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# Protection Against H1N2 Swine Influenza Virus Challenge Following Vaccination with Swine Influenza Vaccine Containing Subtypes H1N1 and H3N2

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## Introduction & Objectives

The H1N2 subtype of Swine Influenza Virus (SIV) was first reported in North America in 2000.<sup>1</sup> Phylogenetic analysis of multiple isolates from numerous states suggests that these viruses arose by a reassortment between the classical H1N1 virus and the triple reassortant H3N2 viruses.<sup>2,3</sup> While the number of H1N2 SIV isolates from diagnostic laboratories is a minor proportion, there is a trend for increasing incidence over the last 3 years.<sup>3</sup> Commercially available SIV vaccines contain both the H1N1 and H3N2 subtypes. The purpose of this study was to confirm whether a commercial vaccine would protect against challenge with a virulent H1N2 field isolate.

## Materials and Methods

A commercial batch of Maxivac® Excel (Lot #229139) containing inactivated SIV H1N1 and H3N2 in Emunade adjuvant was used. Pigs were 5-6 weeks of age at first vaccination. Group A pigs (n=14) remained as unvaccinated controls. Group B pigs (n=14) were vaccinated twice, 15 days apart, with 2 ml by the intramuscular route. Pigs were challenged with 4 ml of H1N2 virus (North Carolina isolate 407-059) by the intranasal route 27 days after the 2<sup>nd</sup> vaccination. Pigs were observed for 5 days after challenge and then necropsied to assess lung lesions. Lung scores were assigned as described previously.<sup>4</sup> Seroconversion to SIV was monitored prior to vaccination and on the day prior to challenge. Antibodies to SIV H1N2, H3N2, and H1N1 were measured by hemagglutination inhibition (HI) assay using antigens homologous to the vaccine or challenge viruses.

## Results and Discussion

All pigs were seronegative to SIV prior to vaccination (data not shown) and the controls remained seronegative prior to challenge (Fig 1). The HI antibody response was stronger to the H1N1 and H3N2 serotypes than to H1N2. The lower cross-reactivity to the H1N2 virus is most likely due to antigenic drift in the H1 antigen. The H1N2 challenge induced little fever or clinical signs during the 5 day observation period. However, the challenge induced lung lesions in 100% of non-vaccinated controls with a median score of 5.65. Despite the low levels of HI antibodies to the challenge strain, the vaccine induced nearly 70% reduction in lung

lesions, which was highly significant ( $p=0.0101$ ). In fact, 43% of the vaccinated pigs had lesions scores  $\leq 1$ , whereas all controls had lesion scores higher than 2. These data indicate that a combination SIV vaccine (containing H1N1 and H3N2) will provide protection against a virulent H1N2 subtype. The high level of protection against this heterologous virus that appears to have a divergent H1 antigen, suggests that cell-mediated immunity may play a role in this protection.

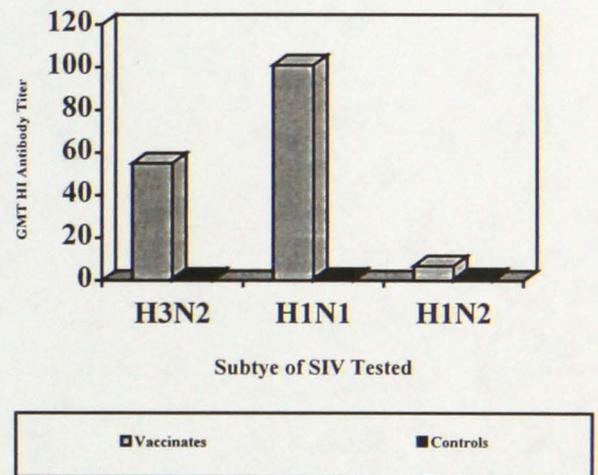


Figure 1. Serological Response to SIV Prior to Challenge (day 39)

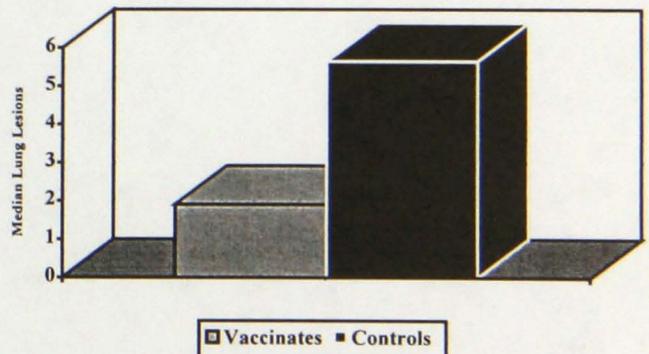


Figure 2. Lung Lesion Scores Induced by H1N2 Challenge

## References

1. Karasin A et al. J. Clin. Micro. 38:2453-2456, 2000.
2. Olsen, C Virus Res. 85:199-210, 2002.
3. Choi YK et al. Arch. Virol. 147: 1209-1220, 2002.
4. Rapp-Gabrielson, VJ et al. AASV Proceedings 2002, pp101-103.