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Production Assistants
Steven Claas
Michael Klatt

Layout and CD-ROM
David Brown

Logo Design
Ruth Cronje, and Jan Swanson;
based on the original design by Dr. Robert Dunlop

My flu dilemma: Minnesota

Mark Wagner, DVM
Fairmont Veterinary Clinic

Introduction

Since 1998, the clinical disease caused by Swine Flu has become more endemic and less seasonal in Southern Minnesota. This has presented a challenge in deciding how to manage and control flu in the swine population, especially when considering the different subtypes that emerge due to antigenic drift (which allows for gradual changes within the genetic makeup of the virus) and antigenic shift (which allows for reassortment of genes between flu viruses). Either change creates challenges for vaccinating or challenges of managing in an endemic population. In many cases there is no standard control approach and ultimately it depends on the clinical, production, and financial implications the flu is causing for that specific sow herd or wean-finish flow.

To provide a perspective of Swine Flu in Minnesota and specifically my practice area, I have provided the following graphs courtesy of Dr. Marie Gramer at the University of Minnesota Veterinary Diagnostic Laboratory.

These graphs demonstrate that, based on diagnostic submissions to the University of Minnesota, approximately 10% of the cases in any particular month involve flu and prevalence of flu in my practice area during the last four years has been included both H1 and H3 subtypes.

Figure 1:

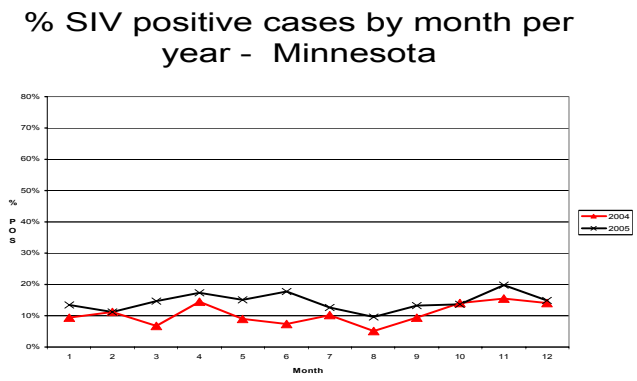


Figure 2:

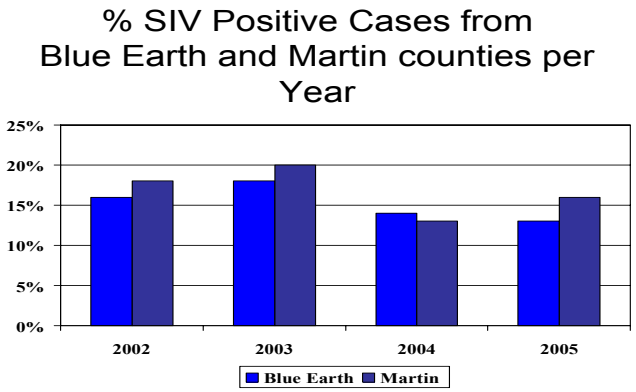
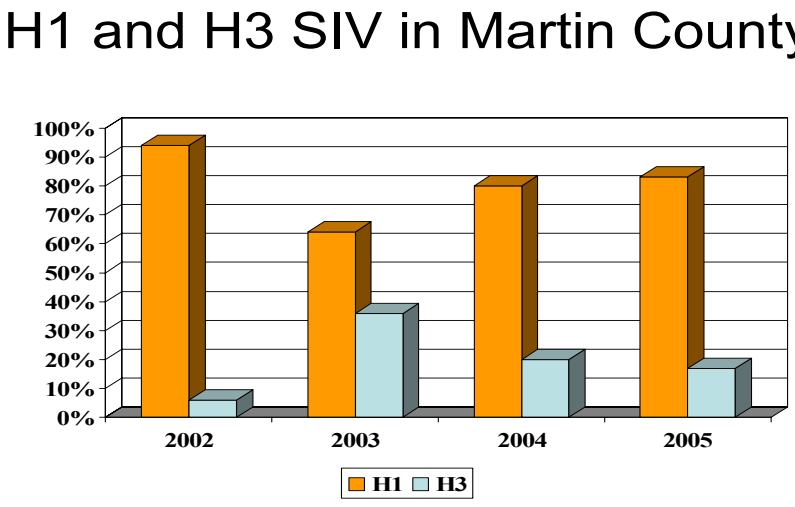


Figure 3:



As, preciously mentioned the control of flu often becomes herd specific. I will now discuss a case history where multiple strategies were applied and their outcomes. This case involved a 1200 sow iso-wean farm that struggled with a persistent H3N2 flu virus despite several interventions over a two year period.

Provided is a summary of when diagnosis was made, in what animal, and what the current vaccine strategy was at the time of diagnosis. (Table 1)

From a clinical perspective, the primary signs at the sow unit were coughing nursing piglets at 15 days of age or older, sporadic off feed gilts (240 days of age) shortly after entering gestation from acclimation, and sporadic off feed sows in farrowing. However, pigs at weaning were in good body condition and sow productivity was good.

Provided is a summary of production at the unit during the period of clinical flu activity. Based on Pig Champ data, there was very little effect on production even though swine flu was endemic and sporadically diagnosed throughout 2004 and 2005. (Table 2)

When one evaluates sow unit performance in the presence of endemic flu in this herd, one would have minimal concerns. However, the nursery picture was very different. (Table 3)

In the nursery, clinical signs were extremely poor appetites, lethargy, gauntness/wasting and cough. Diagnostics revealed not only influenza, but also a significant number of cases of post weaning beta-hemolytic *E. coli* (both K88 and F18), secondary bacterial pneumonias (*H. parasuis* and *Pasteurella multocida*), and more active PRRSV circulation.

