PMWS-Related but Diet-Induced Colitis
“A Practical Understanding of Causation Factors Involved with Finish Floor Diarrhea and the Potential Relationship with PCV-II-PMWS”
Frank Marshall, DVM, Marshall Swine Health Services Ltd., Camrose, AB

Introduction
Altered intestinal function is an important and common clinical problem of growing pigs raised under intensive production practices world-wide.

With the inevitable progression towards non-use of antimicrobial agents for control of enteric bacterial diseases and growth promotion, it is critical that alternative strategies be investigated and implemented. The need for basic understanding of dietary management of pigs throughout the feeder period is essential to our goal of non-usage of antimicrobials. Dietary manipulation and management has great potential to control and influence the common gut diseases we observe today on our finish floors.

It is not uncommon to observe transient to persistent diarrhea on the finish floor within the first few weeks of placement, or in association with diet transitions. The effects can be dramatic in terms of poorer weight gains, increased variation in weight within the group, extended marketing period for the group, and a higher number of culls, mortality, and lightweight pigs.

Finish floor diarrhea can be a perplexing diagnostic challenge. The processes leading to diarrhea are multifactoral and typically involve a complex of:

i) Precipitating environmental factors, (Temperature fluctuations, drafts, inappropriate temperature)
ii) Feed (type, form, ingredients, particle size, method of delivery-feeder type) and Water related factors,
iii) The Genetic host-the pig, it’s acquired capabilities, it’s acquired natural bacteria and micro-flora, it’s target tissues involved, and it’s state of immune activation.
iv) Several infectious pathogenic disease agents that are potentially observed in western Canadian Swine herds and systems.

The process of delineating all of the above is a learning curve in itself! Parts of our industry would have you believe that all diarrheas are due to Ileitis! If this were true life on the finish floor would be easy…especially with the advent of an effective Ileitis vaccine, however it is the objective of this presentation to shed differential light on the processes involved on the finish floor diarrhea complex in western Canadian swine production.

Our Veterinary Pathology Laboratories now have extensive powerful diagnostic capabilities with the advent of PCR-tests (DNA detection mechanisms). These tests provide us with much insight into disease agent presence with the associated pathology. Never-the-less we are just starting to get a glimpse of what these disease complexes entail.

Our discussion will be centered on the issues surrounding enteritis, Ileitis, colitis, and entero-colitis, as observed on our typical finish floor operations. This is not meant to be a total treatise on the entire realm of possibility for causal components of diarrhea, but a practical discussion of what is observed within our finish floor systems, the diagnostic explanations, with a view towards development of a holistic approach to resolving the problem.

With the clinical signs of diarrhea the portion of the intestinal tract involved helps define the terminology involved. That is, disease affecting the small intestine generally is referred to as “enteritis”, likewise disease specifically affecting the ileum portion of the small intestine is called “Ileitis,” and disease processes involving just the colon are referred to as “colitis”. Disease processes involving both the small and large intestine are referred to as “entero-colitis”. It will become obvious that proper pathologic diagnosis is critical to the management of the “diarrhea complex”. When diagnostic laboratory investigations fail to identify the specific pathogen in these colitis diarrhea cases, the term “non-specific colitis” is used. These non-specific colitis cases will be accompanied by bacterial overgrowth-referred to as “dysbacteriosis”.

Some facts worthy of note include: That 75% of the colon has to be effected to observe clinical diarrhea. Retrospective analysis in the UK has revealed that over 92% of cases of “non-specific colitis” have been shown to have an infectious causal agent. Of that 92% a total of 52% were shown to be associated with B. pilosicoli. (Duhamel)

Understanding the Pathogenesis
The basic function of the small intestine is to help digest and absorb nutrients (carbohydrates-and their breakdown products, proteins, and minerals), whereas the colon’s function is to absorb water, electrolytes, and volatile fatty acids-products of fermentation.

The colon has the potential to function similar to a cow’s rumen if given the opportunity. This opportunity comes in the form of undigested or non-digestible material that is presented to the colon from the small intestine.

