20th International Pig Veterinary Society Congress

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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-cultural 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
THE DOWN-REGULATION OF MHC MOLECULE ON MONOCYTIC CELLS AFTER CLASSICAL SWINE FEVER VIRUS INFECTION

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Introduction
Classical swine fever (CSF) is a highly contagious viral disease of pig. Infected pigs display severe impairment of immune system. CSF virus (CSFV) has high affinity for vascular endothelial cells and lymphoreticular cells including T cells, B cells, and monocytes (1). Severe depletion of B cells and T cell in PBMC and virus persistence in lymphoid tissues has been thought the most important characteristics of CSFV infection that leads to the acquired immunosuppressive state (2, 3, 4). Many viruses utilize different mechanisms to avoid viral elimination by the host’s immune response. Some viruses with monocytic tropism may have favoured the development of a broad range of immune evasion strategies (5, 6). Though leucopenia is the centre of immunosuppression during CSFV infection, the effect of CSFV infection on monocytic cells may play an important role in the viral immune evasion and persistent infection. Therefore the aim of this study was to evaluate the effect of CSFV on the phenotype and phagocytic activity of monocytic cells.

Materials and Methods
Peripheral blood mononuclear cells (PBMC) were prepared as described previously (2). Monocyte-derived macrophages (MDM) were cultured by adhesion of PBMC on plastic tissue culture plates with PRPMI-1640 complete culture medium (CCM). Monocyte-derived dendritic cells (Mo-DC) were prepared by cultured MDM in the presence of GM-CSF/IL-4 for 9 to 12 days (6). Alveolar macrophages (AM) were prepared from bronchoalveolar lavage fluid by adhesion on plastic tissue culture plates. Mock or CSFV-infected MDM, Mo-DC and AM were trypsinized and collected for phenotype and functional assays after 3 days infection of virulent CSFV.

Phenotype was assayed by indirect immuno-fluorescence antibody staining of specific monoclonal antibodies (mAb) to swine leukocyte antigen (SLA)-I (mAb 7-34-1), SLA-AB (mAb 74-11-10), SLA-DR (mAb MSA2) and SWC3a (mAb 74-22-15). Phagocytosis were assayed by co-incubation of MDM, Mo-DC and AM with FITC-conjugated *Salmonella typhimurium* or *Pasteurella multocida* in 1:100 ratio at 37 °C for 30 minutes. Cell viability was assayed by staining of Annexin V-FITC/ PI apoptosis detection Kit (R&D Systems). All assays were analysed with FACS Calibur™ flowcytometer and Cell Quest® software.

Results
Flow cytometry data were calibrated by fluorescence mean difference between each assay and cell auto-fluorescence. The expressions of SLA-I molecules were significantly decreased in CSFV infected Mo-DC (Figure 1A), MDM (Figure 1B), AM (Figure 1D) and AM from CSFV-infected pigs (Figure 1C) (p<0.05). The expressions of SLA-AB were also significantly decreased in virus infected AM (Figure 1C, D) (p<0.05), but not in Mo-DC and MDM (p>0.05). There was no significant change of SLA-DR (MHC class II) (Figure 1) and SWC3a (data not showed) molecules between virus infected and mock infected groups. Both phagocytosis and cell viability were not affected after virulent CSFV infection (data not showed).

Discussion
The results showed here a particular effect on SLA-I expression of monocytic cells after CSFV infection. SLA-I molecules present endogenous peptides to activate CD8 T lymphocytes that control virus replication within the cells. Therefore, the decrease of MHC class I expression on cell surface and not enhance of apoptosis of monocytic cells may be helpful for CSFV escaping from host immunosurveillance and persistence in tissues.

References