
POOR IMMUNITY FROM A *STREP. SUI* AUTOGENOUS BACTERIN

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Immunity

Streptococcus suis is a major cause of death and disease losses, especially among recently weaned pigs. This disease results from bacterial infection. While it may respond to antibiotics or vaccination, *S. suis* is difficult to control since there are many strains (more than 35 serotypes) that can cause sickness.

The disease often strikes quickly and makes pigs quite sick, with bacteria in the brain and joints, before treatment can be started. During a flare-up, the best chance for an infected pig is an injection of antibiotic.

A potential advantage of vaccination is the herd can be protected before disease has flared up and expensive antibiotic treatments may be unnecessary. A successful outcome will depend on antibody level and antibiotic at the site of infection.

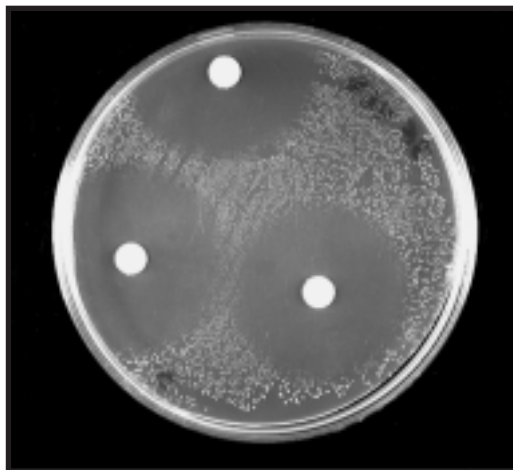
Our objective was to measure the amount and variation in the antibody response

Producers who have to control this disease must make a difficult choice of where to spend limited resources for disease control. Vaccination to prevent disease can be very cost-effective if the vaccine is designed to control the type of bacteria causing disease. This problem can be addressed by having a custom-made vaccine (called an autogenous bacterin) produced from bacteria that are isolated from an untreated sick pig. In this type of situation, the protection from disease depends on the presence of antibody in the serum of vaccinated pigs.

If all vaccinated pigs made a potent immune response, then the choice to get an autogenous bacterin would be economically sound. If there were a large amount of variability or poor response after vaccination, then an alternative disease control strategy might be more effective.

Study herd and trial design

We conducted this study in a Saskatchewan herd of pigs with high quality commercial genetic background. The herd experienced disease loss due to *S. suis*, and there has been on-going treatment for acute and chronic disease. The difficulty and cost of treatment has increased because the *S. suis* now causing disease in this herd are resistant to penicillin.

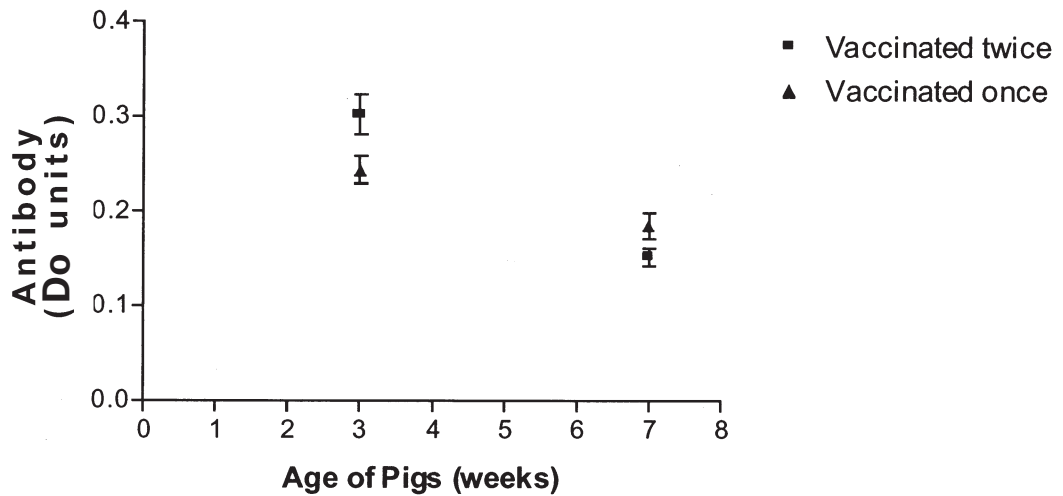


This test of antibiotic susceptibility shows that *Strep. suis* SX-428 is resistant to penicillin and has only intermediate susceptibility to ampicillin and cephalosporin.

A strain of *Streptococcus suis* was isolated from an untreated pig six weeks of age and designated SX-428. This strain was sent to a vaccine manufacturer that prepared an autogenous bacterin. This strain was also used in our laboratory to measure the amount of immune response in vaccinated pigs. The response was measured by using a technique called a "whole-cell ELISA". A positive control pig immunized with *S. suis* (serotype 2) had antibody level of 0.331 with a coefficient of variation of 15.6 per cent.

*Better
vaccines or
antibiotics
are needed
to control
this
pig-killing
disease.*

Antibody to *Streptococcus suis* in Pigs Immunized with Autogenous Bacterin



Each pig in the trial was given a unique ear tag ID, a “pre-vaccination” blood sample was taken at weaning (three weeks of age) and a “post-vaccination” blood sample was taken at seven weeks of age. The 50 pigs in Group 1 were vaccinated twice (at weaning and 16 days later) in accord with the recommendations of the consulting veterinarian. The 50 pigs in Group 2 were only vaccinated once, at weaning.

Response to Vaccination

The graph above shows the average (and standard deviation) amount of antibody present in the blood of the pigs in this study against the strain of *S. suis*. The effect of number of vaccinations (1 or 2) is considered not significant ($p > 0.41$). The effect of time is highly significant ($p < 0.01$); however the total amount of specific antibody declined. Environment, disease challenge and host immunity can all affect the occurrence of disease. The vaccine given during this study did not reduce the amount of clinical streptococcal

disease in this herd.

Implications

The autogenous bacterin failed to increase antibody levels in vaccinated pigs. The lack of clinical response in the herd following vaccination is consistent with the absence of an increase in specific antibody level. The lower antibody level at seven weeks of age is consistent with normal decline in passive antibody level and is probably not influenced by vaccination in this case. The autogenous bacterin used in this herd was not economically justified since vaccinated pigs did not have increased immunity to *S. suis*.

Acknowledgements

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