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My flu dilemma: H3N2 introduction into the negative swine population of Ontario (coinciding with increasing PCVD)

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History

In 1998, H3N2 viruses emerged and rapidly spread throughout the U.S. swine population. These are unique “triple reassortant” genotype viruses with human, swine and avian lineage. Despite geographic proximity and cross-boundary trade in pigs and turkeys between the U.S. and Canada, these reassortant viruses did not infect animals in Canada until late 2004.¹

Introduction of H3N2 into Canada

The first diagnosis of H3N2 was made by the Manitoba Veterinary Services Laboratory in the fall of 2004 and the virus subsequently spread through the swine population in pig dense southern Manitoba over the next several months.²

Presumably as a result of isowean and feeder pig movement between Manitoba and Ontario, the first diagnosis of H3N2 in Ontario occurred in the spring of 2005 with widespread virus movement into the fall.

Again, presumably due to isowean and feeder pig movement, H3N2 was introduced into Quebec in the summer of 2005.

Historical H1N1 prevalence, clinical signs, impact, and control measures

The Ontario pig industry is primarily in a smaller region and is thus relatively pig dense. It has a range of farrow to finish to multi-site production with a historically high prevalence of H1N1.³

Clinical signs of SIV (H1N1) in the industry typically were:

- Low grade endemic circulation in late suckling and nursery pigs. Empirically it has been difficult to determine the cost of infection.
- Periodic and sporadic acute breaks in finisher barns – undetermined generally whether virus was from source or lateral introduction. Again, empirically it is difficult to determine the cost of infection.

Vaccination with monovalent or bivalent commercial SIV vaccines in the sow population was low albeit with better

diagnostics (Influenza A Antigen Elisa from nasal swab) there were more sow herd immunization programs (gilt and pre-farrow) in place attempting to reduce the clinical and presumed economic impact of endemic circulation in the young pig. Growing pig vaccination was negligible.

H3N2 spread, clinical signs, impact, and control measures

The virus moved quickly through the industry during the spring and summer. We were surprised by the amount of lateral spread and in some cases, the distance of lateral spread during the hot summer months.

Not surprisingly, in susceptible populations, classic clinical signs were seen in all stages of production. Again, not surprisingly, some herds/populations sero-converted without any signs.

The impact was transient in most cases and without trailing measurable economic cost. Acetylsalicylic acid was, and remains, the intervention of choice with a cost per 100lb pig of less than 1¢ per day.

The endemic nature and cost of circulating SIV appears to have changed little since the introduction of H3N2.

We have continued to be “underwhelmed” by the effectiveness of sow herd commercial vaccination programs and their impact on this endemic clinical picture. Consequently, through 2006, we have discontinued most of these vaccine programs except for gilt acclimation.

The change in strategy is to continue monitoring herds, diagnose changes in clinical signs with virus isolation and sequencing (try to put some science into SIV) and consider homologous immunity through autogenous vaccine (now available in Canada).

PCVD impact vis-à-vis H3N2 prevalence

The primary disease impact through the last 1.5+ years in Ontario has been due to PCVD. Our practice summary data on disease impact is shown in **Table 1** and **Figure 1** with 1st Q’06 finisher mortality increasing from the previous year from 3.6% to 6.9% with the range dramatically changing. H3N2 spread through the industry in the

second and third quarter of 2005. Was there a material impact of H3N2 on PCVD expression through this period? I don't know and within a diverse and complex industry structure there are daunting challenges in defining impact. It is apparent the primary clinical impact of H3N2 was in the middle quarters of 2005 but by the first quarter of '06 PCVD driven mortality was still trending higher. This region's empirical observations would suggest the H3N2 epidemic may have been an additional co-factor in some PCVD herd cases but was not nor remains a significant co-factor.

including SIV. The key summary points of this are:

- AIAO
 - site
 - barn (max 2 barns/site)
 - room
- Sanitation
 - Site & transport
- protocol
- audit
- Do not co-mingle from
 - sow
 - nursery
 - gilt sourcing

