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The Innate Immune Response in Pathogenesis and Vaccination

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Although pathogenesis and vaccination appears to be unrelated topics, there is a common link between the pathogen and vaccination and that is the innate immune response in the host. Pathogens have evolved over time to avoid activating the host defenses, while the host has developed pathogen recognition receptors (PRR) to warn the host of the presence of the pathogen. This latter idea was first proposed by Charles Janeway in 1989, which also stated that rather than the host immune system recognizing each individual pathogen the PRR would respond to different classes of molecules or pathogen associated molecular patterns (PAMPs), such as lipopolysaccharides or certain types of DNA or RNA. We now know that there are multiple extracellular PRR in mammals that recognize different PAMPs. Furthermore, we find further elaboration of the innate immune system because of the discovery of cytosolic systems that recognize molecular components of pathogens, especially those that might be associated with intracellular pathogens, like flagellin or lipopolysaccharides. Pathogens have evolved to avoid activating the host's Toll-like receptors (TLRs), the most well studied type of PRR. One example is TLR4 which recognizes lipopolysaccharides that are associated with the outer cell membrane of Gram negative bacteria. TLR4 is one of the more complex TLRs because there are more than one signal transduction pathway that can be activated in response to the LPS of the pathogen. It appears that some Gram negative pathogens have an altered LPS or assembled structure that does not activate or sub-optimally activates TLR4. We will show examples of these phenomena, and discuss a vaccination study, where we took advantage of another TLR ligand as an adjuvant to enhance the host immune response to the vaccine through an endosomal expressed PRR.

Biography

Kei Amemiya is a Principal Investigator in the Bacteriology Division at the US Army Medical Research Institute of Infectious Diseases. He has been at USAMRIID since 1999 and has been involved in developing vaccine candidates against plague, glanders and melioidosis. His interest is in studying the host's immune response to the pathogen and vaccines in small and large animal models. Before coming to USAMRIID, he was at the National Institutes of Health and Georgetown University, where he was studying the host response to bacterial and viral pathogens or autoimmune diseases in humans.

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