelph, and from the Veterinary Laboratory Services Branch, OMAF, revealed the following information:

Table 1. E. coli prevalence in Ontario.

Year	1992	1993	1994	1995	1996	1997	1998
K88 Cases	28	52	47	41	47	114	221
# of Herds	27	48	41	40	46	103	187

The mean age of piglets affected in 1998 was 25.6 days as compared to the average of 13.6 days in 1995. This tends to substantiate the reports from producers that post weaning pigs are more often affected, than piglets prior to weaning. In some cases pigs are clinically normal at weaning and have been found dead on arrival at the nursery after periods of transit of approximately 24 hours.

The most commonly accepted theory explains the increased incidence of this disease is the spread of a specific O149:K91 F4(K88) E. coli that elaborates all three enterotoxins. This seems to be borne out clinically with most persistent problematic cases of E. coli involving this specific serotype.

PATHOGENESIS

In order for Enteropathogenic E. coli to cause disease they must enter the intestinal tract, proliferate and attach to the intestinal lining and secrete their enterotoxins. Enterotoxigenic E. coli cause disease by attaching to enterocytes lining the intestine by means of their fimbrial attachment adhesins. Once attached they begin to secrete one or more of the enterotoxins which then cause the crypt cells to secrete fluids and electrolytes into the lumen of the intestine. These enterotoxins may also impair the absorption of fluid and electrolytes from the intestinal lumen. Fluid loss may be so severe that 30 to 40% of the pigs body weight may be lost into the lumen of the intestine within hours. Dehydration, electrolyte imbalance and metabolic acidosis and death will follow.

CLINICAL SIGNS

Pigs may be found dead with no previous clinical signs. These pigs often have distended bellies and sunken eyes. In many cases where sudden death has been a problem the F18 or Gut Edema strains of E. coli have been suspected. The F18 or Verotoxic E. coli has not been found to be a significant contributing factor to the problem in Ontario with the exception of a few isolated cases. The F18 E. coli is however a more significant player in some other regions of Canada and the United States.

The diarrhea may be mild to profuse. The consistency of the scour may be pasty to watery. The colour of the scour may be white, brown, green or very clear. Pigs may appear emaciated or "bony". A sunken eyed appearance becomes more prominent as the scour progresses. The pigs become depressed. Vomiting may occur occasionally. The skin may take on a blue discolouration especially over the extremities such as the ears, nose, toes and belly. The skin may also take on the appearance of dry parchment as dehydration progresses. The anus or perineum may have a red irritated appearance due to the irritation caused by alkaline faeces. The severity of clinical signs depends on the pig's age, immune status and the virulence factors of the E. coli involved. Endotoxic shock may be seen in some cases as the E. coli invades the blood stream late in the course of the disease.

DIAGNOSIS

The diagnosis of post weaning colibacillosis is based on a review of herd history, clinical signs, gross post mortem findings and histological lesions. The E. coli is often isolated in large numbers and relatively pure cultures from affected animals. Once the E. coli is isolated virulence factors such as enterotoxins can be further identified by gene probe. Gross post mortems may reveal dehydration, a dilated stomach containing undigested feed, venous infarcts on the greater curvature of the stomach and dilation of the intestine with fluid. There may be congestion of the intestine and other visceral organs. The gut content may be blood tinged. If the intestinal content has a blood tinged appearance the term "Haemorrhagic Gastro Enteritis" is often used. The lungs almost always deflate normally. Histological or microscopic examination reveals large numbers of bacteria adhering to the lining of the

intestine, as well as vascular congestion and some haemorrhage. White blood cells can be seen migrating into the lamina propria and lumen of the intestine especially in the jejunum and ileum and occasionally the colon. Occasionally microthrombi or plugged up blood vessels are seen in other body organs.

TREATMENT, CONTROL AND PREVENTION

Treatment, control and prevention of post weaning colibacillosis caused by enterotoxigenic E. coli should include a review of the following points. It should be noted that there is little hard evidence regarding the effectiveness of many of the following interventions. This list is presented as a review of the current thought process.

Feed

Protein. The crude protein level must be adjusted for the age and weight of the pig as well as the average daily feed intake. Diets containing excess crude protein may contribute to E. coli scour because the excess crude protein acts as a food source for the E. coli thus enhancing its' growth. An increase in the amount of synthetic amino acids can help to reduce the amounts of crude protein required. Easily digestible proteins, such as those of animal origin, can be broken down and absorbed by the pig, making them less available to the bacteria.

