**Bacillus anthracis** and complement

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*Bacillus anthracis* is the causative agent of anthrax. Little was known regarding the interactions between this bacterium and the complement system, a critical aspect of the immune responses. Studies from our laboratory showed that activation of the classical complement pathway (CCP) by *B. anthracis* was the primary mechanism for spore phagocytosis by macrophages. Surprisingly, CCP activation on the spore surface was independent of immunoglobulin G or M, and was primarily mediated by a spore surface glycoprotein BclA. Purified recombinant BclA proteins were able to directly bind C1q, the ligand recognition component of the C1 complex. Spores from an isogenic BclA deletion mutant had significantly less CCP activation compared with spores from the parent strain. Furthermore, BclA expressed on the surface of a heterologous host surface was able to significantly increase binding to C1q as well as CCP activation on the surface of the bacteria. Thus, we have identified a novel ligand for C1q that likely activates CCP via a previously uncharacterized mechanism. Infection studies using mouse models indicated that BclA was recognized by C1q in vivo and that the interaction had biological consequences with respect to bacterial survival and the progression of infections. In addition, *B. anthracis* spores were also found to interact with other components of the complement system. Thus a theme is emerging that *B. anthracis* spores have a sophisticated mechanism to manipulate the complement system. These findings will have significant implications to the *B. anthracis* field as well as the field of host-pathogen interactions in general.

**Biography**

Yi Xu has completed her Ph.D. from University of Texas and postdoctoral studies from Texas A&M. She is currently an associate Professor in the Center for Infectious and Inflammatory Diseases, Institute of Biosciences and Technology, Texas A&M Health Science Center, Houston, Texas. She has published more than 25 papers in reputed journals in the area of bacterial pathogenesis and pathogen-host interactions.

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