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Direct evidence for role of anti-saliva antibodies against salivary gland homogenate of P. argentipes in modulation of protective Th1-immune response against Leishmania donovani

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Currently the main concerns regarding control of visceral leishmaniasis (VL) caused by L. donovani are immunosuppression, relating toxicity of antileishmanial drug and little development in appropriate vaccine and vector (P. argentipes) control. Reports available from ex vivo studies reflect significance of vector salivary gland homogenate (SGH) in reverting immunosuppression of infected VL subjects and as such the immunogenic nature of SGH can be a strategy to modulate immune system and antileishmanial function to enable immune response to control the disease. Several related studies also identified a better utility of vector anti-saliva antibodies in achieving such effects by an adoptive transfer approach instead of direct stimulation with SGH protein. However, conclusive evidences on VL cases are far beyond satisfactory to suggest role of SGH into modulation of host immune response in VL subjects in India. This study was undertaken to make comparison on change in cytokines (TH1 and TH2) response pattern and antileishmanial macrophage (Mϕ) function following stimulation of their PBMCs with SGH protein derived from P. argentipes sand fly vector for VL or anti SGH antibodies raised in rabbit. This study reports for the first time that L. donovani sensitized healthy subject demonstrates an up-regulated interferon-γ (TH1) and down regulated interleukin-10 (TH2) production following stimulation of their PBMCs by P. argentipes anti-saliva antibodies accompanied with an improvement in antileishmanial Mϕ function for nitric oxide (NO) production. Subsequent experiments suggest that P. argentipes based anti-SGH antibodies when used to stimulate LD infected PBMCs in healthy subjects resulted in better clearance of Leishmania amastigotes load compare to SGH protein. Possibly the immunogenic components of anti-saliva an antibody maintains the level of protective cytokine (INF-γ) and seems to restrict the infection by host protection by vector saliva.

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