Understanding the Genetic Mechanisms Controlling Sow Longevity

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Introduction and Background

Sow longevity, or more accurately called sow productive life (SPL), has been a major focus point of concern in the swine industry for some time now. In addition to the animal and worker welfare concerns, industry leaders are realizing the substantial economical losses that occur when a farm has poor SPL in its commercial females. The economic benefits for increasing the average age of the females of reproductive age in the herd by one tenth of a parity are an additional $0.23 and $0.13 for every hog marketed in a farrow-to-finish and farrow-to-wean operations, respectively. Taken collectively, this sums up to over 15 million dollars in increased income in the US swine industry alone. Therefore even minor improvements to SPL can make an enormous impact of the financial bottom line for swine operations.

Previous to this research, no studies have focused on the genes that play a pivotal role in SPL. Many researchers using model organisms such as nematodes, flies, and mice have shown a clear network of genes that play an integral role in increasing simple lifespan of these animals, primarily through a reduction in caloric intake or changes in the genes that mimic caloric restriction. These observations also hold true for human longevity studies. It is therefore our working hypothesis that these same genes may be involved in SPL. We realize that SPL and simple lifespan are not completely correlated. We are fully aware that in swine production thinner sows are typically culled earlier because they typically don't have the body reserves to rely on during times of extreme nutrient requirements. We expect (and have encountered) alleles in these same genes from the model organisms that are associated with higher backfat to actually be the beneficial alleles for SPL. Our research has shown that there are several genes that can be selected for to improve the SPL of females at the commercial level.

Materials and Methods

For this research project we have used four distinct populations. The first population consisted of approximately1000 females where half of them had less than four parities and the remaining half consisted of sows that had greater than six parities. No other information was available on this data set. The second population was comprised of nearly 200 sires where reproductive data was collected on their daughters. The third population contained about1000 nucleus females where only reproduction data were obtained. The final population is the most inclusive and serves as our validation population. This population consists of 2,000 commercial females that were sampled from three farms in October of 2005. We collected tissue for DNA isolation using ear tags purchased from IDnostics that allow for simultaneous identification and tissue collection with minimum chance of misidentification. Half of the females were gilts that had just entered the production system and the remaining half were older sows that had a minimum of five parities. Equal numbers of gilts and old sows were sampled from each farm. PigChamp^{TM} records are used for information regarding current status of the females, reproduction records, culling date, and culling reason.

To date, we have started investigating the effects of seventeen genes on part or all four populations. These genes are either involved in the insulin pathway and/or play integral roles in reproductive pathways. Eight genes have been dropped due to insufficient association with SPL, two genes are currently undergoing analysis using the fourth populations, and seven genes have undergone complete analysis in all populations. The genes that have undergone complete analysis are *IGFBP1, IGFBP2, IGFBP3, IGFBP5, COX2, CPT1A,* and *SLC22A5*.

To test the association of the gene with the number of parities a sow has, we used a Chi-squared test between the genotypic frequencies between the old and young groups of population 4. To test the association of the gene with reproduction data, we used SAS Mixed Models fitting parity or age group, line and farm fixed effects.

Results and Discussion

The genetic markers were screened in populations 1 and 2 with only those showing at least a tendency for association with a component of SPL being validated in the forth population. The gene markers for *IGFBP1*, *IGFBP3*, *SLC22A5,* and *CPT1A* were all significantly associated ($P < 0.05$) for remaining in the herd until 5 parities (see Figures 1 thru 4). Additionally for *IGFBP1,* the same genotype favored for greater SPL showed a tendency ($P < 0.1$) for the number of pigs born alive throughout a sow's productive life (see Figure 5). Therefore a sow with the beneficial genotype for *IGFBP*1 will not only have a greater probability of staying in the herd until parity 5, but will also produce an additional 1.5 pigs while doing so. *IGFBP2* was significantly associated with the number of pigs born alive over the sows' lifetime

(see Figure 6). *SLC22A5* was associated with the number of mummies during the sow's lifetime. CPT1A was significantly associated ($P < .05$) with the number of pigs born alive in parities three and greater with effects as large as 0.7 of a live pig per litter. These results are evidence that there are genes causing variation in sow productive life and give promise to the use of marker assisted selection to improve sow productive life.

