

Mycobacterium Tuberculosis-Microvesicles the Transporter of Proteins

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Abstract

In current circumstance Tuberculosis (TB) is a fundamental issue of mortality and dismalness which is achieved by *Mycobacterium tuberculosis*. Late WHO report of 2015 declared TB as ruinous as HIV according to overall passing rate. *M. tuberculosis* is a convincing intracellular organism of alveolar macrophages of lungs where it resides inside phagosomes. *M. tuberculosis* can control blend among phagosome and lysosome due to these bacilli is obligated for hosing the maturation of phagosome which ensures its life expectancy. The initial step during bacterial infection is having microorganism joint effort what start by various antigenic proteins of *M. tuberculosis*. During tainting bacilli releases distinctive mycobacterial antigenic sections from phagosome in minimal membranous vesicles. These vesicles are carrying on as transporter or carrier of various hurtful proteins which control have immune system.

Keywords: Microvesicles; Apoptosis; Tuberculosis; Innate immunity; Neutrophils

Introduction

In current situation Tuberculosis (TB) is an essential issue of mortality and grimness which is brought about by *Mycobacterium tuberculosis*. Late WHO report of 2015 announced TB as destructive as HIV as per worldwide passing rate [1]. *Mycobacterium tuberculosis* is a compelling intracellular microbe of alveolar macrophages of lungs where it lives inside phagosomes. *Mycobacterium tuberculosis* has the ability to restrain combination among phagosome and lysosome because of these bacilli is liable for hosing the fermentation of phagosome which guarantees its life span. The first step during bacterial disease is having microorganism collaboration which starts by different antigenic proteins of *Mycobacterium tuberculosis*. During contamination bacilli discharges different mycobacterial antigenic segments from phagosome in little membranous vesicles [4]. These vesicles are carrying on as carrier or transporter of different harmful proteins which regulate have insusceptible framework in support of its.

Immunomodulatory compounds

The microvesicles are the heterogeneous membrane bound nanovesicles which contains proteins, phospholipids and LPS and also contains some virulence factor like adhesins, toxins and immunomodulatory compounds that are vital for the pathogenesis. Also the composition of MVs depends upon the cell type from which they are originated [2]. MVs are produced by the process of budding and fission of membrane vesicles from the plasma membrane. MVs are produced by most of the cells and are involved in the various intercellular communications by acting as nanovectors and transferring proteins, mRNA, microRNAs and also have significant role in immune modulation.

Rab proteins

Rab proteins are involved in the endosome trafficking and have crucial role in the pathogenesis of *M. tuberculosis*. It is also seen that MVs which are released from circulatory cells have functional role in immunity and coagulation. Environmental condition is also an important factor in the production of these vesicles and it had been seen that *M. tuberculosis* MVs production increases under the iron limitation and restriction [3]. MVs that are released at such condition contain mycobacterium which helps in the replication of iron starved mycobacteria.

Proinflammatory response

MVs infected from the bacilli stimulate both innate and adaptive immune receptors and contributes to the genesis of CD4+ T-cell response. Certain MVs are also released by the neutrophils and it is seen that innate immune response is accompanied when these neutrophils migrate at the site of infection and the infected neutrophils has the ability to influence the survival of *M. tuberculosis* within macrophage. Although neutrophils are not able to kill the virulent *Mycobacterium tuberculosis* but they are important in granuloma formation and suppression of infection. *M. tuberculosis* infection causes rise in the neutrophil apoptosis and which elicits proinflammatory response in macrophages [4].

Apoptotic MVs

The incessant study, MVs can be used as biomarkers in disease, therapeutic targets, drug delivery etc. One major advantage of using MVs as biomarker is that it can be easily accessible in the biological fluids. It has been known that apoptosis is involved in the development of autoimmune disease and the apoptotic MVs can hasten autoimmune response in the specific tissue or organs. Apoptotic MVs are said to be the combination of apoptotic bodies and microparticles which are mainly released by the infected macrophages and these carries several mycobacterial antigens that stimulate the CD8+ T-cells response and provide protection from *Mycobacterium tuberculosis*.

Micro vesicles

The impact of MVs can be studied by measuring the production of human and murine macrophages in mice during mycobacterium infection. MVs, released from the infected alveolar macrophages contribute to the disruption of epithelial cells which later on facilitate formation of granuloma, which in turn stimulate the production of Tumor Necrosis Factor (TNF) and enhance the transfer of antigens to Antigen Presenting Cells (APCs). Also there are certain proinflammatory cytokines and chemokines are released from the infected macrophages. Further research in the field of microvesicles need to be done for the future research and development work as we know that microvesicles have easy accessibility in the biological fluids. On the basis of this research area may be best possible way in the drug discovery [5].

Conclusion

Tuberculosis is global threat to human health due to its insidious nature of bacilli has drawn profound interest of many researchers. Bacilli contain several antigenic proteins which involve in pathogenicity which actively participate in host pathogen interaction. Bacilli uses host membranous trafficking mechanism to transport its virulent proteins which modulate host immune system. During mycobacterial infection various MVs are released which have distinct function in the transfer of cargo from donor.

References

1. Meena PR, Meena LS (2015) Fibronectin binding protein and Ca²⁺ plays an access key role to mediate pathogenesis in *Mycobacterium tuberculosis*: An Overview. *Biotechnol Appl Biochem* 63: 820-826.
2. Kumari P, Meena LS (2014) Factors Affecting Susceptibility to *Mycobacterium tuberculosis*: a Close View of Immunological Defence Mechanism. *Appl Biochem Biotechnol* 174: 2663-73.
3. Meena LS, Rajni (2010) Survival mechanism of pathogenic *Mycobacterium tuberculosis* H37Rv. *FEBS J* 277: 2416-27.
4. Govender VS, Ramsugit S, Pillay M (2014) *Mycobacterium tuberculosis* adhesins: Potential biomarkers as anti-tuberculosis therapeutic and diagnostic targets. *Microbiology* 160: 1821-1831.
5. Ramsugit S, Pillay M (2016) Identification of *Mycobacterium tuberculosis* adherence-mediating components: a review of key methods to confirm adhesin function. *Iran J Basic Med Sci* 19: 579-584.