Mohammad Kazemi Arababadi, J Antivir Antiretrovir 2014, 6:2
http://dx.doi.org/10.4172/1948-5964.S1.022

OMICS Group Conferences
Accelerating Scientific Discovery

4th World Congress on
Virology

October 06-08, 2014 Hilton San Antonio Airport, TX, USA

Decreased expressions of TLR9 and its signaling molecules in chronic HBV infected patients

Mohammad Kazemi Arababadi
Iran

Context: Toll like receptors (TLRs) play crucial roles in immune responses, especially innate immunity, against viral infections. The TLR9 recognizes intracellular viral double strand (ds)DNA which leads to the activation of nuclear factor kappa-light-chain-enhancer of activated B cells (NF- B) through the myeloid differentiation primary response 88 (MYD88) pathway. Defects in the expression of TLR9 and its signaling molecules may cause attenuated immune responses against hepatitis B virus (HBV).

Objective: The aim of this study was to determine expression levels of TLR9 mRNA along with MYD88, IRAK1, IRAK4, TRAF6, TRAF3, IRF7 and NF- B in the peripheral blood mononuclear cell (PBMCs) obtained from chronic HBV infected (CHB) patients.

Design: In this study, 60 CHB patients and 60 healthy controls were recruited and the expression of TLR9 and its downstream signaling molecules were examined by Real-Time polymerase chain reaction (PCR) techniques using beta-actin as a housekeeping gene.

Results: Our results showed that expression of TLR9, MYD88, IRAK4, TRAF6, TRAF3, IRF7 and NF- B in PBMCs of CHB patients were significantly decreased in comparison to healthy controls.

Conclusions: According to our results, it appears that CHB patients are unable to appropriately express genes in the TLR9 pathway which may impede immune responses against HBV infection. These results suggest a mechanism that may partially explain the fact that immune responses are disrupted in CHB patients.

Key words: Chronic HBV infection, TLR9, MYD88, IRAK1, IRAK4, TRAF6, TRAF3, IRF7, NF- B.

dr.kazemi@rums.ac.ir