Products that are undigested and unabsorbed, or simply non-digestible can present themselves from several situations:

a) Feed intake of a highly digestible diet exceeds the small intestine’s ability to digest and absorb nutrients,
b) In various diseases of the small intestine that interfere with digestion and absorption. (for example; Rota virus, TGE, severe E. coli enterotoxaemia, soy protein hypersensitivity)
c) Un-digestible dietary ingredients present to the hind gut or colon. (for example; non-soluble fiber, non- starch mucopolysaccarides-NSP’s, lignins, and too coarse of a particle size)
In these instances the rapid fermentation that takes place produces a situation similar to “grain overload” observed in ruminants, that is “acidosis” of the hind gut or colon. This process will also allow normal and abnormal bacteria and flora to proliferate in the bowel where it is not normally supposed to be (dysbacteriosis). This inflammation is ultimately “colitis”. This will predispose the colon to potential pathogenic bacterial proliferation and consequent disease presentation. Simple “acidosis” of the colon will lead to diarrhea as the “mucosa” or lining of the colon is damaged.

Last but not least, we also have the pathogenic disease agents that will damage and alter colon function or small intestine digestive and absorptive capabilities. These include the spirochete diarrhea agents – *Brachyspira hyodysenteriae* (swine dysentery), *B. pilosicoli, B. innocens*, as well as *Lawsonia intracellularis* – the Ileitis organism, *Salmonella typhimurium*, k88+ *E. coli*, and in nursery situations Rota- virus and TGE.

**With regard to circio-viral (PCV-II) related disease (PMWS),** ultimately we observe significant immune stimulation of the gastro-intestinal tract for what-ever-the-reason. This “immune system activation” is understood to be the major precipitating factor required to allow the PCV-II to produce disease in the tissues involved. It has been our observation to typically see PMWS associated pathology, with these cases of enteritis and colitis, whether we have nutritional factors or actual pathogenic disease agents involved.

### Nursery Diarrhea

In the nursery we can observe all of the above described situations in cases of nursery diarrhea.

**Nutritional related digestive upsets (leading to diarrhea)** occur if weaned pigs are placed on inappropriate diet phases for the age and digestive capabilities of that pig. This will also occur if one moves too quickly through the phase-feeding nursery regimen. The phase fed regimen moves progressively from highly complex-whey based and very digestible diets to non-complex soy based rations. In this instance we can present inappropriate or non-digestible ingredients to the small intestine-which cannot be digested at this stage and are passed on to the colon where the acidosis/dysbacteriosis will occur. The ‘Soy-protein hypersensitivity model’ is also a good example where undigested material will be presented to the hind gut. In this instance the weaned pig will develop an allergic immune reaction to soy where the immune system sees the Soy protein as “foreign” and mounts an antibody response to it. This then leads to small intestinal damage, leading to malabsorption of digested ingredients, which then presents to the colon where acidosis and “dysbacteriosis” develops.

**Infectious Agents** potentially encountered typically include *E. coli, coccidiosis (rare) and Rota-virus involving the small intestine, and B. pilosicoli, Salmonella typhimurium, B. hyodysenteriae (Swine Dysentery*) involving the colon.

**PMWS** is currently being observed in circumstances of infectious or reputed non-infectious disease, wherein the consequent immune activation process is initiated. This obviously needs more intensive study and investigation to ultimately prove that even this non-infectious gut-related immune activation will lead to PMWS. It has certainly been proven that even Freund’s adjuvant (used to enhance

---

**Hit Your Al Target ... with the IMV Team**

**A Winning Combination**

For further information about any of IMV’s top quality products, please call Nancy or Bruce at: 1-800-265-4058.

Visit our On-line Swine Catalogue http://www.imvgencor.ca (Click on Porcine)
the immune response to a particular vaccine agent) will induce PMWS. It would seem logical that our above described situation would indeed fit with the PMWS activation theme.

**Finish Pig Diarrhea**

On the finish floor we typically will see both non-infectious and infectious related diarrhea. The non infectious diarrheas are potentially observed with pelleted diets, where digestive capabilities of the upper small intestine are exceeded, or non digestible ingesta—“NSP’s” are presented, or too coarse-non-digestible particles are presented to the hind gut. Infectious colitis agents that can be present or induced from the dysbacteriosis include: *B. pilosicoli*, and *B. hyodysenteriae* (rare), true “Ileitis”- *Lawsonia intracellularis*, and entero-colitis due to *Salmonella typhimurium*.

Again PMWS is observed here as well, wherein infectious or reputed non-infectious disease, produces the consequent immune activation process and consequent PMWS related disease.

