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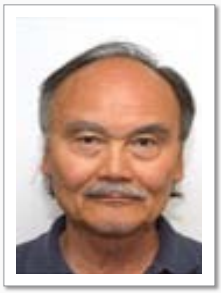

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Augmentation of the immune response to a vaccine by innate immune modulators

The development of a protective vaccine may take years of evaluation of vaccine candidates that offer only limited protection in animal models. In many cases, the evaluation process goes through a whole-cell stage to subunit components that are more effective in providing protection. This was the case for the vaccine against Yersinia pestis, the etiological agent of plague. Although the presence of the disease has been noted for more than a thousand years, it was not until a little more than 100 years ago that the organism that caused this zoonotic disease was first identified. The whole-cell plague vaccine apparently only provided limited protection against a virulent challenge, while being more immunoreactive. Further understanding of the pathogenesis and identification of virulence factors of Y. pestis led to the development of a subunit vaccine that appears to offer a more robust and long-lasting protection in animal models of plague. The current vaccine candidate has gone through initial human clinical trials in both the United States and United Kingdom. Nevertheless, we still do not completely understand how protection is mediated by the vaccine candidate. As we learn more about the immune response in the host, and in particular the innate immune response that may alter the development of protective immunity, this provides us with additional parameters to evaluate and improve the efficacy of a vaccine candidate. We will present examples how immunomodulation of the innate immune system can augment the immune response to vaccine candidates, leading to an enhancement of their efficacy in their respective animal models.

Biography

Amemiya received his doctoral degree from Rutgers University in Microbiology in 1973. He did his postgraduate 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.

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