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ONLINE SUPPLEMENTARY DATA

Supplementary Table 1. Summary of the association between specific genotypes and SLE.

Locus/Variant Alleles	Pathological Role in SLE
IL-1 receptor antagonist (IL-1 RN) genes / rs315952	Variations in the number of tandem repeats (VNTRs) in intron 2 of the IL-1RN gene are associated with increased susceptibility to SLE[1-4].
IL1β -511 /CT	The TT homozygotes are associated with a higher risk of SLE than is the homozygous CC genotype in African Americans[5]. The frequency of the T allele was associated with increased susceptibility to SLE and predisposition to neurological disorders in SLE[6].
IL6-174 / GC	The homozygous GG genotype was more prevalent in Malaysian and Bulgarian SLE patients[7, 8]. The IL6-174 genotype may contribute to the manifestation of certain diseases, including nephritis[9].
IL10-1082, - 819, - 592 / AC	The IL-10 promoter is highly polymorphic, and 3 single nucleotide polymorphisms (SNPs) at positions −1082, −819, and −592 from the transcription start site are correlated with IL-10 production[10]. A recent meta-analysis of 38 case-control studies concluded that the IL10.G microsatellites, the *IL-10* -1082G/A and -592C/A polymorphisms and the haplotype -1082G/−819C/−592C are associated with SLE susceptibility[11].
FcγRIIa / 131 FR	FcγRIIA is a low-affinity receptor for IgG expressed on macrophages, neutrophils, monocytes and platelets[11]. A meta-analysis in 2009 including 29 studies showed that the overall homozygosity of RR and the low-binding allele of FcγRIIa were associated with the development of SLE and lupus nephritis[12].
FcγRIIIa / 158 FV	The Fcγ RIII a is anchored on the transmembrane on natural killer cells and macrophages[13]. The FF homozygotes have low-binding phenotypes with less engagement and activation of NK cells that has been shown to predispose to autoimmune disorders and nephritis[14]. Several studies support the association of the F allele with susceptibility to SLE[15, 16].
FcγRIIIb/ NA1/2	The Fcγ RIII b molecule is expressed in neutrophils (13) and preferentially removes small immune complexes from the circulation[17]. NA2/NA2 homozygotes have been associated with SLE development in Thai and Spanish populations[18, 19]. NA1/NA1 homozygotes are protected against central nervous system involvement[20].
TNFα-308 / AG	A meta-analysis of 28 studies concluded that the TNF-α promoter-308-A/G polymorphism is associated with susceptibility to SLE, although this effect may vary according to different ethnicities[21]. The genotype (AA + GA) at the TNF-308 promoter was significantly associated with clinical manifestations, such as malar rash, arthritis, oral ulcers, serositis and the SLE disease activity index[22].
LTA-252 / AG	The LTA 252 AG polymorphism has been associated with SLE susceptibility in German, Asian and Egyptian patients[22-24]. This polymorphism has also been associated with renal lupus and arthritis in SLE[9, 22].
ITGAMrs1143679 / HR	Several studies have demonstrated a link between the missense mutation at rs1143679 and susceptibility to SLE in various populations[25-30]. Certain manifestations of SLE, such as arthritis and nephritis, have also been linked to the missense mutation of ITGAM rs1143679[29, 30].
MBL2 Exon 1/ AO	Variant alleles are collectively denoted as O, while non-mutated alleles are referred to as A. A meta-analysis concluded that the MBL2 A/O

	polymorphism may be associated with SLE[31]. Several articles have also suggested the role of the MBL2 gene as a disease-modifying locus, predisposing to nephritis and complicating infections[32, 33].
MBL2 promoter rs7096206 / XY	The MBL2 -221X allele is the main promoter polymorphism affecting the MBL concentration[34]. A meta-analysis that was conducted in 2005 including 4 studies on the MBL2 -221 polymorphism concluded that the X allele was strongly associated with susceptibility to SLE[35]. Some studies have also linked the MBL2 -221 polymorphism with clinical manifestations of SLE, such as early onset, cutaneous manifestations, pleuritis/pericarditis and antiphospholipid antibodies[32, 36].
Vitamin D	A meta-analysis that was conducted on vitamin D and SLE revealed that the BsmI polymorphism showed a significant association with SLE and lupus nephritis (LN) in Asians[37]. It has also been reported that B-allele-negative patients have a greater risk of developing lupus nephritis[38].
XRCC1	The XRCC1 Arg399Gln polymorphism has been linked to SLE susceptibility in Taiwanese and Polish populations[39, 40]. This polymorphism has also been associated with disease manifestations, such as photosensitivity, malar rash, arthritis, and hematologic abnormalities[39, 40].

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