Table 2. Studies with SLE patients

Authors & Reference	N	Gender	Country	Age (years)	BMI (kg/m²)	Disease duration	SLEDAI	Sequencing	α and β-diversity	*FIB* ratio	Phylum	Class	Order	Family	Genera	Species	Up-regulated functional pathways	Main findings
Guo et al. [26]	17 SLE patients without glucocorticoid therapy (SLE G–) 20 SLE patients with glucocorticoid therapy (SLE G+) 20 HC	Female	China	SLE G–: 34.41 ± 3.404 SLE G+: 34.25 ± 2.945 HC: 30.35 ± 1.907 (mean ± SEM)	SLE G–: 22.41 ± 0.4314 SLE G+: 23.14 ± 0.763 HC: 22.53 ± 0.686 (mean ± SEM)	SLE G–: 15.74 ± 5.984 SLE G+: 47.75 ± 8.039 Months (mean ± SEM)	SLEDAI < 6 SLE G+ (*n* = 10) SLE G– (*n* = 9) SLEDAI ≥ 6 SLE G+ (*n* = 10) SLE G– (*n* = 8)	16S rRNA: V4 regions Illumina Hiseq	SLE G–: lower α-diversity. HC and SLE G+: similar microbial communities, and bacteria abundance. No differences in α-diversity, community structure, or abundance of top 10 genera between SLEDAI < 6 *vs.* SLEDAI ≥ 6.	SLE G–: lower than HC.	SLE G–: *Bacteroidetes* and *Proteobacteria* significantly increased *vs.* HC and SLE G+. Decreased *Verrucomicrobia*, *Firmicutes*, and *Proteobacteria.*	NA	NA	NA	SLE G– *vs.* HC: increased *B., Parabacteroides* SLE G+ *vs.* SLE G– and HC: increased *Akkermansia* and *Lactobacillus*	NA	SLE G– *vs.* SLE G+ and HC: citrate cycle Glycolysis/Gluconeogenesis, Fructose and mannose metabolism, Galactose metabolism, other glycan degradation, MAPK signaling Glycosaminoglycan degradation, and Glycerophospholipid metabolism, and Pentose phosphate. SLE G+: ABC transporters, Amino acid metabolism, Bacterial motility proteins, Bacterial secretion system, fatty acid metabolism, Lipid metabolism, Glycan biosynthesis and metabolism, and Phosphotransferase system.	Glucocorticoid therapy has the potential to stabilize the gut microbiota of SLE patients, further decreasing the production of cytokines.
He et al. [27]	45 SLE patients 48 HC	Female	China	SLE: Train: 46.0 ± 1.8 (25, 61) Test: 39.9 ± 4.3 (18, 62)	SLE: Train: 21.5 ± 0.6 (16.4, 28.8)	SLE: Train: 7.9 ± 1.2 [1, 28] Test: 5.0 ± 1.6 [1, 16]	SLE: Train: 7.5 ± 0.5 [3, 14] Test: 6.7 ± 0.8 [4, 10]	16S rRNA: V3-V4 regions Illumina Miseq	α-diversity: no significant differences between SLE and HC.	NA	SLE: increased *Bacteroidetes*, *Actinobacteria*, and *Proteobacteria*;	NA	NA	SLE: increased *Prevotelaceae.*	SLE: increased *Rhodococcus, Eggerthella, Klebsiella, Prevotella, Eubacterium,*	NA	NA	Significantly altered genera could have the ability to discriminate

				HC: Train: 43.5 ± 2.4 (22, 68) Test: 42.7 ± 1.9 (20, 56) [mean ± SD (min, max)]	Test: 21.2 ± 1.2 (18.3, 27.7) HC: Train: 22.1 ± 1.0 (20.5, 27.7) Test: 21.6 ± 0.2 (20.6, 25.8) [mean ± SD (min, max)]	[mean ± SD (min, max)] Years	[mean ± SD (min, max)]		β-diversity: difference between SLE and HC.		decreased *Firmicutes*.				*Flavonifractor*, and *Incertae sedis.* Decreased *Dialister* and *Pseudobutyrivibrio.*			between SLE patients and HC.
Gerges et al. [28]	20 newly diagnosed SLE patients 20 HC	SLE Female: 18 Male: 2 (10%) HC Female: 16 Male: 4 (20%)	Egypt	SLE: 25.6 ± 6.3 HC: 29.9 ± 6.6 (mean ± SD)	SLE: 25.57 ± 4.02 HC: 23.78 ± 3.74 (mean ± SD)	3.5 ± 1.63 (mean ± SD) Months	9.25 ± 3.9 (mean ± SD)	RT-PCR	NA	SLE: lower than HC Inversed correlation between SLEDAI-2K and *F/B* ratio ($r = -0.451$; $P = 0.04$).	SLE: *Firmicutes* are significantly decreased; *Bacteroidetes* are significantly enriched.	NA	NA	NA	SLE: *Lactobacillus* are significantly decreased.	NA	NA	Gut microbiota dysbiosis may correlate with disease activity.