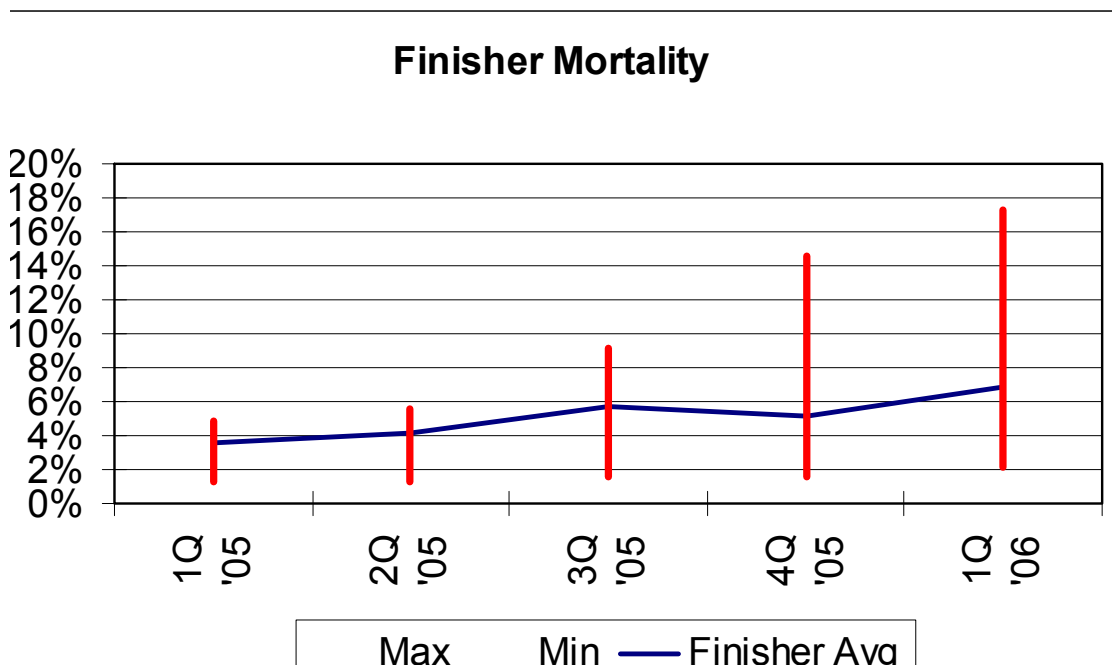
Summary: Principles of disease control – SIV & PCVD

The primary intervention strategy has been centered on taking the principles of Madec's PCVD strategy and customizing it to North American production practices. This strategy forms the basis of control direction for all diseases

Table 1

	1Q '05	2Q '05	3Q '05	4Q '05	1Q '06
# Barns/Groups	32	32	41	35	38
# Placed	52798	62015	68012	64242	63363
% Mortality - avg	3.6%	4.2%	5.7%	5.1%	6.9%
% Mortality - min	1.3%	1.3%	1.5%	1.5%	2.1%
% Mortality - max	4.8%	5.6%	9.2%	14.6%	17.3%

Figure 1:



Doug MacDougald

- boar sourcing
- Limit pig movement within AIAO groups
 - minimal age spread within all group stages.
- specific minimum and maximum limits
 - cross-foster restrictions
- Effective sow herd immunity
 - gilt acclimation - separate site from sow site
 - sow herd immunity program
 - colostrum management
- Biosecurity
 - protocol
 - audit
- Effective pig care
 - space
 - air
 - feed/rations
 - water
 - pig/population intervention
- Effective diagnosis and monitoring of disease.
- Control or eliminate primary pathogen circulation in S-N-F
- Effective information system
 - timely
 - accurate
 - integrated production, financial and health
 - targets

References

1. Olsen, CW et al. (2006) NIAID, *Emerging Infectious Diseases*
2. Pasma, T, personal communication
3. Friendship, RM; Dewey, CE; Poljak, Z; Carman, S (2006) *IPVS*, Increased Prevalence of Swine Influenza H3N2 in Canada

