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Contribution of host thrombosis and fibrinolytic cascades to the pathogenesis of gram-positive infections

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Abstract

Exploiting or usurping host pathways are critical elements to proper establishment of infections *in vivo* by many human pathogens. Gram-positive *Staphylococcus aureus* (*S. aureus*) and *Streptococcus pyogenes* are among the deadliest pathogens to humans and clinical interest into mechanisms that govern their infectivity has increased, partly due to their increasedantibiotic resistance. Exemplified here, coagulase-positive *S. aureus* is one of these emerging 'Superbugs' that hijacks the clotting cascade to effectively limit phagocytic immune cell infiltration. *In vivo* bacterial colonization occurs at sites of endothelial damage and complex fibrin-networks quickly develop. *S. aureus*forms thisprotective barrier throughexpression of a redundant virulence factors, namely staphylocoagulase and von Willebrand factor binding protein (vWbp) that bind the zymogen prothrombin, thereby forming a prothrombin-activator complex that cleaves fibrinogen to fibrin. Recently, we took advantage of this unique mechanism of prothrombin recruitment to detect *S. aureusin vivo* in a mouse model of endocarditis via noninvasive fluorescence and PET-CT methods (Panizzi *et al*.,*Nat Med*. 2011 August 21; 17(9): 1142–1146) by systemic injection ofspecifically designed prothrombin analogs. Use of strains of *S. aureus* deficient in one or both bacterial prothrombin activators, provided the first *in vivo* validation to the importance of these factors in the establishment of endocarditis. Much unlike *S. aureus*, *Streptococcus pyogenes* harnesses host fibrinolytic pathways to mediate potential dissemination of the pathogen *in vivo*. The mechanisms employed by these pathogens will be discussed to highlight this interplay between the misregulation of host systems and pathogens during infection.

Biography

Peter Panizzi, Ph.D. is an Assistant Professor at Auburn University. He received his doctorate from Vanderbilt University in 2004 and completed at post-doctoral fellowship at the Center for Systems Biology at the Massachusetts General Hospital from 2007-2010. He has co-authored >32 high-impact articles and was awarded a NIH pathway to Independence Grant (K99R00) from NHLBI before coming to Auburn.His research focus is to better understand how certain bacteria are able to cause human disease and in turn use this knowledge against the microbe to detect specific sites of infections and identify casual pathogen.