

In-Silico Characterization of Rv0169 Gene Product from *Mycobacterium tuberculosis* †

G. Roni Mrudula ¹, Neesar Ahmed ^{1,*}

¹ School of Life Sciences, B S A Crescent Institute of Science and Technology, Vandalur, Chennai-600048, Tamil Nadu, India

* Correspondence: neesar.sls@crescent.education;

† Presented at Virtual symposium to observe World Antimicrobial Awareness week “Applications of biotechnology and microbiology with special emphasis on Antimicrobial resistance”, 18-24 November 2020, Chennai, India

Received: 10.11.2020; Revised: 15.11.2020; Accepted: 17.11.2020; Published: 10.01.2021

Abstract: *Mycobacterium tuberculosis* is a harmful tuberculosis-causing bacillus, a malady that causes many deaths worldwide every year. 33% of the total population is evaluated to be contaminated with *Mycobacterium tuberculosis* (Mtb). Tuberculosis is, in this manner the main source of irresistible mortality around the world, answerable for in excess of 8 million new cases and 2.9 million deaths every year. The accomplishment of *Mycobacterium tuberculosis* (Mtb) as a pathogen settles upon its capacity to develop intracellularly inside the host macrophages. Mtb cell membrane has some of the proteins that help in the interaction between host and pathogen. Those proteins are called membrane proteins. Those membrane proteins of Mtb are mainly involved in: Invasion, adsorption, virulence. Specific protein and gene functions for *Mycobacterium tuberculosis* are characterized to clarify its pathogenesis role in humans and to regulate tuberculosis. Our current research is Structural prediction, analysis of protein-protein interactions, cloning, and characterization of RV0169 from *Mycobacterium tuberculosis* (H37Rv) strain. So that helps to immunomodulate the membrane proteins. mce 1A is the *Mycobacterium tuberculosis* gene product, which can be annotated as the RV0169 454 amino acid long basic protein. As the capacity of the mce 1A is obscure, however, thought to be associated with have cell intrusion (passage and endurance inside macrophages).

Keywords: *Mycobacterium tuberculosis*; structure prediction; protein–protein interactions; cloning; characterization; mce 1A; pathogenesis.

© 2021 by the authors. This article is an open-access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

Funding

This research received no external funding.

Acknowledgments

This research has no acknowledgment.

Conflicts of Interest

The authors declare no conflict of interest.