Chen et al. [41]	117 newly diagnosed SLE patients 115 HC	SLE Female: 107 Male: 10 (9%) HC Female: 97 Male: 18 (16%)	China	SLE: 30.8 ± 10.9 HC: 32.4 ± 11.3 (mean ± SD)	SLE: 20.8 ± 3.1 HC: 22.2 ± 3.0 (mean ± SD)	4 (mean) Months	10 (mean)	Metagenomic shotgun sequencing Illumina HiSeq	α-diversity: lower in SLE than HC (Shannon Index).	NA	NA	NA	NA	NA	NA	Enriched in SLE patients and reduced after treatment: *Clostridium* sp. ATCC BAA-442, *Atopobium rimae, Shuttleworthia satelles, Actinomyces massiliensis, B. fragilis, Clostridium leptum,* and an unclassified *Escherichia.*	Positively related to lupus: biosynthesis of *L*-arginine and *L*-ornithine, tryptophan, menaquinol, palmitoleate and oleate, phosphatidylglycerol biosynthesis, as well as purine nucleotides salvage and degradation. Negatively related to lupus: peptidoglycan biosynthesis and BCAAs biosynthesis.	*Clostridium* sp. ATCC BAA-442 positively correlated with SLEDAI. *Lactobacillus salivarius* positively correlated with SLEDAI and disease duration. *R. gnavus* was enriched in LN. In SLE patients but not in HC, the oral microbiota may contribute to gut inflammation and autoimmune pathology development.

Xiang et al. [29]	Two-sample MR, multi-ethnic large-scale GWAS. 7,219 SLE cases and 15,991 HC of European ancestry.	NA	United States of America, Canada, Israel, Netherlands, Belgium, Sweden, South Korea, Germany, Denmark, Finland, UK.	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	*Bacillales, Coprobacter,* and *Lachnospira* were negatively related to the risk of SLE. *Bacilli, Lactobacillales,* and *Eggerthella* might be risk factors for SLE onset.
Wei et al. [30]	14 SLE patients 16 HC	SLE Female: 13 Male: 1 (7%) HC Female: 14 Male: 2 (13%)	China	SLE: 40.71 ± 13.85 HC: 38.63 ± 14.50 (mean ± SD)	NA	4.86 ± 5.23 (Years ± SD)	NA	16s RNA: V3–V4 regions MiSeq	α-diversity: no difference (Shannon, Simpson, and Heip indexes); higher than HC by Sobs, Chao, Ace, and Pd indexes. β-diversity: difference between SLE and HC.	NA	SLE: increased *Proteobacteria.*	NA	NA	SLE: increased *Enterobacteriaceae*; decreased *Ruminococcaceae, Prevotellaceae, family_XI_o_Clostridiales*	SLE: increased *Streptococcus.* Decreased *Prevotella_9, Roseburia, Ruminococcaceae* UCG-003, *Ruminococcaceae* NK4A214_group*, Paraprevotella, Ruminococcaceae* UCG-013, *Ezakiella, Porphyromonas.*	NA	SLE: reduced Sphingolipid metabolism, and biosynthesis of Polyketide sugar unit Lysosome, Glycosphingolipid globo series, Butirosin and neomycin Adipocytokine signaling pathway and Glycosphingolipid.	*Proteobacteria* and *Ruminococcaceae* may be related to SLE occurrence. Alterations in the pathways associated with proteins and enzymes in SLE patients may be due to