FIGURE 1

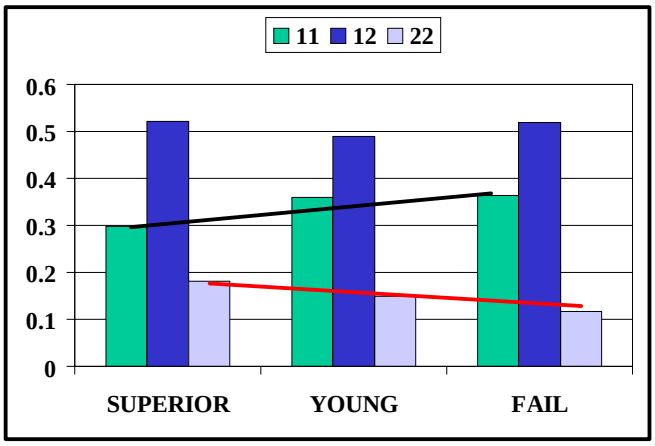

FIGURE 2

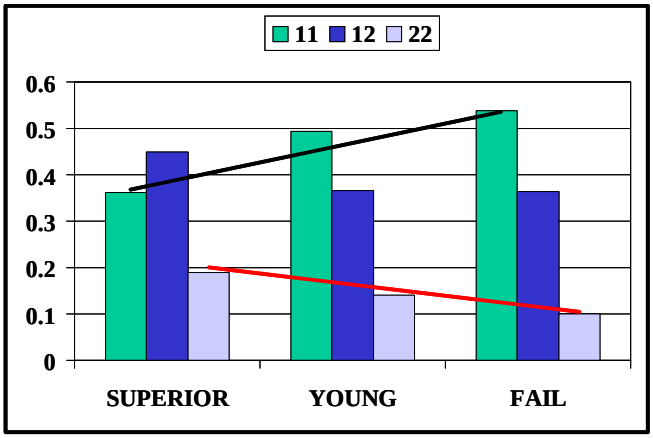

FIGURE 3

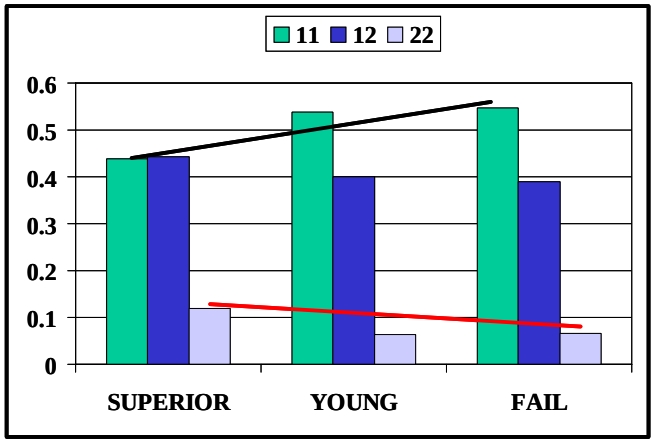

FIGURE 4

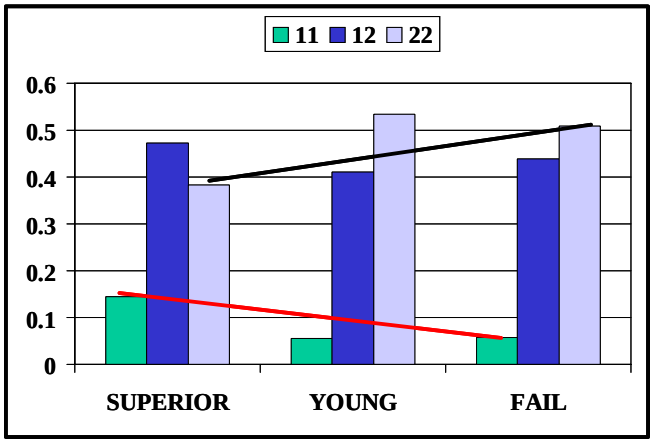

FIGURE 5

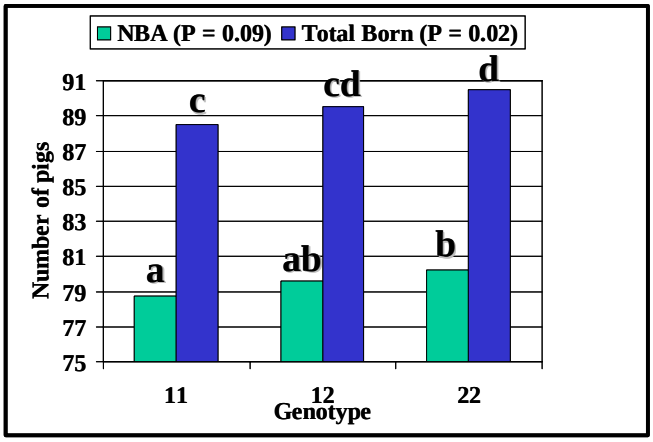

FIGURE 6