

Research Theme: Infection and Immunity
Research Project Title: Mechanisms of <i>E. faecalis</i> Immune Modulation in Macrophages
Principal Investigator/Supervisor: Assoc/Prof Kimberly Kline (SBS/SCElse)
Co-supervisor/ Collaborator(s) (if any):
Project Description
<p>a) Background:</p> <p>Inflammation is a critical component of nearly every human disease. It is also a vital part of the protective response to most pathogens, and it is therefore highly advantageous to bacterial pathogens to have evolved mechanisms to suppress inflammation. So it is not surprising that diverse bacteria have evolved sophisticated mechanisms to interfere with the signaling pathways involved in inflammation. Understanding the mechanisms by which pathogens suppress inflammation will inform not only our treatment of those particular pathogens/diseases by identifying potential immunotherapeutic targets, but may also enable the development of novel approaches to the suppression of the inflammatory components of many other diseases</p> <p>b) Proposed work:</p> <p>We have defined experimental systems in which <i>Enterococcus faecalis</i> modulates the normal host anti-microbial and inflammatory response to infection. Our goal is to identify the different molecular mechanisms involved in these effects.</p> <p>The aim of the project is to determine the host and bacterial mechanisms by which previously identified bacterial factors contribute to the ability of <i>E. faecalis</i> to suppress the LPS-induced NF-κB activation in macrophages <i>in vitro</i> using cell culture models. <i>In vitro</i> findings will then be validated in a variety of <i>in vivo</i> mouse infection models, including urinary tract infection, wound infection, and gastrointestinal infection. Methodologies that may be employed in these studies include bacterial and mammalian genetics, tissue culture, animal infection, RNA-sequencing, metabolomics, fluorescent and super-resolution microscopy, flow cytometry, high content screening, and more.</p>
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