



Efficacy of the new *Yersinia pestis* subunit vaccine in animal models of plague

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Abstract

Until recently, the vaccine against *Yersinia pestis*, the etiological agent of plague, consisted of a formalin-inactivated, whole-cell vaccine. The vaccine was discontinued because it apparently only protected the vaccinated host against bubonic plague but not pneumonic plague. We have since found that the whole-cell vaccine only induced antibodies against the capsule F1 protein but not antibodies against the virulence protein (V-antigen) that appears to be required for a robust protection. The new plague vaccine consists of subunits of the F1 capsule protein and V-antigen either as individual subunits or a fusion of the two subunits. The genes for each these proteins are encoded on two separate virulence plasmids; one of the plasmids is specific for *Y. pestis*. Initially, it was proposed that only antibodies against the vaccine subunits were sufficient for protection against an exposure to the pathogen. Part of this reasoning was from studies with immune serum or monoclonal antibodies against the F1 or V-antigen subunits that showed that these sources of antibodies can passively protect an animal against a plague infection. Nevertheless, we have shown that the participation of the innate immune system is required for complete protection against a pneumonic plague challenge with *Y. pestis* CO92 a fully virulent strain of plague. Although it is not completely clear how protection is mediated by the new subunit vaccine, the subunit vaccine has been through a Phase IIa human clinical trial. We will present the efficacy of the new *Y. pestis* plague subunit vaccine in two animal models of plague

Biography

Amemiya received his doctoral degree from Rutgers University in Microbiology in 1973. He did his post-graduate studies in gene regulation in the laboratory of Lucy Shapiro at Albert Einstein College of Medicine, Bronx, N.Y. Later, he went to the National Institute of Neurological Diseases and Stroke in 1986, where he examined gene regulation in JC virus that caused the demyelinating disease progressive multifocal leukoencephalopathy in immune suppressed patients. In 1999, he went to the U.S. Army Medical Research Institute of Infectious Diseases, Bacteriology Division, where he has been involved in vaccine development for *Burkholderia mallei* and *Yersinia pestis*. His primary interest has been in the immune response and innate immunity in animal models.

This work is partly presented at International Conference on Biothreats & Biodefense
October 15-17, 2012 Double tree by Hilton Chicago-North Shore, USA

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