Maximizing piglet immunity through the sow

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Introduction

Maximizing piglet immunity at first glance seems the proper approach in pig production but this is not always a science based option or in some cases a good practice. Certainly different diseases require differing control strategies and management interventions. There are several approaches that have been historically effective when dealing with some of the more classical swine agents, often leading us to re-try or mimic them on the next pugnacious disease coming along. Although an intuitive approach is often prescribed, they are not always successful and in some situations may cause considerable harm. I will attempt to discuss those disease agents that respond well to maximization and stake out some detail on those diseases that may be enhanced by this process later in the paper.

It was the “Brits” that truly raised pig husbandry to the peak of perfection by the late 1960’s. Many British procedures associated with maximizing the survival of neonatal and the gestating fetal embryos through sow care goes to their credit. When we were averaging 10 to 12 pigs per sow per year weaned they were close to 20. Part of their secret was maximizing piglet health and immunity through good husbandry practices. This opened many eyes and it wasn’t long before those ideas and processes immigrated westerly across the Atlantic. It wasn’t just the procedures as many prolific genetic lines and genetic companies followed. In those days the US industry had few if any productivity benchmarks to boast about but we had good markets, excellent profit margins, and were generally buffered from global economics. We had conquered Hog Cholera; PRRSv and PCV2 were many years away and Pseudorabies just didn’t cause big problems for most of us. No one had even considered gasohol – corn was mostly raised for livestock feed. Few were concerned with export markets – we had cheap corn and soy protein along with diversified agriculture that kept us in business during off year pig markets. We simply got in or out of the pig business depending on market conditions. The pig was the mortgage lifter for the Midwestern and Eastern farmers. Life was good. With margins high and the British example as a guide we quickly headed down the productivity road which included greater management and care leading us to current industry standards and even structure. New investors took interest and both operation size and productivity came to be important determinants of success.

Historical methods of piglet immunity maximization

So what does all this have to do with maximizing piglet immunity (and productivity) through the sow? First of all sow care during gestation is essential for maximum lactation performance in farrowing. Colostral antibodies will not be in ample supply if the sow isn’t healthy, perfectly conditioned, not too old, and equipped with enough functional teats, etc. Piglet performance is prescribed by the amount of energy and protein the sow provides.
during lactation and the amount and quality of the start-up immunity provided in that colostrum. Colostrum not only provides initial energy and immunity but many other growth and hormonal factors that will influence the pig for the rest of its life. Healthy properly conditioned sows yield robust piglets. The overall beneficial effect of colostral intake by the piglet is recognized by the producer but it not just quantity, quality is also essential. Our job is to maximize both.

The British pig producers were especially good at this and although they did not understand the physiology their common sense approach to maximizing piglet immunity should not be forgotten. Unfortunately some of this has slipped through the cracks. Fecal and reproductive feedback (mummies) was the backbone of maximizing pre-farrowing sow and gilt immunity long before we had effective vaccines. This process was adopted by US producers soon after we brought farrowing indoors. Hank Harris and many other innovators quickly publicized the value of this procedure and it was widely accepted. Later it was used to eradicate TGE from individual sow herds and as protection against many of the indigenous potentially pathogenic enteric bacteria and viruses. Even prior to widespread feedback, E.M. Kohler at Ohio State University had developed a process that later became known among veterinarians as the “Kohler Vaccine”. This was a complex process of identifying specific strains of \textit{E.coli} that were involved in individual farm colibacillosis (baby pig scours); culturing and maintaining the isolate and growing lots of bacteria in milk to produce an oral sow vaccine. This was wonderfully effective but labor intensive and the whole mess made clinics and farms smell like septic tank stench. Many of our current ideas on maximizing initial piglet immunity goes back to the Kohler vaccine, fecal and mummy feedback, and the husbandry emphasis on sow and gilt care.

Effective \textit{E.coli} vaccines came on the market by 1980 and feedback rapidly lost favor especially in larger production units, eventually the process was only relegated to putting out outbreak fires. It was a dirty job. As discussed later, unfortunately these guiding principals of maximizing colostrum quality against indigenous enteric pathogens has either been abandoned (feedback) or no longer produce similar results today. The onset of PRRSv in the U.S. along with a gross lack of knowledge concerning its ecology led us to “hand wringing” over feedback and many veterinarians believed this process was associated repeated PRRSv outbreaks. My own experience was the opposite where small herds that continued feedback after PRRSv introduction quickly returned to negative status after 12 to 24 months.