																		the gut microbiota.
Wen et al. [31]	33 SLE patients (children). All with LN 28 HC	SLE Female: 26 Male: 7 (21%) HC Female: 14 Male: 14 (50%)	China	SLE: 12.39 ± 2.40 HC:10.61 ± 3.67 (mean ± SD)	SLE: 18.79 ± 2.3 HC: 18.57 ± 4.51 (mean ± SD)	NA	0–4: 22 (66.67%) 5–9: 2 (6.06%) 10–14: 9 (27.27%)	16S rRNA: V4–V5 regions 18S rRNA: V9 and ITS1 Illumina MiSeq	α-diversity: no difference. β-diversity: no difference.	NA	SLE: increased *Proteobacteria*.	SLE: increased *Alphaproteobacteria*, *Gammaproteobacteria*, Bacilli.	SLE: increased *Enterobacteriales*, *Xanthomonadales*, *Caulobacterales*, *Sphingomonadales*, *Lactobacillales*.	SLE: *Streptococcaceae*, *Caulobacteraceae*, *Enterobacteriaceae*, *Sphingomonadaceae*, *Rhodanobacteraceae*.	SLE: increased *Escherichia_Shigella*, *Clostridium_innocuum*_group, *Streptococcus*, *Hungatella*, *Erysipelatoclostridium*, *R._gnavus*_group, *Klebsiella*, *Lachnoclostridium*, *Rudaea*, *Sphingomonas*, *Kluyvera*.	NA	SLE: protein digestion and absorption (involving *L*-tryptophan, tyramine, *L*-phenylalanine, *L*-leucine, *L*-methionine, *L*-alanine, *L*-glutamine, *L*-valine, *L*-isoleucine, and *L*-tyrosine).	No significant difference in alpha-diversity between SLE and HC. *Proteobacteria* was increased in SLE patients. Sphingomonas with a positive correlation with protein digestion and absorption pathway.
Li et al. [32]	40 SLE patients 20 RA patients 22 HC	Female	China	SLE: 37.46 ± 14.17 HC: 37.18 ± 14.67 (mean ± SD)	NA	NA	SLEDAI ≥ 8 (*n* = 19) SLEDAI < 8 (*n* = 21)	16S rRNA: V3–V4 regions Illumina MiSeq	α-diversity: no difference between SLE and HC (Shannon and Simpson index). Higher in HC than SLE (Chao1 and Observed species).	Decreasing trend between SLE and HC, and between active and inactive disease (without	SLE: decreased *Tenericutes*, *Mollicutes*, and *RF39*. Bacilli showed clustered differences between SLE and HC. *Actinobacteria* showed clustered differences	NA	SLE: *Actinomycetales* and *Bifidobacteriales* showed clustered differences	SLE: increased *Streptococcaceae* and *Lactobacillaceae*.	SLE: decreased *Faecalibacterium*, and *Roseburia*. Increased *Streptococcus*, *Lactobacillus*, *Megasphaera*.	SLE: decreased *Faecalibacterium prausnitzii*. Increased *Streptococcus anginosus*, and	SLE: enrichment of *Streptococcus* positively correlated with increased apoptosis pathway, and negatively correlated with cancer pathways. Enrichment of *Streptococcus* negatively associated with pathways of alanine	Reduced bacterial α-diversity in SLE (Chao1 and Observed species). No significant difference in diversity

									No significant difference between active and inactive disease. β-diversity: different between SLE patients and HC; no associations with treatments.	significant difference).	between active and inactive disease.		between active and inactive disease.		*Bifidobacterium* reduced in active *vs.* inactive disease.	*Lactobacillus mucosae.* *R. gnavus* reduced in active *vs.* inactive disease. *Streptococcus anginosus*, positive correlation with SLEDAI.	aspartate and glutamate metabolism, primary and secondary bile acid biosynthesis, and positively associated with increased pathways of synthesis and degradation of ketone bodies, apoptosis, lipid metabolism, secretion system, and *Staphylococcus aureus* infection.	between SLE and RA. The genera *Streptococcus* and *Megasphaera* were increased in SLE patients *vs.* HC and RA patients. Different gut microbiota profiles between active and inactive disease. *Streptococcus* might play an important role in the disease progression in SLE.
Liu et al. [33]	35 SLE patients 35 HC	SLE Female: 32 Male: 3 (9%)	China	SLE: 41.46 ± 12.36 HC: 45.06 ± 11.24 (mean ± SD)	SLE: 23.03 ± 3.57 HC: 24.56 ± 2.89 (mean ± SD)	2 months to 20 years	Range between 1 and 12	16S rRNA: V3–V4 region	α-diversity: lower in SLE *vs.* HC.	No diferences between SLE and HC.	No diferences between SLE and HC.	NA	NA	SLE: decreased *Ruminococcaceae.*	SLE: increased *Lactobacillus, Prevotella, Blautia.* Decreased *Bifidobacterium.*	SLE: increased *L. iners* Decreased unclassified bacterium of	NA	Several genera with differences between low and high