This nursery performance prompted us to intervene at the sow level with flu vaccinations to lessen the clinical disease in weaned pigs. However, this became more difficult to implement than anticipated.

Diagnostic results were evaluated and the following H3N2 dendrogram was generated to compare the flu isolates detected in this herd between 2003 and 2005. (Figure 4)

The conclusion to this flu dilemma

Upon the initial appearance of clinical signs and diagnosis of flu (Dec 2003), it was decided that a commercial

Table 1:

Time line of diagnostics	Animal diagnosed in	Vaccine strategy being used
December 2003, H3N2	Nursing piglet	None
April 2004, H3N2	Nursing piglet	Commercial vaccine to replacement gilts (H1N1/H3N2)
January 2005, H3N2	Nursing piglets	Commercial vaccine to replacement gilts and all animals prefarrow (H1N1/H3N2)
August 2005,H3N2	Nursing piglets	Autogenous area specific vaccine to whole herd, replacement gilts, and prefarrow (H1N1/H3N2)

Table 2:

	2004	2005
Total born/sow	12.0	12.6
Born alive/sow	11.0	11.2
Pig weaned/sow	9.9	10.0
Pre wean mortality	8.6%	10.6%
Average age pigs at weaning	18.7	19.2
Average weight at weaning	13.1 lbs	13.3lbs
Farrowing rate	85.7%	83.2%

Table 3:

	Avg. mortality (Wk 0 – Wk 8 post wean)	Range in mortality
2003 (prior to flu)	3.1%	1.2% - 5.0%
2004 (endemic flu)	5.5%	3.5% - 10%
2005 (endemic flu)	6.5%	3.5% - 15%

Figure 4

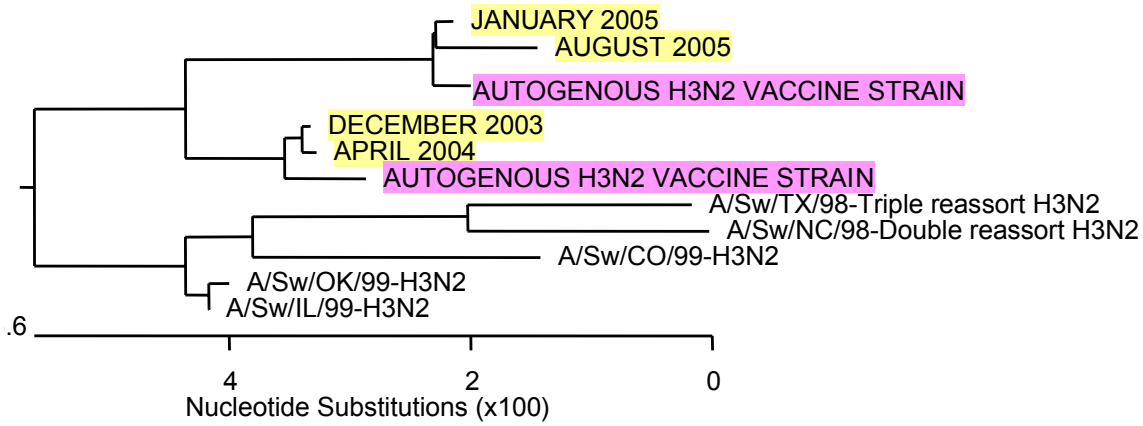


Table 4:

	Percent different
Dec 2003 original isolate vs April 2004 isolate	0.5%
April 2004 isolate vs January 2005 isolate	3.2%
January 2005 isolate vs August 2005 isolate	1.0%
Dec 2003 original isolate and TX, NC, CO reference isolates	6.8% to 8.5%
Dec 2003 original isolate and OK, IL, reference isolates	3.8% to 3.9%
Jan 2005 isolate and Area Autogenous isolates (two strains)	1.8% to 4.4%

H1N1/H3N2 vaccine would be given to all replacement gilts entering herd. Each gilt would receive two doses. This strategy seemed to work until April 2004 (5 months later), when flu was re-diagnosed in nursing piglet population. Genetic comparison between the December 2003 and April 2004 isolates showed 99.5% similarity. Because the virus did not appear to change significantly and we did not have flu activity for several months, we decided to continue with commercial product and vaccinate all sows and gilts prefarrow. This seemed to again work until January 2005 (10 months) when we diagnosed flu again. Genetic comparison showed the April 2004 and January 2005 isolates were 96.2% similar. Because virus had apparently drifted or a new virus had entered population and the current vaccine was not protecting against this recent isolate, the decision was made to use an area autogenous vaccine. Two H3N2 isolates were in this vaccine and genetic comparison showed them to be 95.6% to 98.2% similar to the field virus. It was decided to complete a whole herd vaccination and then continue with a prefarrow vaccine. Again this seemed to work until August 2005 (8 months later) when flu activity again was detected in nursing piglets. Genetic sequencing of this isolate compared to January 2005 isolate showed 99% similarity. At this time, the decision was made to utilize a monovalant farm specific vaccine using the August 2005 isolate. The herd was mass vaccinated in January 2006 and booster vaccinated in March of 2006. Sows and gilts continue to receive a prefarrow monovalent H3N2 autogenous vaccine. To date no clinical relapses of flu have occurred and nursery mortality has ranged from 1.5% to 3.0%.

Summary

This case demonstrates the complexities that can sometimes surround flu. In many flu cases, the problem can easily be solved with the use of either commercial or autogenous vaccine. However, we must continue to be diligent and strive to obtain good diagnostics and then evaluate each intervention as we are able, realizing that some solutions take time and patience.