\textbf{Techniques to maximize specific colostral antibody production}

Maximizing colostral intake is probably the most important production strategy in protecting the piglet from infection during the suckling period. Aside from bringing the sows into farrowing in ideal body condition she also must be ready and have the desire to eat once the piglets are born. She must enter the farrowing house in a healthy state. This requires quickly returning body stores after the previous litter, preventing over condition and slightly increasing feed intake the last couple of weeks before the next litter due date. It is critical to shorten parturition (labor) time, shorten birth to suckle times, and maximize early colostral intake\textsuperscript{ii}. These three are most important in maximized antibody transfer and subsequent protection. Sow appetite and ability to consume large quantities of feed during
lactation is also essential. Fineness of grind, multiple feeding times, pellet feed, comfortable temperatures, genetics and the chronic disease processes on a farm ultimately dictate piglet health and performance through a variety of mechanisms. Methods to maximize lactation feed intake will always affect litter quality, pigs weaned per sow, piglet health, and subsequent litter size.

As discussed, the Kohler vaccine and feedback have historically produced milk antibodies (IgA & IgG) that effectively protect suckling piglets against the indigenous enteric and systemic pathogens lurking on every farm. These techniques weren’t without compliance and delivery problems, creating dirty jobs that no one wanted. The advent of efficacious *E. coli* bacterins ushered in a new vaccine approach for the control of disease in the offspring. They were highly effective but abortions shortly after administration in a small percentage of sows slowed initial acceptance. This was quickly solved by competition and these vaccines became an industry standard. Maximizing piglet immunity with late gestation sow vaccine continues but success has been minimal beyond enteric protection. PRRSv vaccines, influenza vaccines, and others have been utilized in this manner but with minimal success.

By 1980 the interest and growing knowledge in piglet immunity maximization and disease elimination without depopulation made it possible to establish high health status herds without a far more costly caesarian derived approach. Tom Alexander and Keith Thornton of Cambridge University discovered the value of the medicated early weaning (MEW) process. This procedure relies on maximized colostral protection and early removal from the far breaking the transmission cycle of environmental pathogens to the piglet. Initially pigs were weaned at very young ages in batches to producing offspring free of *M. hyo*. Eventually other economically important diseases present in the sow farm of origin would fall to the elimination process. This became the first practical tool which effectively produced *Mycoplasma hyopneumoniae* free breeding stock. A few years later Hank Harris and Barry Wiseman discovered that pigs free of multiple agents could easily be produced in batches by the MEW process and at older ages. Over the years MEW has successfully eliminated *M. hyo*, TGE, *Actinobacillus pleuropneumonia*, Pseudorabies, and other agents from endemically infected sow herds. Marie Gramer and Bill Christianson extended this to PRRSv in stable but exposed sows. It also appears that PCV2 can be eliminated by the same process (Pat Halbur personal communication) and this may periodically occur in batches of replacement gilts destabilizing sow herds and leading to Porcine Circovirus Associated Disease (PCVAD) in growing pigs. In large systems feedback or serum inoculation may be important tools in the fight against this agent. This has not been studied or justified in the field. Even so, the benefit of continuous stabilization of breeding herds by homogenous exposure methods such as feedback has been lost over time.

These evolving discoveries utilizing immune maximization and segregated rearing of pigs pushed early adopters to experiment with all-in, all-out and later multi-site production. Several breeding stock companies took full advantage MEW and other similar techniques to clean up marginal health status. By understanding the power of passive antibody transfer from sow to offspring many new methods of disease control and
elimination have been developed. Unleashing through understanding and then manipulating this power may be the single greatest advancement in modern pig production.

Microbial agents and especially pathogens have a great innate ability to adapt and change over time. Antimicrobial resistance problems are a testament to that evolutionary potential. These microbes respond through a vast array of mechanisms and genetic diversity which creates an almost infinite selection gradient responding to immunity, production method, population size pig movement, vaccine, antibiotic, and the environmental melee. The effect of vaccines on highly changeable agents merely stimulates them to eventual adaptation and thus presents us with a limited time of effectiveness. This may be years or in the case of PRRSv weeks. Feedback and other continuous exposure methods don’t have this limitation since exposure and immunity move along the same continuum. I do not intend to insinuate that it can solve all problems associated with piglet immunity but if part of a carefully planned and managed process it is effective against E.coli, Clostridium, parvovirus, PCV2, ROTA virus, TGE virus, adenoviruses, Enteroviruses, Teschoviruses, and likely a host of other undiagnosed endemic agents.