		HC Female: 32 Male: 3 (9%)														*Ruminococcaceae* family, *B. adolescentis* and *B. Longum.*		disease activity. *Prevotella* and *Sneathia*: significant difference between low and high disease activity. Negative correlation between SLEDAI and abundance of *Acholeplasma*, *Capnocytophaga*, and *Leptotrichia.* Disease phenotype explained the variation in bacterial composition between SLE and HC.

																		Bacterial richness and diversity were not significantly different between SLEDAI cathegories
Luo et al. [42]	14 SLE patients (active disease) 17 non-SLE	SLE Female: 10 Male: 4 (29%)	USA	Between 21 and 73	Between 21 and 43.5	NA	Range between 0 and 13	16S rRNA: V4 region Illumina MiSeq	α-diversity: lower in SLE (Shannon index).	No differences between SLE and HC.	SLE: increase in *Proteobacteria*.	NA	NA	NA	SLE: decreased *Odoribacter*, increased *Blautia*, and an unnamed genus (family *Rikenellaceae*).	SLE: increased *Blautia wexlerae* (strain AUH-JLD17 and AUH-JLD56), *Blautia* spp. strain *Marseille* (strain P3602 and P3387), strain GD8, and strain AUH-JLD3, *Blautia faecis*, *Blautia caecimuris*.	NA	The gut microbiota of SLE patients differed in several bacterial species and was less diverse, with increased representation of Gram-negative bacteria, than HC.

																Decreased *Odoribacter laneus* (strain JCM and YIT), *Odoribacter splanchnicus* , *Alistipes onderdonkii, Alistipes* sp. (strain LS-J and LS-M), *Alistipes shahii, Alistipes obesi, Alistipes ihumii, Alistipes* spp. strain cv1, *Alistipes indistinctus* (strain JCM and YIT), *Alistipes* spp. strain S216, *Alistipes finegoldii* (strain DSM 17242, JCM		

																16770, CIP 107999 and AHN 2437).		
Hevia et al. [34]	20 SLE patients 20 HC	SLE Female: 20 HC Female: 20	Spain	SLE: 49.2 ± 10.7 HC: 46.9 ± 8.6 (mean ± SD)	SLE: 26.1 ± 5.3 HC: 25.2 ± 4.2 (mean ± SD)	2 years to 24 years	SLEDAI ≤ 8 *vs.* SLEDAI > 8	16S rRNA: V3 region Ion Torrent PGM	α-diversity: no difference (Shannon index).	SLE: lower.	SLE: increased relative abundance of *Bacteroidetes*.	SLE: increased *Bacteroidia*.	*Bacteroidales* and *Clostridiales*: significant difference between SLE and HC.	NA	SLE: high *Bacteroidetes* spp.	NA	SLE: glycan degradation pathways, lipopolysaccharide biosynthesis proteins, oxidative phosphorylation.	SLE patients had lower *F/B* ratio. Positive association between *Firmicutes* and HC and *Bacteroidetes* and SLE patients.

