Supplementary Methods

Targeted capture and enrichment was performed using a NimbleGen array including 1,853 genes selected based on their known involvement in systemic inflammatory autoimmune diseases. The design and the implementation of such array, as well as the following re-sequencing experiments have been outlined elsewhere (1). The generated re-sequencing data were processed and quality-controlled according to the procedures described in the work of Thorlacius and collaborators (Thorlacius et al. 2020, in press), resulting in high-quality dataset that was used for further analyses.

For all the samples included in the study (292 patients with lupus nephritis, 563 with lupus without nephritis and 1030 healthy individuals), the genotypes of low frequency and rare (MAF < 0.05) variants overlapping with the extended TLR1-TLR10 loci (RefSeq Hg19 coordinates +- 10Kb) [2]shown below were extracted using VCFtools (3)We calculated deleteriousness score, CADD PHRED, for the variant's potential functional importance as described elsewhere(4)

Using PLINK (5), Fisher's exact test was employed to test whether the allele frequency of any of the variants identified in the TLRs genes showed a statistically significant difference between the group of patients with LN (n=292) and the group of individuals without LN (n=1593). Descriptive statistics were applied to examine the association between LN subtypes or all other ACR domains and rs142003616. SPSS V25 was used for statistics.

Genomic coordinates of the analysed TLR genes:

Chr	Start	End	Gene
1	223,272,748	223,326,624	TLR5
3	52,245,096	52,270,179	TLR9
4	38,763,860	38,794,611	TLR10
4	38,787,876	38,816,814	TLR1
4	38,815,325	38,868,438	TLR6
4	154,595,404	154,637,243	TLR2
4	186,980,309	187,016,252	TLR3
9	120,456,453	120,489,769	TLR4
X	12,875,202	12,918,480	TLR7
X	12,914,739	12,951,288	TLR8

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