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Changes of toll-like receptor (TLR)4-NFκBp65-IL1β signaling but not TLR4 gene Asp299Gly and Thr399Ile polymorphisms are associated with primary gouty arthritis

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Objective: To investigate the changes of Toll-like receptor (TLR) 4-NFκB-IL1β signaling in peripheral blood and TLR4 gene Asp299Gly and Thr399Ile polymorphisms in patients with primary gouty arthritis (GA).

Methods: Asp299Gly and Thr399Ile polymorphisms were detected in 218 male GA patients and 226 age- matched male healthy subjects using 5' exonuclease TaqMan® echnology. TLR4 mRNA expression was measured using real-time quantitative PCR, IL1β production was detected using ELISA in 86 GA patients including 52 acute GA (AGA) and 34 non-acute GA(NAGA) and 78 age- and sex-matched healthy subjects. TLR4 and NFκBp65 proteins were measured using western blot. TLR4 inhibition in AGA or stimulation in NAGA was investigated by whole-blood with anti-TLR4 antibody or lipopolysaccharide (LPS). NFκBp65 transcriptional activity and IL1β production in response to TLR4 inhibition or stimulation was determined using ELISA.

Results: No Asp299Gly and Thr399Ile mutations were detected in both GA patients and healthy control subjects. Significant increases in the levels of TLR4, NFκBp65 and IL1β were observed on the peripheral blood of patients with AGA compared with those of NAGA patients and healthy subjects ($P<0.05$, respectively), and higher in NAGA patients than those in healthy subjects ($P<0.05$, respectively); significant positive correlations between concentration of UA and TLR4 mRNA level, serum IL-1β, between level of TLR4 mRNA and serum IL-1β ($P<0.05$, respectively) were observed in 52 AGA patients. NFκBp65 transcriptional activity and IL1β production inhibited by anti-TLR4 antibody was significantly reduced in patients with AGA, compared with that in healthy control subjects ($P<0.05$, respectively). TLR4 level, NFκBp65 transcriptional activity and IL1β production stimulated by LPS was significantly increased in patients with NAGA, compared with that in healthy control subjects ($P<0.05$, respectively).

Conclusions: TLR4-NFκB-IL1β signaling play a crucial role in the development of acute inflammation in primary gout patients, TLR4 activation in GA patients might be not associated with Asp299Gly and Thr399Ile mutations, and gouty inflammation susceptibility might be not associated with TLR4 gene Asp299Gly and Thr399Ile polymorphisms.

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