Tomofuji et al. [35]	47 SLE patients 203 HC	SLE Female: 43 Male: 4 (9%) HC Female: 104 Male: 99 (49%)	Japan	SLE: 42.9±15.9 HC: 35.4±12.0 (mean ± SD)	NA	Newly diagnosed: 18 (38.3%) Not newly diagnosed: 29 (61.7%)	Mean: 11.1±8.7	Whole-genome shotgun sequencing.	α-diversity: lower in SLE *vs.* HC; not different between the newly onset patients *vs.* other patients, patients with and without LN, or patients with high and low SLEDAI. β-diversity: altered in SLE *vs.* HC; not different between the newly onset patients *vs.* other patients, patients with and without LN, or patients with high and low SLEDAI.	NA	NA	NA	NA	NA	NA	SLE: increased *Streptococcus anginosus* and *Streptococcus intermedius.*	SLE: sulfur metabolism, redox reaction, flagellar assembly, starch and sucrose metabolism, cyanoamino acid metabolism, energy metabolism, ABC transporter, cell motility.	Altered gut microbiota in SLE *vs.* HC. Eight genes increased in the SLE metagenome derived from Streptococcus, including a gene related to redox reaction. SLE–specific link between biological pathways of the gut microbiota and the host genome.
He et al. [36]	21 SLE patients 10 HC	SLE Female: 21 HC Female: 10	China	SLE: 37.48 ± 2.44 HC: 37.50 ± 3.02 (mean ± SD)	NA	NA	NA	16S rRNA: V3–V4 regions Illumina HiSeq	α-diversity: no difference (Shannon, Simpson, Chao1 indexes). β-diversity: altered between SLE and HC.	SLE: increased.	SLE: increased *Bacteroidetes*, *Proteobacteria*.	SLE: decreased *Bacteroidia*, Clostridia; increased *Gammapr*	SLE: decreased Bacteridales, Clostridiales;	SLE: increased *Enterobacteriaceae, Enterococcaceae*; decreased *Ruminococcaceae*.	SLE: decreased *Bacteroidetes, R._2* and *R.* UCG-002, *Faecalibacterium*; increased *Proteobacteria*, *Enterococcus, Escherichia_Shigella.*	NA	NA	The gut microbiome of SLE patients displayed altered β-diversity. ~ 1-fold increase in the

												oteobacteria.	increased Enterobacteriales.					*F/B* ratio in SLE patients. Dysbiosis, including the decrease of potentially beneficial *Bacteroidetes* and the increase of the potentially pathological *Proteobacteria*, may contribute to SLE pathogenesis.
Azzouz et al. [37]	61 SLE patients 17 HC	SLE Female: 61 HC Female: 17	United States of America	Between 20 and 79	NA	NA	SLEDAI < 8 (*n* = 47) SLEDAI ≥ 8 (*n* = 14)	16S rRNA: V4 region Illumina MiSeq	α-diversity: lower in SLE than HC; a numerical trend towards an inverse correlation with SLEDAI; high disease activity (SLEDAI ≥ 8) lower diversity *vs.* HC. (Chao1 index). β-diversity: altered in SLE *vs.* HC; less variability in patients	NA	NA	NA	NA	SLE: *Veillonellaceae, Ruminococcaceae*.	SLE: *R.*	SLE: high *R. gnavus*. *B. uniformis*: lower in patients with highest disease activity.	NA	SLE patients with LN have intestinal expansions of the *R. gnavus*. The overabundance of *R. gnavus* correlated with lupus disease activity, and it was higher in patients with

									with low *vs.* high disease activity.									active renal disease. SLE patients with the highest disease activity had the lowest abundance of *B. uniformis.*
van der Meulen et al. [38]	35 SLE patients 40 pSS patients 965 HC	SLE Female: 33 Male: 2 HC Female: 554 Male: 408 (42%)	Netherlands	SLE: 47 ± 14 HC: 45 ± 13 (mean ± SD)	SLE: 27 ± 5 HC: 25 ± 4 (mean ± SD)	SLE: 11 ± 9 (mean ± SD)	SLEDAI ≤ 4 *vs.* SLEDAI > 4	16S rRNA: V4 region Illumina MiSeq-v2	α-diversity: lower richness in SLE than HC (Chao1); without differences in diversity (Shannon index). β-diversity: altered in SLE *vs.* HC; different β-diversity between high or low disease activity.	SLE: lower	SLE: higher *Bacteroidetes* and *Proteobacteria.*	NA	NA	NA	SLE: higher *B.*, and *Alistipes.* Genera associated with SLE: *Butyricicoccus, Dorea, Erysipelotrichaceae*-UCG-003, *Eubacterium ruminantium* group, *Marvinbryantia, Phascolarctobacterium*, *Ruminoclostridium*_9, *R. gauvreauii* group.	SLE: higher *B. vulgatus, B. uniformis, B. ovatus,* and *B. thetaiotaomicron.*	NA	Factors contributing to the microbiota variation: age, sex, BMI, smoking, and use of PPI. Lower richness of the gut microbiota of SLE patients than HC.
Li et al. [39]	40 SLE patients (20 receiving PPI: P-SLE and 20 not receiving: NP-	SLE Female: 40 HC	China	P-SLE: 34.25 ± 10.54 NP-SLE:	P-SLE: 22.49 ± 3.39 NP-SLE: 23.16 ± 2.78	P-SLE: 1.5 (0.15–4.7) NP-SLE: 5.5 (0.7–10.75)	P-SLE: 9.65 ± 4.78 NP-SLE: 6.5 ± 4.51 (mean ± SD)	16S rRNA: V3–V4 regions Illumina	α-diversity: lower in NP-SLE *vs.* HC; P-SLE with higher diversity than NP-SLE.	NA	Higher Veillonellaceae and Enterobacteriaceae in NP-SLE than P-SLE and HC.	NA	NA	Higher abundance of Enterobacteriaceae in NP-SLE	NP-SLE: higher Rothia, Morganella, Escherichia, Pseudomonas, Stenotrophomonas,	NA	Increased in P-SLE and NP-SLE: carbon fixation pathways in prokaryotes, cell growth and death, thiamine metabolism, immune system,	SLE patients using PPI showed different gut microbiota than