Minerals. Calcium levels that are excessive have been suggested to act as a buffer which can then lead to an increase in gastrointestinal pH. This is especially true when limestone is used as a source of calcium and phosphorous. The recommended levels for calcium according to the National Research Council (NRC) is between 0.6% and 0.8%. Zinc oxide at levels of 1500 to 3000 ppm has been effective as an aid in the control for E. coli. In some countries the use of elevated levels of zinc in pig feeds is prohibited for environmental reasons. It is advisable to target the higher zinc levels during the problem period.

Fibre. Increasing the level of fibre has been reported to decrease the incidence of colibacillosis. Sugar beets, oats, alfalfa and barley are commonly suggested sources of fibre. By increasing the rate of passage in the small intestine there is reduced opportunity for proliferation and adherence of E. coli. Fermentation of fibre to short chain fatty acids will help to increase acidity. Non starch polysaccharides (NSP's) are the structural carbohydrates in cell walls and include the pectins, cellulose, and arabinose. They are soluble and gel forming when exposed to heat, which decreases the digestibility of other nutrients. This again will lead to increased food for E. coli growth. The goal here is to find the "optimum" amount of the "appropriate" fibre such that E. coli is better controlled with minimum negative effect on average daily feed intake, average daily gain, and feed efficiency. The use of enzymes to break down the fibre has been suggested. Growth rates may be reduced if the ration is so excessively bulky that the pig is unable to increase feed intake adequately to compensate for a lower energy density. Increased fibre may negatively affect pellet quality or flowability of feed through feed bins and feeders.

Acidifiers. Acidifiers can be added to feed to help reduce the stomach and intestinal pH. Organic acidifiers such as citric acid, sorbic acid, fumaric acid, lactic acid and formic acid can be added to the feed. Acid salts such as calcium formate and sodium formate can be used. Inorganic acids such as phosphoric acid and hydrochloric acid and sulphuric acid can be added to the feed but have not been as effective and may decrease performance significantly.

Feed Manufacturing. Heat treatment of feed such as pelleting, expansion or extrusion has been suggested to increase the proliferation of E. coli by increasing the amount of available nutrients required by the E. coli for growth. It has been suggested that in an increasing order of importance pelleting, expansion and extrusion will increase the relative risk of enterobacterial proliferation.

Feeder Management. It is essential that the newly weaned pig begin consuming feed as soon possible after weaning. Pigs that consume feed poorly for the first few days after weaning may have a tendency to engorge on feed at some later time when their appetite increases. A pig that can engorge or eat large meals will have more feed in their stomach than their stomach can adequately acidify. When the quantity of acid is not adequate, the pH of the stomach and intestinal contents will rise and create an environment more suitable for the growth of E. coli. In addition, lack of acid reduces the conversion of the digestive enzyme pepsinogen to pepsin. This leads to poorer protein digestibility and more food for E. coli growth. Once pigs are being fed ad libitum, self feeders should be adjusted so that 50% of the feed trough is visible. This means that the pigs are having to work at the agitators to get feed to drop into the trough from the hopper. This will force pigs to eat small meals frequently and reduce the opportunity for pigs to engorge.

Prebiotics. (eg. Mannin oligosaccharides or Biomass) have been suggested as a potential control. The proposed mode of action of prebiotics is to bind with E. coli and prevent their attachment to intestinal cells. In addition it has been suggested that prebiotics will stimulate the immune system. Most of the information that is available is based on in vitro experimentation.

Water

Acidity. E. coli grows best in an environment that is basic or a high pH. Maintaining a reduced water pH has been suggested as a means of control of the number of E. coli in the gastrointestinal tract. Citric acid, formic acid or proprionic acid can be added to water to maintain an acidic pH. The pH of farm water should normally be between 6.5 and 7.5. pH should be checked and adjusted on a daily basis to within a range of 5.5 to 6.0 depending on the severity of the scour problem. Water can be acidified from the time the pigs have entered the barn and continued throughout the nursery phase or pulsed on a strategic schedule depending on when the scour is most common.

Sanitation. Water sources and delivery systems may become contaminated with disease-causing organisms such as E. coli. Total coliforms and fecal coliforms in both the well and the water lines should be assessed at least yearly or when problems arise. This can be done through a Public Health Unit or a private lab.

Water Management. Pigs should have access to clean fresh water at all times. A water flow rate at the drinker of 0.5 litres per minute at 10 to 20 pounds pressure is recommended for the nursery.

