The role of intracellular recognition molecules like NODs expressed by cells of the female reproductive tract (e.g. Fallopian tube epithelium) in the response to chlamydial infection, or any STI, remains poorly understood. A suboptimal innate immune response may result in a permissive environment for pathogen colonization, whereas an over-exuberant response will cause excessive inflammation and tissue damage. Modulation of the host response to infection is an attractive alternative or adjuvant approach to antibiotic therapies in treatment of genital tract infections. Genome sequence analysis has revealed that Chlamydia possesses numerous novel genes that might be involved in the manipulation of the host cells. The infected cells often display altered metabolic, immunological and cell biological characteristics, however, at the same time the microbes have to maintain the integrity and viability of host cells before completing their own intracellular replication. To achieve this goal chlamydiae have evolved the ability to both prevent the infected cells from undergoing apoptosis induced by intracellular stress and to protect these cells from recognition and attack by lymphocytes. Analysis of C. trachomatis genome had identified more than two dozens of open reading frames encoding proteins with potential proteolytic activity. Some of these proteases may be used to target host cell proteins because some proteins are cleaved and/or degraded in infected cells. The attacked host proteins include transcriptional factors, pro and anti apoptotic proteins, DNA repairing enzymes, cyclins and cytoskeletal protein. However, survival strategies of Chlamydia at the tissue level and the relevance of these findings in disease pathogenesis have yet to be determined. Since till date a Chlamydia vaccine is unavailable, a better definition of human immune response along with Chlamydial survival strategies needs to remain an important research priority if we are to develop a vaccine against C. trachomatis infection which has protective and not deleterious effects.

Emerging functions for the Staphylococcus aureus RNMe: Its relationships with antibiotic resistance, immune evasion and toxic peptide secretion

Staphylococcus aureus is a serious pathogen for animals and humans, being one of the most frequently isolated bacteria in hospital-associated infections and also causing diseases in the community. To coordinate the expression of its numerous virulence genes for growth, survival and adaptation, S. aureus uses various signalling pathways that include two-component regulatory systems, transcription factors, and hundreds of regulatory RNAs (sRNAs). Biological roles have only been determined for a handful of these sRNAs, including cis, trans, and cis-trans acting RNAs, some internally encoding small, functional peptides and others possessing dual or multiple functions. Recent investigations from the author's lab have identified as RNA that influence antibiotic resistance in S. aureus, a novel sophisticated translational control of an mRNA by two differentially expressed sRNAs that ensures supervision of host immune escape by S. aureus, and a novel toxin-antitoxin system producing membrane and secreted toxic peptides destroying host cells and competing bacteria with dissimilar strengths.