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Swine influenza case presentations

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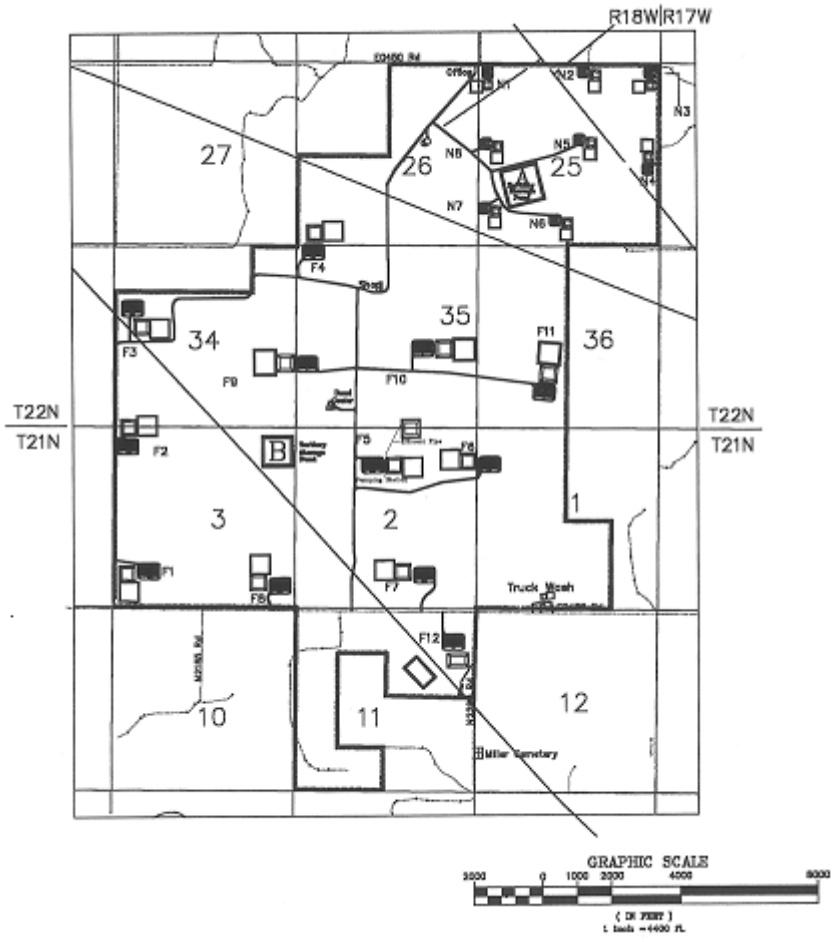
Swine influenza continues to be a challenge in swine production units across the United States. Some of the risk factors to consider include maternal antibody levels, strain type and sequence involved, vaccination compliance, biosecurity at the farm level, transmission, and population dynamics. This list is not all-inclusive, but describes some of the issues I have dealt with in understanding influenza in our production system.

After two years of grief and frustration understanding the clinical syndrome and interaction of disease in our grow/finish system, I have implemented various control strategies. Some were successful; others failed miserably. This discussion will focus on those strategies that were beneficial in our system.

The first lesson learned: Determining maternal interference associated with influenza vaccine requires taking a look at sow vaccine timing, maternal antibody decay, and variation among pigs in the nursery. If groups are run AI/AO in the nursery, it is possible to protect nurseries from influenza by vaccinating sows prefarrow. The maternal antibody titers typically are high enough to protect the pigs through the nursery period. When we vaccinated sows prefarrow, the maternal antibodies typically waned to unprotected levels in early finishing or when pigs were about 13 weeks of age. The pigs were then susceptible to the disease in the finisher phase. If you suspect transmission of the virus will occur in the finisher you need to decide if you need to vaccinate pigflow and if such a cost is justified. If you suspect there is little exposure to SIV

Figure 1. Nursery and finisher geographical locations (note the close proximity of the sites)



in the finisher you may have success with this program without pig vaccination. In our system vaccination prefarrow and not vaccinating pigflow resulted in clinical disease of SIV in the early finishing stage predictably in most groups of pigs.

We failed to understand the population dynamics of this system and the spread of the virus. Our finisher pigs were susceptible by about 13 wks of age, and the environmental conditions perfect for the spread of the virus through the system. Aerosol spread has been implicated in the spread, persistence, and ubiquity of the disease, the explosiveness of epidemics, and the prompt region–wide occurrence of outbreaks (Hammond et al., 1989). Our grow/finish system involves 190,000 pigs 2–29 weeks of age in a 2 mile radius (Figure 1). In 2002–2003 average wind speed has been 10.31mph with a range of 2.2–70.7mph (Oklahoma Mesonet website). These conditions provide an ideal situation for influenza virus to persist and circulate in the population.

Understanding the challenges we have with environmental conditions and density, we have changed our strategy to include vaccination of both sows and pigs to achieve the highest possible level of protection in all age groups. Our strategy is to vaccinate sows and gilts post-breed with bivalent SIV vaccine. By performing cross-sectional serologic sampling in the nursery we calculated the maternal antibody decay. We vaccinate pigs based on the results of serology. We continue to vaccinate both the sow population and pigflow with consistent product, timing, and procedure. Over time, this strategy has proved to decrease clinical disease and improve performance. We have not—and will most likely never—eradicate SIV in our system, but we seem to be controlling the number of clinical cases and hopefully decreasing the amount of virus that is aerosol-spread in the system.

References

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