20th International Pig Veterinary Society Congress

June 22-26
Durban
South Africa

We are delighted that the International Pig Veterinary Society Congress 2004, decided to select South Africa as the host country for the 20th IPVS Congress. The Pig Veterinarians of South Africa will ensure that this congress lives up to the best traditions of previous congresses; incorporating an interesting and topical scientific programme, fascinating accompanying persons tours and an excellent social programme, allowing delegates the opportunity to network with their overseas colleagues.

This, the first IPVS congress on the African continent, will undoubtedly be of enormous benefit in generating solutions to the emerging pig veterinary challenges, especially those related to exotic and changing viral diseases, decreased use of antimicrobials and nutritional advances. The congress is important to further pig veterinary science in South Africa, to encourage younger veterinarians to join the pig industry, as a vehicle to generate funds for research and to improve the pig industry in Southern Africa.

South Africa is a magnificent and beautiful country, and offers tourists value for money. Thus, pre and post congress tours will be a major attraction for delegates to come to South Africa. Durban, in KwaZulu Natal, is a vibrant multi-cultured city with magnificent beaches, easily accessible game parks, theme villages and a moderate winter climate making it an ideal tourist destination. We urge our colleagues throughout the world to use this opportunity to get a glimpse of the continent’s rich and fascinating wonders and to enjoy the hospitality of their African friends.

Dr Peter Evans
Chairman: Local Organising Committee: IPVS 2008
INTRODUCTION

In the last few years we registered more and more cases of necrotizing enteritis due to Clostridium (C.) perfringens type C in pigs in Switzerland. The farmers of a high breeding herd have to be free of infections with C. perfringens type C and vaccination against the agent (1) is forbidden.

Necrotizing enteritis in suckling piglets is caused by the spore-forming anaerobe and toxin-producing C. perfringens type C (2). A typical clinical symptom is bloody diarrhoea of several piglets of a litter during the first day after birth. In certain cases the infection leads to death immediately without clinical manifestations. A treatment is futile when symptoms appear. C. perfringens type C produces α- and β-toxin. The β2-toxigenic C. perfringens are not assigned to specific toxino-type. The β2-toxin can be found in C. perfringens in association with the α-toxin (type A) or with α- and β-toxin (type C). Both, β- and β2-toxin are sensible for trypsin (3) what explains the early occurrence of the disease.

The objective of this study is (i) to investigate the presence of β- and/or β2-toxigenic C. perfringens in diseased and healthy animals and (ii) to find out predisposing factors for an outbreak of necrotizing enteritis in a swine herd.

MATERIALS AND METHODS

One hundred swiss farms with at least ten sows were selected randomly and classified in two groups. In the negative group farms are classified which had never necrotizing enteritis due to C. perfringens Type C. The positive group includes the farms which had a case of necrotizing enteritis during this study or had a case before the beginning of the study and now they are using a vaccination containing β-toxoid of C. perfringens. The data of the farms were collected by a questionnaire. In 17 herds faecal samples from sows and piglets were taken twice in an interval of 14 days and the material was analyzed for presence of C. perfringens. The Detection of α-, β- and β2-toxin genes was performed by PCR (4). The results were compared to the anamnesis of the farms.

RESULTS

At the moment we analyzed 1262 faecal samples of piglets and sows in 17 farms. In 716 swab samples we detected C. perfringens. In 616 samples we could find β2-toxigenic C. perfringens and in 71 samples C. perfringens with β- and β2-toxin genes. The β-toxin gene was detected always in combination with the β2-toxin gene. Furthermore the β-toxin gene was detected only in the positive classified farms and exceptionally in a sample of one piglet of a farm classified in the negative group. Two months later there was a classical outbreak of necrotizing enteritis due to C. perfringens type C in this swine herd. With exception of one farm, we found more pigs excreting β- and/or β2-toxigenic C. perfringens in the first taking of sample than in the second (Fig. 1). In piglets we isolated more C. perfringens in the majority of cases. Furthermore we had only nine sows with a positive result for the β-toxin gene, eight of them were in the same farm. All other β-toxigenic C. perfringens positive faecal samples came from piglets.

DISCUSSION

Our results show that it is irrelevant whether a grower had an outbreak of necrotizing enteritis or not, C. perfringens is present on his farm. The great difference seem to be the presence of the toxin genes. All farms we detected the β-toxin gene were associated with an acute outbreak of necrotizing enteritis or an incorrect management of vaccination. In all farms we detected β2-toxigenic C. perfringens. This fact points out the important role of the β2-toxin gene for an outbreak of necrotizing enteritis in a swine herd.

Furthermore we observed a higher quantity of β- and/or β2-toxigenic C. perfringens in the first sampling than in the one two weeks later. Usually, we collected the faecal samples before any medical treatment of the piglets. But this was not possible in those farms with an acute outbreak because the farmer had had to apply medicaments which decrease the quantity of proved bacteria. We can not exclude the presence of β- and/or β2-toxigenic C. perfringens with the analysis of a faecal sample of a sow.

REFERENCES