Detection of ER stress after infection of human macrophages by *Mycobacterium tuberculosis*

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Post infection, macrophages, the first cells in the lungs that propel defense against pathogen invasion and play crucial activity in the onset and maintenance of immune responses against Mtb. The macrophages play this crucial defense role by phagocytosis, which have the macrophages ''eat'' up the Mtb *bacilli*. Macrophages therefore become infected with mycobacteria and may undergo apoptosis (programmed cell death) to destroy pathogens and prevent further spreading. Apoptosis, which results in the elimination of Mtb can be triggered by endoplasmic reticulum (ER) stress which is the physiological or pathological processes that disturb protein folding in the endoplasmic reticulum caused by the phagocytosis of the Mtb *bacilli* by the macrophages. The dysregulation of ER homeostasis can cause chronic diseases in humans and it is crucial to study ER stress using mammalian cells to understand ER-stress related diseases such as Tuberculosis. Here, we studied the ER stress induction and the extent of ER stress induction using human monocytes derived macrophages (hMDMs). We used the ER stress inducers tunicamycin and thapsigargin, and also infecting the macrophages with different doses of Mtb and analysing CHOP and ATF6-alpha expression by western blot. This indicated that both inducers triggered CHOP activation, that a low dose of Mtb suppressed the expression of these ER-stress markers in most donors, and that infection with a higher dose of Mtb stimulated expression of both markers in 4 out of 6 donors. Alternatively, live microscopy was also performed on Raw macrophages and 16HBE epithelial cells after transfection with the ER stress plasmid sensor pEGFP-XBP1dDBD-STOP-tagRFPt and stimulation with tunicamycin and purified protein derivative of tuberculin (PPD). We have here confirmed the detection of ER-stress in human monocyte derived macrophages using positive inducers, and shown that low doses of Mtb decreases induction of ER-stress whereas, high dose of Mtb induces ER- stress

Speaker Biography

Samuel Eguasi Inkabi has completed his MSc in Medical Biology from Linkoping University, Sweden. He also holds a Bachelor's in Biochemistry from Kwame Nkrumah University of Science and Technology, Ghana. His research focuses on infectious diseases, cancer and avian genetics. He has co-authored a publication and authored peer review papers in reputed journals.

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