J Clin Cell Immunol 2018, Volume: 9
DOI: 10.4172/2155-9899-C4-058

10th World Congress and Expo on
Immunology, Immunity, Inflammation & Immunotherapies
October 19-20, 2018 | New York, USA

Mycobacterium tuberculosis host cell interaction: Role of latency associated protein Acr-1 in differential modulation of macrophages

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Mycobacterium tuberculosis (M.tb) contrives intracellular abode as a strategy to combat antibody onslaught. Additionally, to thrive against hostile ambiance inside host macrophages, the pathogen inhibits phagolysosomal fusion. Finally, to further defy host cell offensives, M.tb opts for dormant phase, where it turns off or slows down most of its metabolic process as an added stratagem. While M.tb restrains most of its metabolic activities, surprisingly latency-associated alpha-crystallin protein (Acr-1) is expressed most prominently during dormancy. Interestingly, several previous studies described the potential of Acr-1 to induce a robust immuno-prophylactic response in the immunized host. It is intriguing to comprehend the apparent discrepancy that how the microbe M.tb overexpresses a protein that has the potential to prime host immune system against the pathogen itself. Keeping this apparent ambiguity into consideration, it is imperative to unravel the intricacies involved in the exploitation of Acr-1 by M.tb during its interaction with host immune cells. The present study suggests that Acr-1 exhibits a diverse role in the maturation of macrophages (MΦs) and related immunological responses. The early encounter (pre-exposure) of bone marrow-derived immune cells (during their differentiation to MΦs) with Acr-1 (AcrMΦpre), results in hampering of their function. The pre-exposure of naïve MΦ with Acr-1 modulates expression of TIM-3 and IL-10 as well. In contrast, exposure of fully differentiated MΦ to Acr-1 results in their activation. Furthermore, Acr-1 mediated activation of MΦ results in the induction of Th1 and Th17 phenotype by activated T lymphocyte.

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