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Supplement Materials and Methods

Skin Lesions

We scored the skin lesions by gross pathology using a grade of 0–3 (0 = none; 1 = mild (snout and ears); 2 = moderate, < 2 cm (snout, ears, and intrascapular); 3 = severe, 2-4 cm (snout, ears, and intrascapular); and 4 = very severe, >4 cm (snout, ears, and intrascapular). Moreover, we assessed the incidence of skin lesions that showed grade 2 or more.

β-Galactosidase

Kidneys were fixed in 4% paraformaldehyde for 3 hours at 4°C, embedded and sectioned at 20-µm thick sections. Cryosections were stained with X-gal (Cat. X4281C10; Gold Biotechnology) overnight at 37°C and subsequently counterstained with Nuclear Fast Red (Cat. N3020; Sigma-Aldrich). We scored expression in tubules on a scale of 0-4 (0 = none; 1 = weak; 2 = moderate; 3 = strong; 4 = very strong) under x40 magnification in at least 20 high power fields per section.

Histopathology

Kidney

Kidneys were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned (4 µm), and stained with PAS. To score kidney pathology we evaluated glomerular, tubulo-intestitial and perivascular pathology as follows. Glomerular pathology was assessed by examining 20 glomerular cross-sections (gcs) per kidney and scoring each glomerulus on a semiquantitative scale: 0 = normal (35–40 cells/gcs); 1 = mild (glomeruli with few lesions showing slight proliferative changes, mild hypercellularity (41–50 cells/gcs), and/or minor exudation; 2 = moderate (glomeruli with moderate hypercellularity (51–60 cells/gcs), including

segmental and/or diffuse proliferative changes, hyalinosis, and moderate exudates); and 3 = severe (glomeruli with segmental or global sclerosis and/or severe hypercellularity (>60 cells/gcs), necrosis, crescent formation, and heavy exudation). Tubulo-interstitial pathology was assessed in 10 randomly selected high power fields (>400) on a scale of 0–3 according to the number of infiltrates and damaged tubules: 0 = normal, 1 = mild, 2 = moderate, and 3 = maximum. Perivascular cell accumulation was determined semi-quantitatively by scoring the number of cell layers surrounding the majority of vessel walls (score: 0 = none; 1 = < 5; 2 = 5–10; and 3 = >10).

Salivary Gland

The salivary glands were fixed in 10% neutral buffered formalin. Paraffin sections (4 μm) were stained with H&E and pathology was evaluated. We scored the salivary gland inflammation on a scale of 0–3 (0 = no inflammatory cells); 1 = few perivascular and periductal inflammatory infiltrates (<100 cells); 2 = moderate number of perivascular and periductal inflammatory infiltrates (100–500 cells); 3 = extensive inflammation with large inflammatory foci (>500 cells).

Renal Function

Albuminuria

To quantify albuminuria levels, we analyzed 20 μl of urine collected over 8 hours by SDS-PAGE. Briefly, bovine serum albumin standards (0.25, 0.5, 1.0, 2.5, and 5.0 μg) were run on the same gel and used to identify and quantify urinary albumin bands. Gels were stained with Coomassie blue, the bands were captured and quantified using ChemiDoc MP imaging system (Bio-Rad). The values of the sample bands were translated into albumin concentrations using the standard curve, which were extrapolated to the 8-hour total urine volume.

BUN

BUN levels were evaluated using a colorimetric analysis kit (Urea Nitrogen kit; Sigma-Aldrich, St. Louis, MO) according to manufacturer's instructions. A standard curve was generated and used to determine the concentration of urea nitrogen in the serum samples, collected at the time of sacrifice.

Serum Creatinine

Serum creatinine concentration was measured using an autoanalyzer (Beckman Instruments, Inc., Fullerton, CA) according to manufacturer's instructions.

Immunofluorescence

To determine IgG and C3 deposits in the glomeruli, we incubated cryostat sectioned tissue with 10% normal goat serum, followed by FITC-conjugated goat anti-mouse IgG and FITC-conjugated goat IgG fraction of mouse C3 (Cappel Laboratory, Malvern, PA). The fluorescence intensity within the peripheral glomerular capillary walls and mesangium were scored on a scale of 0-3 (0 = none; 1 = weak; 2 = moderate; 3 = strong) in at least 10 glomeruli per section.

To determine the number of intra-renal proliferating Mø, cryostat sectioned tissues were stained with anti–mouse F4/80 Ab (clone BM-8, Invitrogen, Carlsbad, CA), and anti–mouse Ki-67 Ab (clone SP6, Vector Laboratories, Burlingame, CA), followed by Cy3-conjugated goat anti-rat IgG Ab (clone polyclonal, Jackson ImmunoResearch, West Grove, PA) and FITC-conjugated goat anti-rabbit IgG Ab (clone polyclonal, Jackson ImmunoResearch, West Grove, PA). We enumerated the number of F4/80+/Ki-67+ cells in 10 HPF.