	SLE) 17 HC	Female: 17		35.95 ± 10.27 HC: 30.12 ± 14.14 (mean ± SD)	HC: 22.48 ± 2.51 (mean ± SD)	(Years, median (IQR))			(Chao1, inverse Gini-Simpson, Shannon index) β-diversity: higher variation within NP-SLE *vs.* HC and P-SLE.		Lower Prevotellaceae in NP-SLE than HC. Without statistical significance between groups.			than P-SLE and HC.	Veillonella, and Enterococcus than HC. P-SLE: higher Desulfovibrio, Oxalobacter, Roseburia, Streptococcus, and *Lactobacillus* than NP-SLE. Lower Veillonella, Escherichia, Pseudomonas, Stenotrophomonas, and Morganella were lower than NP-SLE.		DNA replication, drug metabolism, other enzymes, NOD-like receptor signaling pathway, and plant-pathogen interaction. Reduced in P-SLE: nitrotoluene degradation, biofilm formation by Escherichia coli, propanoate metabolism, pentose phosphate pathway, phosphotransferase system PTS, ABC transporters, membrane transport, and environmental information.	patients not taking it. Characteristics of the gut microbiota of P-SLE patients similar to HC. PPI use was associated with altered microbial metabolic pathways in SLE and improved the microbiota composition of SLE patients.
Ma et al. [40]	18 SLE patients 7 HC	SLE Female: 18 HC Female: 7	China	NA	NA	NA	NA	16S rRNA: V4 region IonS5 XL	α-diversity: no differences. β-diversity: differences not statistically significant.	NA	NA	NA	NA	NA	SLE: increased *Turicibacter*.	SLE: increased *Clostridium papyrosolvens*, *Lactobacillus reuteri*, *Lactobacillus intestinalis*, *Lachnospiraceae*	NA	Fecal transplant from SLE patients to germ-free mice led to an increase in autoantibodies, and an approximation of the gut microbiota

																bacterium A2 and *Lachnospiraceae bacterium* M18–1.		composition between donors and recipients. Fecal microbiota from SLE patients can induce lupus-like phenotypes and alter histidine metabolism in germ-free mice.

ABC: ATP-binding cassette; *B.*: *Bacteroides*; BCAA: branched-chain amino acid; BMI: body mass index; GWAS: genome-wide association study; IQR: interquartil range; ITS1: internal transcribed spacer 1; MAPK: mitogen-activated protein kinase; MR: mendelian randomization; N: number of individuals; NOD: nucleotide-binding oligomerization domain; NP-SLE: not taking proton pump inhibitors; PGM: personal genome machine; PPI: proton pump inhibitors; P-SLE: taking proton pump inhibitors; pSS: Primary Sjögren's syndrome; RA: rheumatoid arthritis; RT-PCR: reverse transcription polymerase chain reaction; SD: standard deviation; SEM: standard error of mean; SLEDAI: systemic lupus erythematosus disease activity index; SLE G–: systemic lupus erythematosus not taking glucocorticoids; SLE G+: systemic lupus erythematosus taking glucocorticoids; UCG: UCG codon