Sow vaccines: effectiveness, value, and philosophy:

As discussed, E.coli vaccines have been an effective method of stimulating the sows’ immune response eliciting antibodies which protect the neonatal piglet. Their advent was hailed as a “godsend” by those of us making the Kohler vaccine or collecting feedback scouring in the farrowing crates. This tool does not cover all the E.coli pathogens we encounter and over time has in some cases lost effectiveness. Even so, these vaccines are the backbone of maximizing piglet immunity alongside sow care and gilt development. Aside from E.coli there are a host of other vaccines that are recommended or labeled for use in sows for the purpose of enhancing early piglet immunity. Influenza and PRRSv vaccines quickly come to mind, both with United States Department of Agriculture – Center for Veterinary Biologics (USDA-CVB) approval for use in sows with an assumption and purpose of protecting the piglets. Both of these vaccines have drawbacks in the immune enhancement arena. Antigenic shift and drift are terms associated with human influenza virus strain evolution but the same processes that produces gradual and rapid genetic changes in influenza virus also occurs with PRRSv. One of the big differences between these viruses is host range. PRRSv is only known to infect pigs while influenza viruses may infect several species including pigs, people and birds. Birds are the primary reservoir for the virus and viral genetic information but transfer of these genetic information sequences between virus sub-types is also active in humans and pigs. It appears the rapid genetic changes we see in pig influenza viruses are the result of human virus introduction into our swine populations. Avian virus sequences also cross species and end up in pig flu strains. These may spill directly from birds but more often come from human circulating strains after those strains have experienced genetic sharing with avian flu isolates. All this is not clearly defined and it is likely that multiple pathways of viral change exist for influenza viruses. PRRSv also has great potential for change but this is apparently accomplished by genetic drift in populations of growing pigs and frequent lateral transmission (area spread) to new susceptible pig populations. Re-assortment isolates are reported and certainly occur but this may not be the primary route of evolution for the PRRSv. The pig is the only reservoir for the virus and it appears to be growing pigs especially finishing pigs where
change rapidly occurs. Finishing floor and nursery isolates often have considerable genetic diversity even in the same barn population based on Open Reading Frame (ORF) 5 sequencing. This diversity likely extends to other genetic components of the virus but this is only rarely determined except by a few research groups.

These and other issues lead us to question vaccination value for these agents in adult herds unless vaccine strains are matched to circulating strains. This is possible with influenza but less appropriate for PRRSv. Cross protection is a limiting factor with both. Cross protection is possible with modified live influenza (MLV) vaccines but not with the killed vaccines we have today unless they happen to be antigenically matched. We only have commercial MLV PRRS vaccines but cross protection does not appear to be significant when used in the field. This may be due to a number of possible scenarios but immune trickery by the virus seems most likely. It is unlikely that there will be much opportunity for effective traditional vaccines for either of these agents since natural protection against re-infection to antigenically new circulating isolates does not occur even though both viruses produce no carriers and homologous immunity is apparently complete. Since the viruses have ability to constantly evolve in ways that sustain them in susceptible and previously exposed populations, it is no great wonder that vaccine has little opportunity to produce protective immunity in the suckling pig beyond early homologous exposure. There is even less opportunity for protection to carry over into or through the nursery stage. Area spread of genetically diverse strains of these two viruses further prevent long term or even short term effectiveness. Influenza appears to be most stable with new strains circulating over winter in many cases. PRRSv also has a winter circulation predilection but both viruses circulate year round in large production systems.

Our field experience using these vaccine products for sow based piglet immunity maximization has generally failed. There is some evidence that Influenza disease may be exacerbated by the presence of weakly or non-neutralizing antibodies in piglets when exposed to an antigenically heterologous virus. This has been referred to as “original antigenic sin” in human papers. The immune failure is thought to be B-cell mediated through immune memory to the previous exposed strain when confronted with a new influenza virus. In this scenario, antibodies against the old strain are thrown into production ignoring the new antigenic type giving the infecting virus extra time to replicate and do harm. This has been demonstrated in vaccinated CDCD pigs when challenged with heterologous virus, increasing pathology over the non-vaccinated but challenged pigs.

