Association beetwen arg753gln and arg677trp polymorphisms of tlr2 gene with active pulmonary tuberculosis in an indonesian population / Arto Yunowo Soeroto, Zulkarnain Dahlan, Cissy B. Kartasasmita, Ida Parwati.

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Abstrak

Background: Toll-like receptor is a pattern recognition receptor (PRR) that recognize pathogen-associated molecular pattern (PAMP) in a microorganism. Macrophages recognize the presence of mycobacteria through Toll-Like Receptor 2 (TLR2) and signaling further lead to the production of cytokines, both proinflammatory TNF-α, IL-1β, IL-6, IL-12, IL-15, IL-18 and IFN-γ, as well as anti-inflammatory IL4, IL-10 and TGF-β. TLR2 gene polymorphism is strongly determined by ethnicity and geography. Therefore it is necessary to uncovered the existence and association between Arg753Gln and Arg677Trp TLR2 gene polymorphism with TB susceptibility and its underlying mechanisms in Indonesian population in Bandung West Java. Methods: analytical observational study with cross-sectional design was conducted in Hasan Sadikin General Hospital Bandung from April 2011 to May 2012. Study population consisted of active pulmonary TB patient with positive AFB smear and Latent TB to ascertain previous MTb exposure. Polymorphism of gen Arg753Gln and Arg677Trp gene was determined with polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) methods. Plasma levels of IFN-γ, TNF-α, IL-10 and IL-12 were also compared between active and latent TB group. Results: heterozygote Arg753Gln TLR2 gene polymorphism was found in 9 of 86 pulmonary TB subjects (10.5%) but none in the latent TB group. The Arg677Trp polymorphism was not found in both groups. The odds ratio for Arg753Gln existence was 28.07 (p=0.022). No differences in the levels of IFN-γ, TNF-α, IL-10 and IL-12 between active pulmonary TB and latent TB subjects with and without Arg753Gln TLR2 gene polymorphism. Conlusion: Arg753Gln polymorphism of TLR2 gene is a risk factor for active pulmonary TB while Arg677Trp polymorphism is not. The Increased risk is not mediated by the difference in IFN-γ, TNF-α, IL-10 and IL-12 serum levels.