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Gαq gene promoter polymorphisms and rheumatoid arthritis in the Han Chinese population are not associated

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ABSTRACT. Mice that lose Gαq from their immune system can spontaneously develop inflammatory arthritis. Gαq expression in the peripheral blood lymphocytes of rheumatoid arthritis (RA) patients is significantly decreased in comparison to that in healthy individuals, and reduced Gαq expression is closely correlated with RA disease activity. These indicate that Gαq plays critical roles in the pathogenesis of RA. To address whether single nucleotide polymorphism in the promoter region of the Gαq gene (*GNAQ*) influenced Gαq expression in RA patients and was a genetic risk factor for RA, we sequenced the promoter region of *GNAQ* in a Han Chinese population. A common dinucleotide polymorphism at position -695/-694, an exchange of 2 adjacent nucleotides (GC>TT), was revealed in 118 RA patients and 101 healthy adults. The proportions of genotypes observed for -695/-694 in the RA group were GC/GC (65.25%), GC/TT (33.05%), and TT/TT (1.70%), and those in the control group were GC/GC (62.38%), GC/TT

(33.66%), and TT/TT (3.96%). No significant difference in the allele and genotype frequencies between RA patients and healthy controls for dinucleotide polymorphism was found in the Han Chinese population, neither in the whole data set nor in stratified subsets, i.e., rheumatoid factors, anti-cyclic citrullinated peptide antibody, and Gαq expression status ($P > 0.05$). We conclude that the *GNAQ* promoter polymorphism is not a genetic risk factor for RA in the Han Chinese population, and that decreased Gαq expression in peripheral blood lymphocytes of RA might potentially be due to other causes.

Key words: *GNAQ*; Polymorphism; Rheumatoid arthritis; Han Chinese population