**Piglet Exposure Tools**

Other innovative methods that stimulate piglet immunity while suckling have evolved. Most successful methods of piglet immune stimulation while in the farrowing room comes from live exposure to bacterial inoculums. Pathogenic farms strains of *Haemophilus parasuis* (Hps) administrated intra-nasally shortly after birth has successfully reduced Hps mortality in large productions systems when piglets from multiple “breed to wean barns/sites” are commingled into nursery housing. Post weaning E. coli diarrhea caused by K-88 and edema disease due to F-18 pilus antigenic types has also been controlled by early inoculation with non-pathogenic (pilus +, toxin-) strains. We now have effective commercial vaccines that to both of these E.coli problems.
Early exposure to other potentially pathogenic colonizing bacteria such as *Streptococcus suis* (*S.suis*) is an important mechanism preventing pathology while farm specific maternal antibodies are still protective and allowing a “golden period” where the piglets own immune shield rapidly develops. This normal process breaks down in large production systems where antigenically different strains of Hps and *S.suis* reside in different breeding herd populations. Mixing of piglets results in a mixing of bacterial antigenic subtypes leading to disease outbreaks. Immunosuppressive agents (PRRSv & PCV2) exacerbate this issue.

It appears that first parity sows (P-1) may do a better job of colonizing their piglets to some agents but prevent colonization by Hps. This is debated but field experience collaborates the theory. It is also believed that P-1 sows don’t deliver a whole package of neutralizing antibodies due to insufficient exposure to all farm pathogens or immune immaturity. This becomes a serious concern in parity segregated systems and large breeding sites. Feedback in not a good option since farm to farm movement of feces and mummies/placentas is risky due to PRRSv variation. Gilt exposure often depends on vaccination programs which can’t complement all farm potential pathogens. It is my prejudice that parity segregation creates opportunities where system health can gradually be improved by improving the health of the gilt supply farms. As an example, *Mycoplasma hyopneumoniae* can easily be eradicated in these systems by supplying negative gilts to the P-1 farms. PRRSv has also been eliminated in some parity segregated systems simply by creative use of PRRS negative gilt placement into stable sow farms.

Serum inoculation of breeding herds in the early stages of a PRRSv outbreak appears to reduce the length of time clinical disease remains in the breeding herd and the growing pigs. This is thought to be a result of maximizing population immunity and concurrently eliminating the virus from each animal of the population shortening the duration of natural exposure and inefficient contagiousness. This maximizes whole herd colostral immunity at some future time preventing the so called “virus trickle” that appears to occur in un-assisted outbreaks. Even so this is not based on sound science and use of serum can lead to unexpected consequences. Slight coding differences in ORF-5 sequences don’t predict cross protection and disastrous results have occurred associated with this procedure. Isolates must come from aborted fetuses during the early stages of a PRRSv outbreak and never from growing piglet populations when intended for use in adults. Likewise, serum should come from recently aborted sows not nursery pigs.

**Conclusion**

Over the past century much has been learned about the power of maximizing piglet immunity though the sow. Colostral antibodies protect pigs through early exposure periods to many of the economically important disease agents living in our farms. Exclusion of most of these is not possible so we must rely on functional immune responses both in the newborn piglet and by the sow prior to farrowing. Antibodies are not the only factors that are passed from sow to piglet via colostrum. Other factors that promote growth and digestion of nutrients have been proposed and are likely. In essence it is extremely important that we maximize early (immediate) suckling and shorten total delivery time.
Many methods such as farrowing induction and split suckling have been promoted by experts in our industry. These approaches need more science but appear to be effective in assuring all pigs receive a minimum effective dose. This may not be the final answer since there has been little scientific exploration and available grant monies in this area.

The tools we have today are effective but insufficient overall. Feedback is a forgotten art and difficult to apply in large systems especially those that are parity segregated. We have made great strides in our understanding but there is still a lot more that should be determined in this arena before we have answers to most of our growing pig health problems. There is a great need for new efficacious technologies both vaccines and products.