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

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Statement of the Problem: Post infection, macrophages, the rst cells in the lungs that propel defence against pathogen invasion and play crucial activity in the onset and maintenance of immune responses against Mtb. e macrophages play this crucial defence 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. e 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 di erent doses of Mtb and analyzing CHOP and ATF6-alpha expression by western blot. is 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 a er transfection with the ER stress plasmid sensor pEGFP-XBP1dDBD-STOP-tagRFPt and stimulation with tunicamycin and puri ed protein derivative of tuberculin (PPD). We have here con rmed the detection of ER-stress in human monocyte derived macrophages using positive inducers, and shown that low doses of Mtb decreases induction of ER-strress whereas high dose of Mtb induces ER-stress.

Biography

Samuel Inkabi holds an MSc 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 a review paper in reputed journals.

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