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In the present study, type A influenza live virus, NC-22-8, which is a combination of a cold-adapted attenuated donor virus (HTCA-A101) and a wild type virus (A/New Caledonia/20/99), was constructed and the efficacy of this new virus was assessed by immunogenicity and protection tests in the mouse model. NC-22-8 ($1'10^7$, $1'10^5$, $1'10^3$ pfu/mouse) was intranasally administered to mice. Four weeks later, the titers of specific IgG and haemagglutinin inhibition (HI) were measured from blood and the titer of secretary IgA (sIgA) was also detected from boncho alveolar lavage (BAL) and mucosal fluid. For the protection test, wild type viruses were intranasally administered to mice immunized previously with the reassortant virus, and then 4 days later, virus plaques were counted from the excised lungs. As a result, a specific IgG titer in serum and the titers of sIgA in BAL and mucosal fluid were 540, 34, and 2, respectively when titers are given as the reciprocal of the calculated sample dilution corresponding with an A_{490}=0.2. The titer of HI for quantification of specific viral antibody was 30.4. In the mouse protection test, there was no wild type virus plaque detected from the excised lungs, indicating a complete protection effect of the vaccine. In conclusion, when compared to an inactivated vaccine, our new reassortant live virus (NC-22-8) showed much potent immunogenicity and protection efficacy.

[PB4-18] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Ginsenoside Rg3 enhances phagocytosis of microglia when activated by β-amyloid in rat primary culture

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β-amyloid (Aβ) peptide produced from amyloid precursor protein (APP) is a major cause of Alzheimer's disease (AD). Therefore, in early phase of AD, imbalance of the production and the clearance of Aβ is regarded as an important factor to progressive AD presenting senile plaque, a hallmark of AD. In the present study, we wanted to verify whether Rg3 can play a role in helping microglia engulfing Aβ peptides. Validations for the study was conducted by using DiI-Ac-LDL, which attached only on type A macrophage scavenger receptor (MSR-A) and ligands for the receptor, fucoidan. In cell culture, we found that microglia started to engulf the studied peptides from 2h with a pick values at 4h when treated with 100㎍/㎖ of Rg3. The whole phagocytosis was finalized by releasing most the engulfed peptides at 8h. From the experiments, we concluded that Rg3 can help microglia to carry out the phagocytosis, effectively, when massive amounts of Aβ peptides are made and existed in the brain.

[PB4-19] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Effects of cyclosporin A and tacrolimus on the cross-presentation capability of dendritic cells

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Previously, we showed that cyclosporin A and tacrolimus, but not rapamycin, inhibit MHC class I-restricted presentation of exogenous antigen in dendritic cells (DCs). We further characterized the effects of cyclosporin A and tacrolimus on the uptake, processing and cross-presentation of a model antigen, ovalbumin (OVA), in DCs. Treatment of DCs with cyclosporin A or tacrolimus did not inhibit phagocytic activity of DCs. Instead, treatment of DCs with cyclosporin A or tacrolimus inhibited the expression of H-$2K^b$ molecules complexed with the OVA peptide, SIINFEKL, specifically. When DCs were allowed to phagocytize microspheres containing both OVA and cyclosporin A, they were also unable to express H-$2K^b$ molecules complexed with the OVA peptide, SIINFEKL. The effects of cyclosporin A on the induction of cytotoxic T cells were investigated in mice with the the microspheres containing both OVA and cyclosporin A. Mice injected with the microspheres containing both OVA and cyclosporin A were unable to generate OVA-specific cytotoxic T cells. Altogether these data suggest that the immunosuppressive activity of cyclosporin A and tacrolimus is at least in part due to inhibition of the cross-presentation capability of DCs.