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Despite the identification of many strategies used by bacteria to survive in the host and the host mechanisms to evade the pathogen, it is still unclear why host system is not getting success in pathogen clearance. Many researchers have focused the study of bacterial proteins and other component for its virulence and the strategies used by *M. tuberculosis* to survive within host cells. Our own lab has been focused towards characterizing host-pathogen interactions by elucidating the interaction of *M. tuberculosis* secretory antigens with dendritic cells (DCs) and the outcome of these interactions on the T cell responses to mycobacteria. These include immune responses mediated by *M. tuberculosis* secretory antigens such as CFP-10 that differentiates and activates DCs and its precursors in a way that they suppresses Th1 responses required for the elimination of bacteria. Continuous secretion of such proteins from the phagosomal complex of macrophages infected with live *M. tuberculosis* would differentiate the DC precursors and induce terminal maturation of immature DCs recruited at the site of infection into a phenotype that is unable to mount inflammatory and protective responses to the secondary interaction with the released mycobacteria. A potentially Th1 response turns into a Th0 response from antigen activated DCs with the progression of infection. This change in response, in turn, favors the pathogen. Thus, above results indicate the possible physiological roles and mechanisms by which *M. tuberculosis* antigens induce suppressor responses at the sites of infection and promote the survival of bacteria. Thus, expression of antigens such as CFP-10, as the infection progresses in time, could be a strategy used by *M. tuberculosis* toward immune evasion and have an important bearing on the immune response mounted against tuberculosis early in the infection.

We have also studied *M. tuberculosis* genes that were expressed inside infected macrophages at different times post-infection and characterized the immune responses mediated by these genes. The modulations in DC and macrophage functions by these
genes as they are expressed during the course of infection were studied. Further, the ability of these genes to alter T cell priming and functions was also investigated. In parallel, the intra-cellular mechanisms employed by the antigens in the modulation of immune responses were also studied.

We and others had characterized many *M. tuberculosis* antigens to regulate its survival inside macrophages and dendritic cells. This time we decided to characterize host genes which might be a good friend of *M. tuberculosis*. A recent report has identified genes in human macrophages that regulate the survival of *M. tuberculosis* wherein inhibiting xenophagic pathways and the formation of autophagosomes as an increase in the levels of LC3 II upon the silencing of some target genes was observed (Kumar et al., 2010).

Keeping the above background in mind, the work embodied in this thesis focused on the role of genes of these two pathways in modulating DC function with respect to *M. tuberculosis* infection by employing pathway specific siRNA libraries. Our results identify a set of as yet unreported genes that are targeted by *M. tuberculosis* in modulating the activation and function of DCs with respect to cytokine secretion and pro-inflammatory T cell responses, anti-defense mechanisms such as autophagy and reactive oxygen species generation.