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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
DEVELOPMENT OF A GENETICALLY MODIFIED NONTOXIC Pasteurella multocida TOXIN AS A CANDIDATE FOR USE IN VACCINES AGAINST PROGRESSIVE ATROPHIC RHINITIS IN PIGS

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Introduction
Pasteurella multocida toxin (PMT) is the primary etiology agent of progressive atrophic rhinitis (PAR) (1). Genetic information on the toxin-encoding gene has enabled the development of a new generation of vaccines against PAR using a modified nontoxic PMT.

Recently, Petersen et al. (2) showed that a deletion derivative (dO) of PMT, lacking 121 amino acids at the N-terminal region is nontoxic and immunogenic. Ward et al. (3) have also pointed out that mutant toxin replacing cysteine with serine at position 1165 (C1165S) is nontoxic to piglets and cultured cells. However, these studies did not clarify whether the mutant C1165S could be a good candidate for a vaccine component against PAR or not.

In this study, we have prepared a mutant toxin by replacing serine 1164 with alanine, together with replacing cysteine 1165 with serine (S1164A+C1165S), and examined the possibility of applying it as a candidate for a vaccine component against PAR.

Materials and Methods
The wild-type and mutant toxins used in this study were produced by E.coli XL-1 blue transformed with pSN1131, containing the full coding sequence of PMT, and with mutagenized plasmid pTH161, encoding mutant toxin S1164A+C1165S, respectively (4). The pTH161 was created from the pSN1131 by sequential PCR steps (5). The toxins were purified as described previously (4).

Dermonecrotic test in guinea pigs and intraperitoneal toxicity test in mice (5) were used to determine the minimal dermonecrotic dose and the 50% lethal dose, respectively.

For evaluation of immunogenicity of the mutant toxin, three pigs were immunized intramuscularly with 16 μg of the mutant toxin twice at a 3-week interval. Another 2 pigs were used as non-immunized control animals. Two weeks after the second immunization, all 5 pigs were challenged intramuscularly with 4 μg of the wild-type toxin/kg of body weight. The pigs were euthanatized and autopsied 2 weeks after the challenge. Turbinate atrophy was examined macroscopically and assigned a score that ranged from 0 to 3: 0, normal; 1, slight; 2, moderate; 3, severe.

Results and Discussion
The elution profiles of the wild-type and mutant toxins by use of chromatography were similar to each other. The SDS-PAGE patterns of both PMTs were totally identical. Western immunoblotting analysis with polyclonal antisera against PMT showed that the immunoreactivities of both PMTs were indistinguishable. These data indicate that this two-amino acid substitution has not affected gross structure, the electrical charge and antigenicity of the toxin.

The minimal dermonecrotic dose in the guinea pig skin test was calculated as 0.5μg/ml for the wild-type toxin and > 840μg/ml for the mutant toxin. The 50% lethal dose for mice was approximately 20μg for the wild-type toxin and > 3,200μg for the mutant toxin. These results show that mutation of S1164A together with C1165S led to a complete loss of toxic activity of PMT.

In protection assay, the immunized pigs became seropositive but the control pigs remained seronegative by the anti-PMT ELISA. At necropy, it was found that the scores of turbinate atrophy of the 2 control pigs were 2 and 3, respectively, whereas those of the three vaccinated pigs were 0 (Figure 1). These data suggest that a nontoxic, modified PMT induces antitoxic immunity that protects pigs against experimental challenge with wild-type PMT.

Figure 1 Cross sections of snouts showing the degree of turbinate atrophy in the vaccinated and control pigs

This study clarified that this genetically modified PMT, S1164A+C1165S, is a potential vaccine component for PAR. More importantly, it documented that the mutagenesis approach enables efficient production of this pure, nontoxic, and highly immunogenic protein.

References