Possible Association Between XRCC1 Genes Polymorphisms and Systemic Lupus Erythematosus

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Dear Editor,

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease that involves multisystem organs. High production of auto-antibodies in this disease leads to hyper activation of the immune system and causes inflammation (1). The pathogenesis of SLE is not completely understood. Previous studies have shown that genetic factors play a key role in this disease (2-5). Anti-genicity could be increased by reactive oxygen species (ROS), which causes DNA conformation changes and DNA base damages or breaks (6). DNA repair enzymes monitor the DNA structure to correct damaged nucleotide produced by methylation, oxidation or oxidative damage; therefore, DNA repair mechanisms have an essential role in genome stability (7). X-ray cross-complementing 1 (XRCC1) is one of the DNA repair genes that may play a critical role in SLE (8). The main goal of this letter was to review the possible impacts of XRCC1 polymorphisms on SLE. Three single-nucleotide polymorphisms (SNPs) of this gene result in amino acid changes of Arg194Trp in exon 6 (rs18799782), Arg280His in exon 9 (rs25489), and Arg399Gln in exon 10 (rs25487) (9). In the first investigation, Bassi et al. could not show the association of XRCC1 polymorphism (rs25487) between SLE and healthy Brazilian individuals (10). Other investigations by Lin et al. and Warchol et al. showed that Arg/Arg genotype of rs25487 had a protective role against SLE in Chinese and Polish populations (8, 11). In the Iranian population, only one investigation was done, in which Salimi et al. showed that Arg/Gln had a protective role against SLE (6). Another SNP (rs1799782) was explored in 2 studies, which showed no association between the related SNP and SLE susceptibility (6, 11). The effect of rs25489 on SLE has not been investigated and the result of another SNP, which has been investigated should be elucidated. Therefore, more investigations are suggested on 3 SNPs of XRCC1 gene to clear its role in the SLE disease.

References