OMICS Group Conferences
Accelerating Scientific Discovery

2nd International Conference on
Vaccines and Vaccination

August 20-22, 2012 Hilton Chicago/Northbrook, USA

Subversion of the innate immune response of the host by gram-negative pathogens

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Understanding the pathogenesis of a pathogen and its interaction with the host's immune system will help toward development of potential vaccines against the pathogens. We have been studying the latest vaccine against Yersinia pestis that has been through initial human clinical trials although the exact mechanism how it protects the host is not completely clear. Studies in murine and nonhuman animal models had shown that the new plague vaccine, which consists of a fusion of two Y. pestis proteins, mediates protection primarily through antibodies against the vaccine. Nevertheless, we previously found that the innate immune system is required for protection in vaccinated mice. This led us to examine more closely how the innate immune system is affected by the introduction of the pathogen into the host. We asked what does the innate immune system of an animal see when the pathogen is prepared as in a challenge study. Previous reports on the activation of the innate immune system have been with purified lipopolysaccharides or heat-inactivated whole-cells. We have examined the activation of either Toll-like receptor (TLR) 2 or TLR4 in transfected HEK cells, two of the TLRs likely to be activated on introduction of a gram-negative pathogen into the host, with live Y. pestis cells. We found that depending on the temperature of growth, either TLR2 and TLR4 or only TLR2 was activated. We have extended our studies to include other gram-negative pathogens and found a common pattern of TLR activation.

Biography

Dr. 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.

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