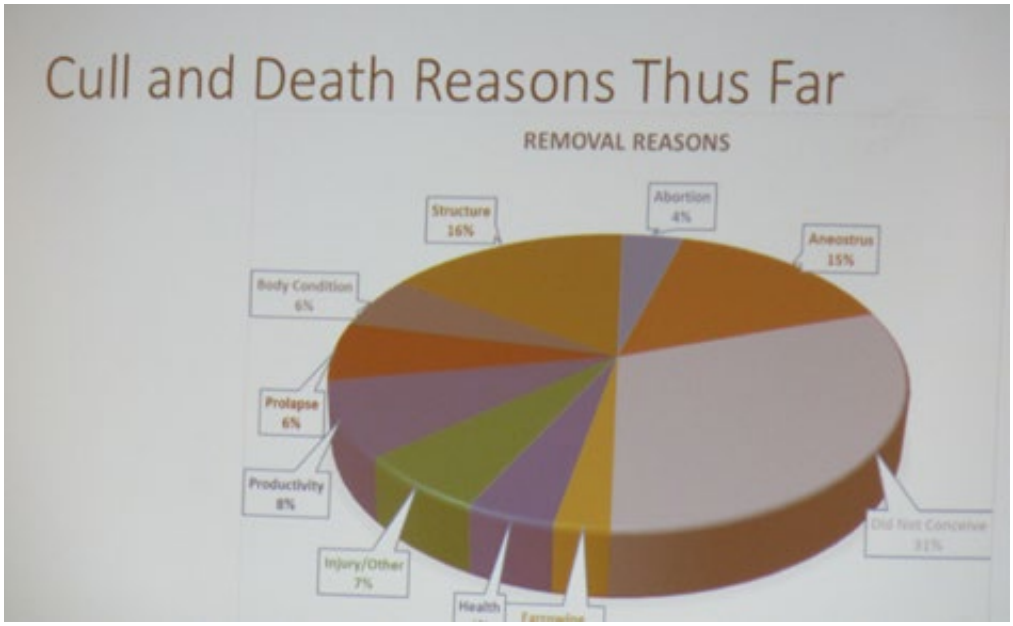
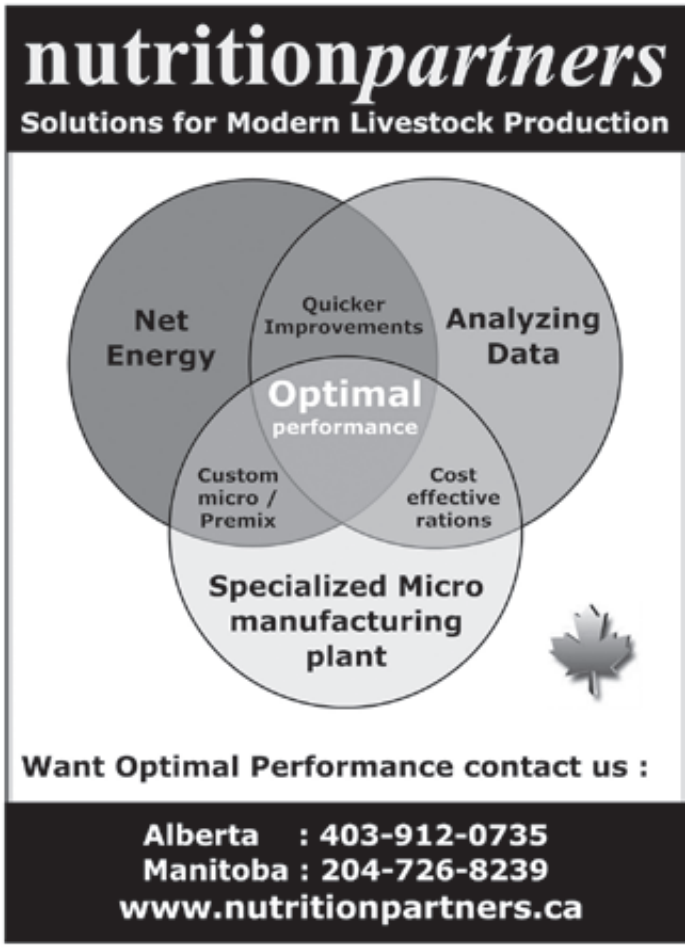
the Genomic Estimated Breeding Values for improved pig health and resilience, as well as optimize commercial management strategies for reducing the impact of disease on resilient pigs.

4. Socio-Economic Analysis: The purpose of this analysis is to identify and measure the social value of using genomics in breeding for disease resilience, for Canadian and global markets. This will help determine the best approach to pig health optimization: the development of vaccines, more specific antibiotics or breeding for disease resilience.



Industry implications

Ultimately, researchers hope to improve the end-user's ability to select, feed and use microbial management tools for the optimal immune response of pigs. This should help Canadian producers meet rising global pork demands by improving health and productivity while reducing the use of antimicrobials in pork production. Now THAT'S a good icebreaker.