Immunoperoxidase

Mouse: We stained frozen kidney sections, fixed in 25% ethanol/75%acetone for 10 min at room temperature, and blocked endogenous peroxidase activity and nonspecific binding of avidin and biotin. We detected the presence of Mø, T cell, double negative T cells, and apoptotic cells in TEC using the antibodies listed in Supplement Table 2. Optimal concentrations of primary Abs were diluted in Ab dilution buffer and incubated with the tissue sections overnight in a humidified chamber at 4°C. We incubated tissue sections with biotinylated anti-rat IgG Ab (BA-4001; Vector Laboratory, Burlingame, CA) for 1 h at room temperature, followed by incubation with ABC complex (PK-6100; Vector Laboratories) for 1 h at room temperature. Then, the stain was developed using DAB (SK-4100; Vector Laboratories), followed by counterstain with Mayer's Hematoxylin (Sigma-Aldrich, St. Louis, MO). Immunostaining was analyzed by counting for the presence of F4/80, CD4, B220, and caspase-3-positive cells in 20 randomly selected HPF.

Human: Kidney formalin fixed tissue sections obtained from human kidney biopsy specimens were stained for the presence of IL-34, CSF-1, cFMS, PTPRZ, Mø (CD68), and T cells (CD3). Antibodies used for immunostaining are listed in Supplement Table 2. Antigens were retrieved by immersion in citrate buffer followed by blocking of endogenous peroxidase activity and nonspecific binding of avidin and biotin. We incubated kidney sections with a primary antibody and detected the primary antibody by incubation with biotinylated rabbit anti-goat antibody or goat anti-rabbit antibody or goat anti-mouse antibody, followed by development with 3-3-diaminobenzidine (Vector Laboratories; Burlingame, CA). Immunostaining was analyzed by counting for the presence of positive cells in 10 randomly selected HPF.

ELISA

Total IgG: Plates were coated overnight at 4°C with goat anti-mouse Ig capture Ab (Southern Biotechnology, Cat. 1010-01) in PBS. The wells were washed 3 times and blocked for 1 h with 1% BSA in PBS. We added Ig standards (mouse IgG-UNLB, Southern Biotechnology, cat. 0107-01) to the plate using a series of 2-fold dilutions, and assessed serum samples at 1/10000 dilution. Standards and serum samples were incubated 2 h at RT, and bound Ig was detected with goat anti-mouse detection Ab conjugated with HRP (Southern Biotechnology, cat.1030-05) and TMB solution (Zymed, cat. 0020-23). The absorbance was measured at 450 nm.

dsDNA: Immunolon (Dynex, cat. 3355) plates were coated for 1h at RT with 2 µg/mL of dsDNA (calf thymus DNA, Sigma) in PBS. To get dsDNA, 400 µL of 0.5 mg/mL thymus DNA was treated with 5 µL of 40 U/µL Mung Bean Nuclease (Amershan cat. E2420Y) for 1 min at 37^0C. The wells were blocked for 1 h at RT with 1% BSA 0.1% Tween 10% in PBS. We added serum 100 µL/well , 1h at RT (dilutions 1:50 to 1:2000). Bound IgG was detected with goat anti-mouse detection Ab conjugated with HRP (Southern Biotechnology, cat.1030-05) and TMB solution (Zymed, cat. 0020-23). The absorbance was measured at 450 nm.

CSF-1: The tissues used for homogenates were from mice perfused with PBS through the heart to flush out CSF-1 in the circulation. Briefly, we homogenized frozen tissue samples in ELISA-lysis buffer (20 mM Tris-Hcl pH 7.5 , 150 mM NaCl, 1% NP-40, with proteinase inhibitors from Sigma) using a homogenizer. Samples were spun down and the supernatant fraction of the homogenates was used for the ELISA. We determined the protein concentration of each sample (supernatant of homogenate) using the BCA Protein Assay Kit (PIERCE, Rockford, IL) and evaluated 200 µg of protein per tissue sample. The ELISA

Capture (Cat. 552513), detection (cat. 552514) antibodies and reagents were purchased from BD Bioscience (San Jose, CA).

IL-34: Plates were coated for 24 h with 0.5 µg/mL of the capture Ab (R&D, Cat. AF5195), blocked with 1% BSA in PBS at RT for 1.5 h and serum samples were added undiluted and incubated at 4^{o}C overnight. Detection Ab (BioLegend, cat. 519303) is prepared in 1 % BSA 0.05 % Tween-20 in PBS and incubated at RT for 1 h. Bound Ab was detected with a Strep-HRP Ab and TMB solution (Zymed, cat. 0020-23). The absorbance was measured at 450 nm.

