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.

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INTRAMUSCULAR APPLICATION OF COLISTIN DID NOT PREVENT EXPERIMENTALLY INDUCED EDEMA DISEASE IN PIGLETS

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Introduction
Shiga toxigenic E. coli (STEC) have been implicated as the cause of edema disease in swine. In some of the STEC strains, fimbrial antigen F18ab has been demonstrated (1, 2). Susceptibility of piglets to infection with strains possessing the colonization factors F18ab is conditioned by the presence of a receptor on enterocytes. Edema disease is being developed in piglets as a toxemia. Only small proportion of the shiga toxin (Stx) present in intestinal lumen reaches the vascular system (3). Further details such as permeation through the intestinal barrier are not known. STEC are frequently isolated in pure culture from swollen or edematous lymph nodes but not from other tissues or blood of piglets suffering from edema disease. The primary objective of the present study was to determine if intramuscular application of colistin can prevent colonization of lymph nodes of piglets after infection with STEC and even prevent the development of edema disease.

Materials and Methods
Two experiments with 12 weaned piglets each were carried out. Each of the experiments included 6 experimental and 6 control piglets. The experimental piglets were injected colistin at a dose 250,000 IU/10kg as Ampisur (CEVA) with ampicillin. All piglets were individually orally challenged with STEC O139:F18, Stx2e at a dose 2x10^{11} CFU per animal. The strain was resistant to ampicillin and sensitive to colistin. In the following 3 days, piglets were applied STEC culture at the same dose mixed in feed. STEC shedding was monitored by bacteriological examination of rectal swabs. Piglets with severe course of edema disease were euthanized. Dead or euthanized piglets were subjected to pathological examination and histological examination of the intestine. The intestines, mesenteric lymph nodes and organs were examined by histological sections using rabbit serum anti O139:F18 and conjugate against peroxidase labeled rabbit Ig. Sensitivity or resistance of the piglets to colonization with F18 positive E. coli was examined by PCR for the presence of nucleotide transition (G → A) at amino acid position 103 of the FUT gene.

Results and Discussion
In the first experiment STEC shedding in feces of piglets continued till day 10 following infection. From day 4 to day 6 STEC were found in feces of all piglets in the prevailing culture. Examination of G → A mutation revealed that all piglets were sensitive to colonization with F18 positive E. coli. In spite of that, all piglets remained healthy and edema disease was not induced in piglets. In the second experiment clinical signs of edema disease were apparent 48 h following infection in 2 piglets which were applied colistin. In this group, edema disease was diagnosed in 5 animals. Besides swelling of eyelids, those piglets suffered from severe neurological dysfunctions. In one piglet, bloody diarrhea was observed. Four piglets died within 2 days following appearance of the first signs, one piglet was euthanized ante finem. In the group without colistin, edema disease was diagnosed in 3 piglets. In 2 piglets, severe neurological disorders were observed and piglets had to be sacrificed. One piglet exhibited a mild course of the infection and a spontaneous recovery could be seen within 7 days. STEC were detected in feces of piglets since day 1 following the first infection. Since day, 3 STEC were detected in all piglets in the prevailing culture and shedding lasted 8 days following infection.

References