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Transcriptional profiling of human Peripheral Blood mononuclear cells exposed to *Bacillus anthracis in vitro*

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Abstract

Dependable and efficient diagnosis of *Bacillus anthracis* (BA) has long been a major concern for the care givers. Nonspecific symptoms during early illness often misguide the diagnosis; thereby jeopardize the proper therapeutic intervention. It is, therefore, crucial to understand the initial events that take place in a host soon after the onset of infection. The present study examines the PBMCs challenged by the BA spores in vitro and subsequently cultured the cells for 2 hrs, 4 hrs, 6 hrs, 8 hrs and 24 hrs, respectively. We observe rapid transcriptomic elevation of a number of cytokines, chemokines and other uptake receptors concurrently with the infection onset. Delayed responses to the BA induced assault include gradual attenuation of the genes linked with pathogenic uptake, such as MyD88 and TLR4 putatively driving the host towards greater vulnerability. The signs of altering host defenses nevertheless are evident immediately after the exposure to the BA spores. The pathogenic insult selectively induces some of the key transcriptomic candidates of apoptotic pathways regulated by the toll-like receptors and the caspase cascade; and suppresses the transcripts related to the p38 MAPK-dependent pathways. The T-cell receptors and CD3-mediated antigenic recognition processes are possibly restrained. In conclusion, BA challenges both innate and adaptive immunity processes and their key interfaces during the early course of infection. We identified a host of early targets across the networks and pathways primarily related to chemotaxis and apoptosis of immune cells that can potentially facilitate next generation anthrax prevention strategy.

Biography

Marti Jett is the Director, Integrative Systems Biology and Chief Scientist, Systems Biology Enterprise USAMEDCOM, USACEHR. Dr. Jett has more than 20 years research leading several key research DOD project as the principal investigator.