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Altered vaccine immune responses to different Toll-Like receptor activators

Although it has been demonstrated that vaccination can protect the health of certain populations against specific pathogens, the creation of an effective vaccine against threatening diseases is not always easily obtainable. An example of this can be applied to a vaccine against *Yersinia pestis*, is the pathogen that causes plague and has killed millions of people throughout the ages. There are three clinical presentations of plague: septicemic, bubonic and pneumonic. The latter form of the disease is the most severe because it is almost 100% fatal without immediate medical treatment. It has been only a little over 100 years that *Y. pestis* was identified as the etiological agent that caused plague, and another 100 years before a potential safe vaccine candidate for human use has been tentatively identified. In between these two discoveries, an inactivated whole-cell vaccine was used against plague, albeit with limited efficacy. The use of a formalin-inactivated, whole cell vaccine had been discontinued in the mid-nineties because it was apparently not protective against the pneumonic form of the disease which is the most deadly form of plague. In addition, the inactivated whole cell vaccine was very immunoreactive and considered not safe for human use. The current plague vaccine candidate consists of two pathogen proteins: a capsule protein (F1), and a virulence protein (V-antigen). It has been shown to be efficacious in both mouse and nonhuman primate models of pneumonic plague, and the vaccine has been through two human clinical trials. Nonetheless, efforts are still being undertaken to enhance the immune properties of the vaccine so that it can provide a more robust protective response against the disease. We will present examples of some of the efforts to achieve these goals for the latest vaccine candidate.

Biography

Kei Amemiya received his Doctoral degree from Rutgers University in Microbiology in 1973. He did his Post-graduation studies in Gene Regulation in the Laboratory of Lucy Shapiro at Albert Einstein College of Medicine, Bronx, NY. He joined 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 US 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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