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No association of polymorphisms in the suppressor of cytokine signaling (*SOCS*)-3 with rheumatoid arthritis in the Chinese Han population

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ABSTRACT. Suppressor of cytokine signaling (SOCS)-3 is a key negative regulator of cytokine signaling that inhibits the JAK/STAT signal transduction pathway; there are reports describing its role in attenuating arthritis through SOCS-3 overexpression. We examined the relationship between polymorphisms in the coding sequence and promoter region of *SOCS-3* and rheumatoid arthritis (RA) in a Chinese Han population. Two single-nucleotide polymorphisms in the *SOCS-3* 5' region: -1044 C>A within the promoter region and rs12953258 (-920 C>A) in the 5'UTR (exon 2) of *SOCS-3* were studied by restriction fragment length polymorphism analysis and tetra-ARMS-PCR in 100 RA patients and 100 healthy adults. The prevalence of the homozygous genotype -1044 CC was 100% in both RA and control

groups. The heterozygous genotype (-920 C>A) was present in 89% of RA and in 82% of the control group, which is significantly different from the distribution in Western people. There was no transmission disequilibrium between these two SNPs ($r^2 = 0.000$). We did not detect significant differences in allele or genotype frequencies for either of these SNPs between the RA group and controls ($P > 0.05$). There was no association between rheumatoid factor and *SOCS-3* SNP rs12953258 ($P = 0.258$). We conclude that *SOCS-3* polymorphism is not a genetic risk factor for RA in Chinese patients.

Key words: Suppressor of cytokine signaling-3 (*SOCS-3*); Single-nucleotide polymorphism; Rheumatoid arthritis; Chinese Han population

