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The innate immune response to the plague vaccine in vaccination and protection

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Pandemics of plague occurred hundreds of years ago, and although it was thought that this was a disease of the past, episodes of the disease still occurred as recently as a hundred years ago. Partly because of its virulence and ease of growth, *Yersinia pestis*, the etiological agent of plague, has been used and considered as a biological weapon within the last century. The first plague vaccine was developed at the beginning of the twentieth century after the identification and isolation of the pathogen. An inactivated whole-cell vaccine had been used in Western countries until recently, but because it was only protective against the bubonic form of the disease, its use has been mostly discontinued. An attenuated strain of plague is still being used as a vaccine in countries of the former Soviet Union, although it is highly immunoreactive. Recently, a subunit plague vaccine has been developed and shown to be efficacious in both bubonic and pneumonic forms of plague in animal models. It has been through a Phase IIa human clinical trial with little reported events. It is believed that protection against plague is primarily antibody-mediated because of passive protection studies with immune serum in murine and nonhuman primate studies. However, there is evidence that suggests that protection in part against *Y. pestis* is cell-mediated. Our results now showed that the innate immune response to the new plague vaccine can influence both humoral and cellular immune responses to the vaccine, and it is also required for protection against *Y. pestis* in vaccinated mice.

Biography

Dr. Amemiya obtained his B.S. and M.S. at Long Beach State University in Microbiology. He received his doctoral degree at Rutgers University in Microbiology in 1973. He went to Albert Einstein College of Medicine, Bronx, NY, to do post-graduate studies in the cell-cycle of the diphasic bacterium *Caulobacter crescentus* and studied gene regulation during development in the laboratory of Lucy Shapiro. Later he went to the National Institute of Neurological Diseases and Stroke in 1986, where he examined gene regulation in JC virus, which is a human polyomavirus associated with the demyelinating disease progressive multifocal leukoencephalopathy in immunosuppressed patients. After looking at transcriptional regulatory factors that are responsible for tissue-specific gene expression of JC virus, he became involved in gene regulation studies in neuroborreliosis caused by *Borrelia burgdorferi*, the agent of lyme disease with Andrew Pachner at Georgetown University Medical School. He went back to NINDS to study the expression of cytokines in cell-mediated inflammatory myopathies. 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 and the immune response to candidate vaccines of glanders and plague. His primary interest has been with adjuvants and the innate immune response to vaccines in animal models.