Environment

Temperature. Chilling can contribute significantly to the increased prevalence of scour by increasing stress levels in affected pigs. Chilling has been observed to reduce the peristaltic activity in the gut of the neonatal pig, increasing the accumulation of E. coli and toxins in the intestine. Consider pre-warming the barn prior to pigs' arrival.

Manure Management. The incidence of post-weaning scour in general is greater in pigs housed on solid floors with no bedding and in pens where the flooring does not stay clean. This relationship is presumably due to an increase in the pathogen load within the pen.

Sanitation

Although the disease can be seen in brand new facilities that have never previously housed pigs it is still important to ensure that facilities are properly sanitized between batches, so as to reduce the pathogen challenge level. A review of washing procedures for removal of organic matter is important. The use of detergents for removal of fats and the use of biofilm removal products will allow the subsequent use of disinfectants to be optimized. Disinfectant selection, application and calibration should be reviewed.

Concurrent Disease

It is possible that some other concurrent disease may worsen the incidence and severity of colibacillosis. The control of internal parasites, external parasites and the presence of viral enteritis such as rotavirus or transmissible gastroenteritis (TGE) should be considered.

Hospital Pen/Recovery Pen

A scouring pig is a source of infection for pen mates and if removed promptly can reduce the spread of scour in the pen by reducing the challenge level to pen mates. In E. coli outbreaks involving a large proportion of the pigs, the use of sorting will have obvious limitations. Thin and dehydrated pigs that are slowly recovering from scour should be collected as a group to enable provision of extra nursing care.

Medication

Injectable, water and feed medications should be selected based on the sensitivity pattern of the Enteropathogenic E. coli isolates or based on response to treatment. Mass medication of affected or at risk pigs may make the problem worse if the disease causing E. coli is resistant as the medication only serves to reduce the populations of non disease causing bacteria allowing the disease causing strains to proliferate due to lack of competition. Electrolytes may be added to the water when pigs are scouring.

Vaccination

Passive Immunity. Colostral protection declines rapidly after birth and lactogenic induced immunity is no longer protective once the piglet stops suckling. Autogenous vaccines given to sows prior to farrowing may only help to reduce the prevalence of these organisms in the sow and subsequently the nursing pig population. Antibodies for E. coli, derived from the egg-yolks of hyper-immunized hens, can be added to nursery feeds to prevent the adhesion of the E. coli to receptors in the intestine. The inclusion of these antibodies has produced inconsistent results in the field. This may in part be due to differences in pathogen load seen on certain farms.

Active Immunity. Injectable intramuscular or subcutaneous vaccines can be designed for use in suckling or weaned piglets based on the enteropathogenic E. coli involved, but are probably only marginally useful. The main problem is the short time period between initial immunization and subsequent challenge post weaning. In addition, intramuscular administration of vaccines is not ideal for stimulating local gut immunity.

Oral vaccines made with a genetically engineered enteropathogenic E. coli is in the experimental stage. The vaccine contains a strain of non-scour causing E. coli designed to compete in the pigs stomach with scour causing E. coli. No antibiotic can be administered to orally inoculated pigs as this will kill the non-scour causing strain of E. coli and allow the scour causing E. coli to increase in numbers and cause scour. The oral vaccine is administered twice while pigs are nursing. The use of low virulence E. coli has been suggested where nonvirulent strains were unavailable. Oral vaccines using field strains of F4(K88) positive but non toxin producing E. coli have been reported to be successful in the USA and Canada. These E. coli are propagated and fed to pigs via the water on entry to the nursery in order to stimulate intestinal immunity prior to the onset of disease.

Competitive Inhibition

Probiotics have been suggested for use in control of post weaning colibacillosis. Probiotics may bind the E. coli or their binding site on the intestinal wall. Probiotics may compete with E. coli for nutrients. Some probiotics are capable of promoting acid production. Probiotic products can be added to feed or water. Heat treatment of feed by pelleting, extrusion or expansion must be evaluated as this processing may kill off the probiotics. Feed or water medication may also reduce the efficacy of the probiotic.

Biosecurity

Ensure that the breeding stock supplier has no history of post weaning colibacillosis. Review all elements of biosecurity that could be involved in entrance of E. coli.

Genetics

It is clear that there are some genotypes of pigs that do not possess the F4(K88) receptor site on their intestinal epithelial cells and as such these lines of pigs are resistant to certain E. coli, where the F4 attachment is a required virulence factor for the E. coli.

CONCLUSIONS

Escherichia coli continues to present challenges for swine producers in Ontario and across North America. It may be that the simultaneous use of a combination of interventions will be required in order to effect control.

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