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In-vitro analysis of cytokines responses of visceral leishmaniasis and pulmonary tuberculosis patients to homologous and heterologous antigen stimulation

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Background: Leishmaniasis-tuberculosis co-infection has been reported many times mainly in the east region of Africa, but little is known about the immunological interactions of the co-infection. A case control study was carried out to analyze in-vitro cytokines responses in visceral leishmaniasis (VL) patients and pulmonary tuberculosis (TB) patients.

Method: The cytokine profiles of 30 leishmaniasis patients, 30 tuberculosis patients and 10 healthy individuals were compared after stimulation with live Leishmania Promastigotes and BCG. Th-1 (IFN-γ and TNF-α), Th-2 (IL-10) and inflammatory cytokine IL-15 were measured in the supernatants of stimulated whole blood samples whole blood using ELISA.

Results: The concentration of Th-1 cytokines (IFN-γ and TNF-α) were significantly higher in the supernatants of stimulated whole blood of VL patients compared with TB patients mainly when stimulated by L.donovani antigen. Th-2 cytokine IL-10 was significantly produced by whole blood of TB patients particularly stimulated with BCG. A significant concentration was detected in stimulated whole blood of VL and TB patients compared by healthy controls.

Conclusion: The Th-1 cytokines expressions to the homologous antigen stimulation in visceral leishmaniasis patients were higher compared to the non-stimulated which suggests a strong adaptive response. Meanwhile, the Th-2 cytokine IL-10 expression to the homologous antigen stimulation in TB patients was higher than the non-stimulated which led to strong suppression the protective Th-1 cytokines expression. This finding suggests that a re-occurring TB infection may generate a weak protective immune response which could lead to a more persistent infection.

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Investigating existing drug targets for autoimmune disease treatment

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Autoimmune diseases are an emerging global health problem. The increasing incidence of these diseases worldwide, in addition to the economic and psychological burdens, calls for a sense of urgency for the development of efficient and robust therapeutic treatments. There has been abundant research on the discovery of drug for autoimmune diseases but researchers still have not been able to restore the pathogenic immune response back to the normal one; instead, the success has been only to curtail the symptoms and related inflammation. The major challenge in the repertoire of treatments for autoimmune diseases that subsists is resetting the immune system back to normal functions. The complex and multifactorial pathogenesis of autoimmune diseases has made the development of robust therapeutics sluggish. Even so, the ongoing studies in parallel with the development of cutting-edge technology have shown promising results in the realm of autoimmunity treatment. This talk aims to study the available drug targets that have shown some promising results along with the challenges they are currently facing, which include anti-TNF therapies and activation of the TNF-TNFR2 pathway, stem-cell transplantation, altering the balance between pathogenic and regulatory T-cells, and genetic modulation. For the effective, feasible and affordable treatment, tilting the pro-inflammatory response to anti-inflammatory and increasing the number of regulatory T-cell or Th2 -either by targeting cytokine IL-6 or construction of APL are favored treatments for autoimmune diseases. Since there is cross-talk among different cytokines, it is important to investigate more on the primary roles of individual cytokines in order to get more insight into the specific drug targets.

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