**Intervention Strategies**

**Nursery and Finish floor Inclusive**

1. Establish a proper diagnostic profile of the disease processes involved.

2. Evaluation of Hygiene procedures between batches. Very important in circumstances involving “trackable” disease agents like salmonellae, ileitis and spirochete disease agents.

3. In the Nursery-usage of the Nursery Budget regimen as described below based on 16 day wean age (diet appropriateness will depend on wean-age). Wherein soy protein introduction is a slow progressive process, and diet composition is carefully evaluated to progress from very complex-whey based diets to soy based diets appropriate for the age and feed intakes of the pig.

4. Dietary Acidifiers, and stomach bypass acidifiers. These products will aid digestion in the weaned pig in particular and inhibit the growth of agents like *E. coli* (nursery) and salmonella (nursery and finish-floor) in the intestinal tract.

5. Mannin Oligosaccaride products – show good promise and general results particularly in the weaned pig, but response can be variable depending on the circumstances. These products bind potential disease agents and are thus passed through the intestinal tract.

6. In the nursery and sometimes early in the finisher phase where *E. coli* is the concern, usage of Zinc oxide or chelated Zinc products are excellent. It is hypothesized that Zinc will inhibit/interfere with the “attachment” of *E. coli* to the intestinal lining, and as well-effect the local immune response, thus preventing the disease process.

7. Usage of Ingredient Specific enzymes (wheat, barley) – to enhance upper gut digestibility and absorption, and thus reduce the amount of non-digestible ingredients presented to the colon. These have to be in the right proportion with the grains involved to be cost effective.

8. Ensure adequate age appropriate husbandry. That is the typical stressors and potential stressors encountered at weaning and placement on the finish floor. For example-inappropriate temperature, temperature fluctuations, and drafts.

9. Evaluate Feed Diet ingredients and composition to avoid NSP’s, as well as irritant-type ingredients appropriate for the stage and age of pig.

10. Evaluate Particle size average and range. Ideally we aim at 650-800 microns, any averages that are smaller or larger we will enhance the potential to provide the colon with undigested or non-digestible ingesta.

11. On the finish floor where pelleted diets are used, immediate usage of “Mash” diets or non-pelleted diets will lessen the impact of presentation of non-digestible or easily digested ingesta to the colon.

12. Where Pathogenic organisms are involved, specific medications can be used to ‘band aid’ the situation until combinations of the above intervention strategies can be implemented. This needs to be discussed with your swine veterinarian.

**Summary**

With finish floor diarrhea it is critical to establish a proper diagnostic profile to allow an adequate holistic approach to resolving the process. Typically, there is no single “magic bullet” to relieve this disease complex. Resolving this multifactoral disease complex requires good team work where good swine husbandry, veterinary diagnostics, and nutrition skills combine to provide the ultimate approach.

<table>
<thead>
<tr>
<th>Stage/Age of Production</th>
<th>Total intake</th>
<th>Total Lysine % in Ration</th>
<th>F/C</th>
<th>Total Gain kgs</th>
<th>Begin/end Weights</th>
</tr>
</thead>
<tbody>
<tr>
<td>16-21 days of age (diet is complex - whey based, minimal soy, high zinc, acidifiers)</td>
<td>0.8-1.0 kg</td>
<td>1.6%-1.7%</td>
<td>1.1</td>
<td>0.90</td>
<td>5.2+/6.1+ kgs</td>
</tr>
<tr>
<td>22-28 days of age (less complex, high zinc, acidifiers)</td>
<td>1.5 kg</td>
<td>1.45 %-1.5</td>
<td>1.2</td>
<td>1.25</td>
<td>6.1+/7.4+ kgs</td>
</tr>
<tr>
<td>29-40 days of age (acidifiers, &lt;less complex, +/- high zinc)</td>
<td>6.0 kg</td>
<td>1.3 %</td>
<td>1.3</td>
<td>5.0+</td>
<td>7.4+/12.0 +kgs</td>
</tr>
<tr>
<td>39-65 days of age (noncomplex)</td>
<td>22 kg</td>
<td>1.2 %</td>
<td>1.5</td>
<td>13+</td>
<td>12.0+/26+ kgs</td>
</tr>
</tbody>
</table>

Saskatchewan Pork Industry Symposium 2002 46