Part three: Impacts of Moving "Clean" Gilts into "Health Challenged" Commercial Sow Farms: Gilt Acclimation Project

Benny Mote

Plastow's work and the latest Genome Canada project outlined in Part II have demonstrated that genetic variations in animals can produce differing levels of resistance to infectious diseases. However few studies have been undertaken combining multiple diseases. Led by Dr. Benny Mote, assistant professor, swine extension specialist at the University of Nebraska-Lincoln, this study followed over 3,000 gilts as they transitioned from high health multiplier farms to cooperating commercial farms where the females would encounter a number of natural disease loads. Genome-wide association studies identified regions of the swine genome controlling the most commercially relevant diseases.

CONTINUED ON PAGE 64

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Background

In the true gilt acclimation part of the study, data was collected on 3,033 females from seven different genetic companies. Gilts were weighed and had their blood drawn before being followed to 23 different commercial farms that had at least one commercially relevant disease, typically PRRS. Each farm applied its standard gilt acclimation protocol, which varied from direct entry to offsite quarantine. Blood was drawn again within 30 days of the gilt's first and second litters, and reproduction, vaccination and treatment records were kept out to parity four.

Outcome

The early data from the outbreak farms led to exciting results. For example, it was shown that two regions of the swine genome on chromosome seven explained 40 per cent of the genetic variation with the PRRS S/P ratios. Not only were those ratios inheritable, but they correlated strongly with traits such as number of mummies and number born alive while under a PRRS challenge. It may be possible to use the knowledge gained from the S/P ratios of commercial animals to help drive selection in nucleus populations that typically never see these challenges, thereby minimizing the impact of diseases on commercial producers.

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Part four: Genetic Improvement of Sow and Gilt Reproductive Performance via PRRS Immunity

This work presented some results from the Gilt Acclimation Project described in Part III. One of the key findings was that S/P ratio has the potential to be used as an indicator trait to select for improved reproductive performance during PRRS infection. Also, while S/P ratio can be predicted with moderate accuracy when two regions of chromosome 7 are used, the accuracy of prediction of reproductive performance during PRRS infection was higher when the other genomic regions were included. Altogether, the results indicate that response to PRRS in gilts and sows is heritable and that immune and reproductive performance might be improved using genomics.

New data is still being generated in this project which will increase our understanding of the genetics of immunity and reproductive performance in replacement gilts and sows.

Part five: Genetics of Host Response to PRRS in Growing Pigs

To capitalize on advances in genomics technology to study the role of host genetics in PRRS infection and develop tools to select pigs for improved resistance or reduced susceptibility, the PRRS Host Genetics Consortium (PHGC) was initiated in 2008. It involved experimental challenge of groups of 200 commercial nursery pigs at experimental facilities with a specific strain of PRRS. The purpose was to determine the genetic basis of differences in response to PRRS between pigs from the same breed or line, thus enabling breeders to improve their lines for PRRS, along with other traits.

Findings

Host response of nursery pigs to PRRS infection was found to have a sizable genetic component under controlled experimental challenge studies. In particular, a major gene for host response to PRRS was identified on SSC4, with the putative causative mutation in a gene that is involved in innate immune response. The WUR SNP (Single Nucleotide

Polymorphism) in this region could be used to select for pigs whose growth rate is less affected by PRRS. While a lot of study still needs to be done, researchers are beginning to unravel the genetic basis of host response to PRRS. Ultimately, this should provide the ability to select pigs that are less susceptible to PRRS infection and its effects on performance. As well, insight into host response to PRRS infection could aid in development of more effective vaccines and therapeutics.

Part six: PRRSV and the Pregnant Female

One of the most devastating effects of the PRRS virus is reproductive failure in pregnant females. While PRRS can cause embryonic death in early gestation, it most commonly manifests in late gestation as abortions, early farrowing, fetal death and the birth of weak, congenitally infected piglets. Although transplacental PRRS infection occurs mainly in late gestation, the exact mechanism by which it transmits to the dam and her fetuses has yet to be determined.

The Pregnant Gilt Model (PGM1) was a large-scale, multidisciplinary project led by University of Saskatchewan researchers in 2012. The goal was to improve understanding of the mechanisms associated with transplacental PRRS infection and identify phenotypic and genotypic biomarkers of resilience to reproductive PRRS.

Some of the key project findings were as follows:

- Larger fetuses are more susceptible to transplacental PRRS infection.
- The presence of fetal and umbilical lesions increased the likelihood of fetal meconium staining, the earliest stage of fetal compromise associated with PRRS.
- Events occurring in the fetus are essential in the pathogenesis of reproductive PRRS. Therefore, the detection of resilient phenotypes should focus on transplacental transmission and fetal immune responses.
- Progression of the immune response to PRRS appears to be faster in resistant pigs than in susceptible pigs, which may contribute to lower levels of fetal pathology in resistant pigs.
- There is new evidence for the genetic basis of fetal response to PRRS, which may ultimately lead to alternative control strategies to reduce the impact of reproductive PRRS.

The PGM1 was the largest study of reproductive PRRS to date. The PGM2 will build on that to validate the genomic markers associated with viral load and fetal autolysis. It will also investigate specific mechanisms underlying transplacental transmission and host responses occurring in the most susceptible fetuses. As PRRS is one of the swine industry's costliest diseases and lacks a completely effective vaccine, exploring other means of control and prevention, such as identifying highly resilient pigs, is more important than ever. ■