Tata Institute of Fundamental Research
Survey No. 36/P, Gopanpally Village, Serilingampally, Ranga Reddy Dist., Hyderabad - 500107

TIFR-UoH (Life Sciences) Seminar Series

HIV-Mycobateria coinfections: the cellular and molecular events behind the emerging threat

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We are working on two human pathogens: Mycobacterium tuberculosis (the tuberculosis causing bacteria) and Human Immunodeficiency Virus (HIV). Besides studying molecular pathogenesis of these pathogens, we are also engaged in deciphering the coalition between mycobacteria and HIV, the synergistic pandemic of which is a major health concern in India. I shall be sharing some of the ongoing work on M.tb and HIV, but shall elaborate on some of the recent observations from our lab on mycobacteria-HIV coinfection. Apart from pathogenic mycobacteria, ubiquitously present environmental mycobacteria are one of the most common co-infections in HIV patients and a serious co-epidemic as there is an increased risk of disease progression from both the pathogens in the individuals concurrently infected with mycobacteria and HIV. These seemingly harmless non-pathogenic mycobacteria, establish as opportunistic infections adding to HIV associated complications, re-emergence of latent infections and drug resistance. The mechanisms underlying the early events by which opportunistic mycobacteria establish infection in macrophages influencing HIV infection are unclear. We undertook a comparative proteomics approach to understand the same and identified differential distribution of host and pathogenic proteins in phagosome-enriched fractions from Mycobacteria mono- and HIV-Mycobacteria co-infected THP-1 cells by LC-MALDI-MS/MS. The validation of the proteomics data showed that HIV co-infection helped the survival of non-pathogenic mycobacteria by obstructing phagosome maturation, promoting lipid biogenesis and increasing intracellular ATP equivalents. In turn, mycobacterial co-infection up regulated purinergic receptors in macrophages that are known to support HIV entry, explaining increased viral titers during co-infection. The intracellular Mycobacteria displayed down-regulation of toxin-antitoxin (TA) modules, up-regulation of cation transporters, Type VII (Esx) secretion systems, proteins involved in cell wall lipid or protein metabolism etc, during co-infection. The bearings of these mycobacterial processes on HIV propagation during co-infection were validated using mutants of mycobacteria. The analyses revealed mycobacterial factors that possibly via modulating the host environment, increased viral titers during co-infection. Some of these observations will be shared.

Wednesday, Mar 28th 2018

11:30 AM (Tea/Coffee at 11:00 AM)

Auditorium, TIFR-H