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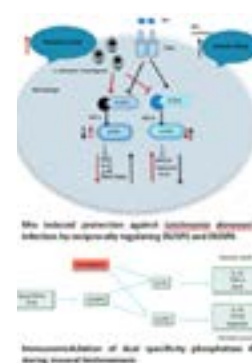
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Regulation of dual specificity phosphatases during *Leishmania donovani* infection: Critical role of immunomodulators

Leishmania donovani resides within the host macrophages by dampening host defense mechanisms and thereby it modulates the host cell functions for its survival. Multiple host cell factors determine who wins the race during the interplay between the host and the parasite. Role of dual specific phosphatases (DUSPs) are implicated in various pathological conditions. However, the reciprocity of these DUSPs was unknown in *Leishmania donovani* infection in a susceptible model. Here, we show that *Mycobacterium indicus pranii* (Mw), an immunomodulator, reciprocally regulates DUSP1 and DUSP6 through Toll-like Receptor 4 pathway. Association of Protein Kinase C- β with DUSP6 increases by Mw treatment resulting into decreased Interleukin-10, phosphorylation of ERK1/2 and Arginase I, whereas, Mw treatment decreases association between Protein Kinase C- ϵ and DUSP1 resulting into increased Interleukin-12, phosphorylation of p38 and inducible nitric oxide synthase expression. In another study, we found that *Leishmania donovani* significantly reduced the expression of DUSP4. Glycyrrhizic Acid (GA), an immunomodulator, already known to suppress *Leishmania donovani* infection, found to up-regulate DUSP4 expression during *Leishmania donovani* infection. On the other hand, GA fails to increase Th1 cytokine production and decrease Th2 response during DUSP4 knock-down condition suggesting the key role of DUSP4 while giving protection during *Leishmania donovani* infection. Therefore, we establish that DUSP6, DUSP1 and DUSP4 can be promising therapeutic targets to provide better treatment support to the patients suffering from visceral Leishmaniasis.

Recent publications

1. Bandyopadhyay S, Bhattacharjee A, Banerjee S, Halder K, Das S et al. (2015) Glycyrrhizic acid-mediated subduement of myeloid derived suppressor cells induces anti-leishmanial immune responses in a susceptible host. *Infection and Immunity*. 83(12): 4476-4486.
2. Bandyopadhyay S, KarMahapatra S, Paul Chowdhury, B Jha, M K Das et al. (2015) TLR2 targeted rectification of impaired cd8+ t cell functions in experimental *leishmania donovani* infection reinstates host protection. *PLoS ONE*. 10(11): e01428 Doi: 10.1371/ journal.pone.0142800.
3. Jawed J J, Majumder S, Bandyopadhyay S, Biswas S, Parveen S, Majumdar S (2016) SLA-PGN-primed dendritic cell-based vaccination induces Th17-mediated protective immunity against experimental visceral leishmaniasis: crucial role of PKC β . *FEMS Pathogen & Disease*. 74(5). pii:ftw041. Doi: 10.1093/femspd/ftw041.
4. Bhattacharjee A, Majumder S, Das S, Ghosh S, Biswas S, Majumdar S (2016) *Leishmania donovani*-induced prostaglandin E2 generation is critically dependent on host toll-like receptor-2-cytosolic phospholipase A2 signaling. *Infection and Immunity*. 84(10): 2963-2973.
5. Das S, Chowdhury B P, Goswami A, Parveen S, Jawed J J, Pal N, Majumdar S (2016) *Mycobacterium indicus pranii* (MIP) mediated host protective intracellular mechanisms against tuberculosis infection: Involvement of TLR-4 mediated signaling. *Tuberculosis (Edinb)*. 101:201-209. Doi:10.1016.



Biography

Subrata Majumdar has his expertise on cell signalling and immunology in infectious disease. He is a Pioneer in establishing ceramide pathway in visceral leishmaniasis. Through studying signalling mechanism, he has established for the first time the reciprocal regulation between phosphatases and kinases during leishmaniasis. He is a Recipient of many national and international awards including many prestigious awards of United States.

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