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Direct LPS recognition and activation of CD8+T cells via TLR4 in patients with rheumatoid arthritis

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Abstract

Background: Rheumatoid arthritis (RA) is an autoimmune disease characterized by abnormal immune responses to self-antigens. Though the pathogenesis of RA is not yet fully elucidated, it is known to be induced by environmental factors on a genetically susceptible background. Toll-like Receptors (TLRs) have been established to recognize specific patterns of microbial components and lead to systemic immune responses in Rheumatoid arthritis (RA). TLRs are expressed by cells in inflamed joints of RA patients and variety of endogenous TLR ligands is present within those joints. This study suggests that the over expression of TLR4 in CD8+T cells from RA patients may contribute to the abnormal immune activation of pro inflammatory cytokines and enhance the acute inflammation.

Methods: Eighty seven RA patients and 70 healthy donors participated in this study. Clinical variations like disease duration, number of actively inflamed joints, number, and type of bones deformities, CRP, RF, Anti-CCP, ESR (Erythrocyte Sedimentation Rate), and therapeutic interventions were recorded for each patient and DAS 28 scores were calculated with the help of the clinician. We analyzed the expression of TLR4 in transcript level by real-time PCR and protein level by flow cytometry in CD8+T cells of RA patients. Different cytokines level was checked after stimulation of CD8+T cells in TLR4 agonist. We have checked the MAP Kinase – ERK signal transduction in CD8+Tcells.

Results: A significant increase of TLR4 in both transcript level and protein level in patients with RA compared to healthy donors. We got a strong positive correlation between TLR4 expression and DAS 28 score. The ROC curve analysis confirmed the significance of TLR4 expression in RA patients. We found that TLR4 ligand responsiveness significantly increased the expression of different inflammatory mediators in purified CD8+T cells of RA patients compared with healthy individuals after in vitro stimulation. Our result showed TLR4 stimulation induces ERK phosphorylation in CD8+T cells.

Conclusion: In summary, our data suggest an increased expression of TLR4 in CD8+T cells play a major role in inflammation of RA patients.

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