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Using molecular characterization and genotyping to control neonatal and postnatal diarrhea

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Neonatal and postnatal diarrhea represents the most significant challenge we face in keeping our pigs healthy and performing well during the first 5-6 weeks of their life. The challenge is difficult due to the many stressors that the pig encounters over this time period such as waning maternally derived antibodies, temperature changes, weaning and establishing a social hierarchy. All of these factors can depress the immune system to a point where pathogens can gain a foothold and create clinical signs of disease.

There are 4 primary enteric pathogens that we deal with during this time frame. They are *E. coli*, Rotavirus, Coccidiosis and *Clostridium perfringens* Type A. They usually are seen at specific periods of time during the first 5-6 weeks of the pig's life. The time frame in which they occur allows the farm staff to identify the causative agent and to treat each syndrome accordingly. **Table 1** lists the time that we most commonly diagnose each of these pathogens.

Molecular characterization of these pathogens has allowed us to gain a better understanding of how they affect our pigs. It also has aided us in developing more effective control strategies for each of the pathogens. The following is an example of how molecular characterization and genotyping is used to control these enteric pathogens.

E. coli

Most *E. coli* infections we have dealt with have been post-weaning colibacillosis. Frequently they are of the F4 (K88) fimbrial type. Unfortunately, most of the pathogenic strains of *E. coli* we have seen are resistant to most of the antibiotics we are allowed to use. This has led us to look for alternative solutions to post-weaning colibacillosis. The most successful approach we have found is using a toxin negative isolate of the same fimbrial type of *E. coli* that is causing the disease. In order to do this, we send in

E. coli isolates and use a multiplex PCR assay to determine which fimbrial and toxin types the isolate can express. Most commonly this is a toxin free isolate of F4 (K88) or F18. That isolate is then used as a "live vaccine" in order to develop a localized immune response. This way when the pig encounters an *E. coli* isolate (of that fimbrial type) that may produce toxins, attachment will be impeded and the isolate will not be allowed to liberate the toxins that cause diarrhea.

We have done some further characterization of *E. coli* isolates in choosing strains to include in sow vaccines in an effort to control K88 *E. coli* in farrowing and in the early post-weaning period. We had a 6000 sow farm where the piglets were being inoculated with a live non-toxigenic strain of K88 *E. coli* at 10 days of age. We were still seeing pathogenic K88 *E. coli* at 3-4 weeks post-weaning with a K88 strain that was able to liberate LT (heat labile toxin) and STb (stable toxin b) causing diarrhea and acute deaths.

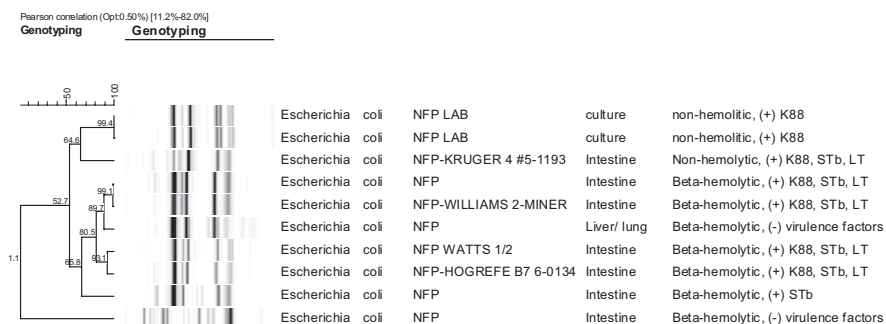
We sent all the *E. coli* isolates that we had isolated from the sow farm and flow to Dr. Simone Oliveira. Dr. Oliveira genotyped these isolates and compared their genomic fingerprints in a dendrogram (see **Figure 1**). We included our two K88 *E. coli* (non beta-hemolytic) non-toxigenic strains that we were using as our live "vaccine" that was being given to the piglets at 10 days of age. In the dendrogram analysis, we found a great amount of variation among the *E. coli* strains. We seemed to have 2 predominant K88 "clusters". One strain was a non-hemolytic K88 strain (NFP-Kruger 4). The remaining K88 isolates were beta-hemolytic and possessed the same toxin profile as the non-hemolytic strain.

After understanding some of the variation we had in K88 isolates, we decided to attack the problem at its source: shedding of the isolate from the sow. Our strategy was 2 fold:

Table 1: Age of onset of diarrhea of common neonatal/postnatal pathogens

Pathogen	Age of onset of diarrhea
<i>Clostridium perfringens</i> Type A	0-4 days of age
Rotavirus	3-7 days post-weaning
Coccidiosis	8 days of age or older in farrowing
<i>E. coli</i>	First 2-3 weeks post-weaning

Figure 1: Comparison of *E. coli* genomic fingerprints in a dendrogram: Pearson correlation (Opt:0.50%)[11.2%-82.0%]



- Replace the current commercial *E.coli* vaccine with an autogenous killed vaccine with 2 of these isolates to give to the sows twice at 4 and 2 weeks pre-farrow
- Grow up the pathogenic K88 isolates and feed it back to the sows at 5 and 3 weeks pre-farrow

Our goal was to decrease the number of K88 organisms shed to the piglets. We chose a non-hemolytic K88 strain (NFP-Kruger 4) and one from the cluster of hemolytic K88 isolates (NFP-Williams 2-Miner) for our killed autogenous vaccine for the sows. We also used the same isolates to create a live oral vaccine that was fed back to the sows. Since implementation of the program we have

dramatically reduced the number of cases of post-weaning K88 colibacillosis.

Molecular characterization of the pathogens that cause neonatal and postnatal diarrhea is another tool we can use to develop new and better strategies to reduce the incidence and severity of diarrhea in young pigs. We are currently performing characterization of Rotavirus and Clostridial isolates, but did not have results in time to be included in this paper.

