巴斯德讲坛-资深系列

Pasteur Colloquium-Senior

New vaccine strategies using cytomegalovirus

[Speaker] **Prof. Klaus Frueh**

[Time] **10:00-11:30AM, November 9, 2017**

[Host] **Prof. Zhikang Qian**

[Venue] **A0201, Life Science Research Building**

[Speaker Introduction]

2011-2016 President, Chief Scientific Officer, TomegaVax Inc (merged with Vir Biotechnologies)
2005- Professor, Department for Molecular Microbiology and Immunology, OHSU
2005- Senior Scientist, Oregon National Primate Research Center, Beaverton, OR
2005- Senior Scientist, Vaccine and Gene Therapy Institute, OHSU, Beaverton, OR
2000- Director, Gene Microarray Shared Resource, Oregon Health & Science University, Portland, OR

[Abstract]

Vaccine vectors based on cytomegalovirus (CMV) represent a novel platform that maintains high frequencies of non-exhausted effector memory T cells in both CMV sero-positive and sero-negative individuals. In non-human primate (NHP) models, CMV vectors can be genetically altered to program highly diverse CD8+ T cell responses that differ in their epitope targeting including CD8+ T cell epitopes presented by MHC class II or non-polymorphic MHC-E molecules. Several viral gene products have been identified that positively or negatively regulate the priming of these unconventional responses. Moreover, MHC-II and MHC-E restricted CD8+ T cell responses require vector targeting to distinct host cells thus enabling immune programming via regulation of viral cell tropism. Using genetically modified CMV vectors it is thus possible to program different populations of CD8+ T cells that recognize distinct, non-overlapping epitopes presented by MHC-Ia, MHC-II or MHC-E. The role of CD8+ T cell responses targeting different conventional or unconventional epitopes in protection against pathogen challenge is being studied in NHP models of AIDS, Tuberculosis and Malaria. The results from these experiments will inform the clinical development of human CMV-based vaccines and immunotherapies. By modifying cytomegaloviral and host determinants that control T cell priming it thus possible to uniquely tailor the CD8+ T cell response to each individual disease target in order to maximize prophylactic or therapeutic efficacy.

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