Leukocyte isolation whole blood (human)

B cells (CD19+; Miltenyi, Cat. 130-050-301)), T cells (CD3+; Miltenyi, Cat. 130-050-101)) and monocytes (CD14+; Miltenyi, Cat. 130-050-201)) (Miltenyi Biotec)were isolated from whole blood. Cells subsets were separated using the magnetic cell isolation and cell separation system from Miltenyi Biotec following the manufactures instructions. We used the following Abs for detection: rabbit anti-human IL34, dilution 1:1000 (Origene/Acris), rabbit anti-human PTPRZ, dilution 1:200, goat anti-human M-CSF, dilution 1:200, rabbit anti-cFMS, dilution 1:200 (Santa Cruz), GAPDH, dilution 1:5000 (Cell signaling), anti-goat HRP and anti-rabbit HRP, dilution 1:10000 (Santa Cruz).

qPCR

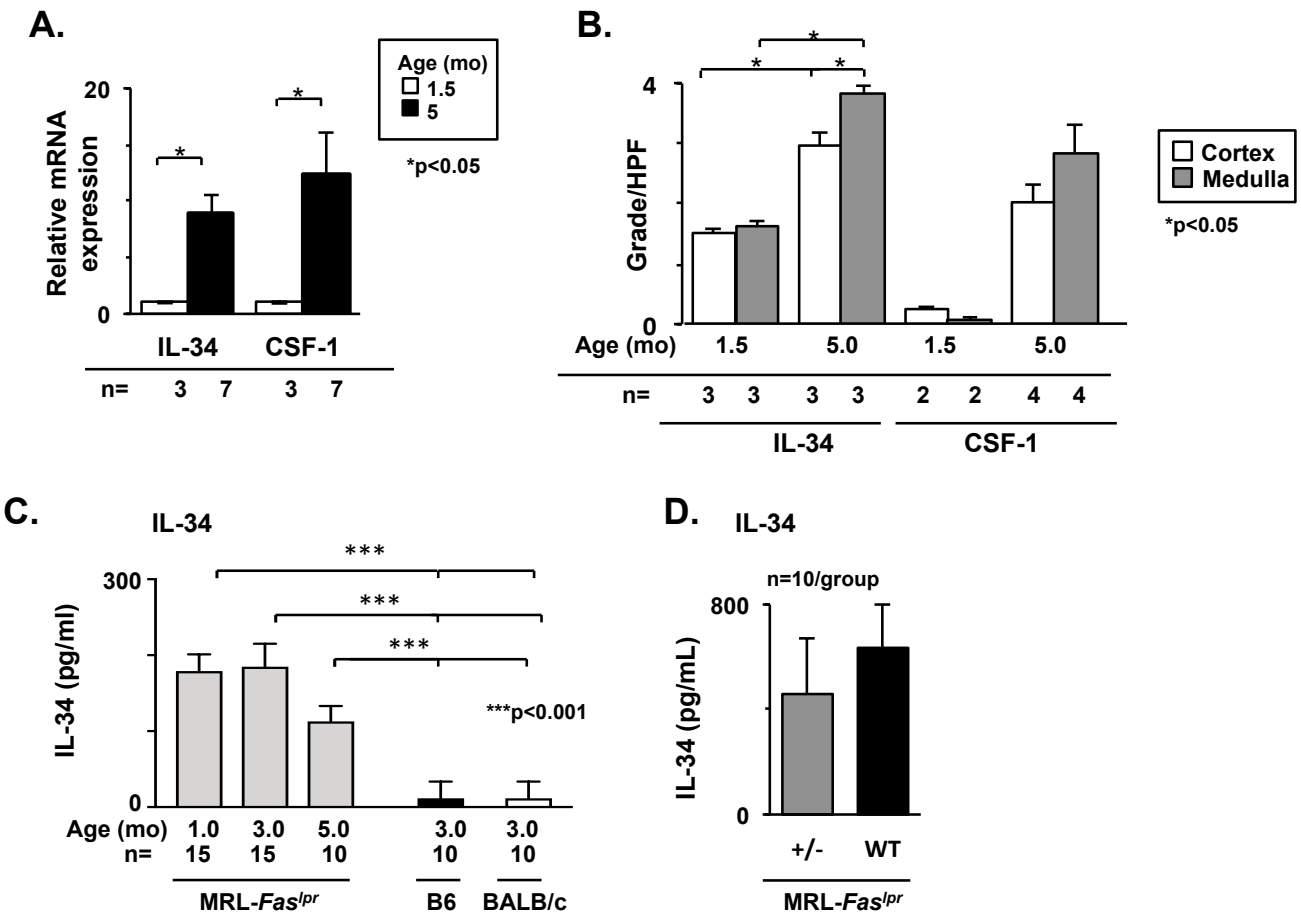
qPCR was performed using real-time, 2-step, quantitative PCR. Total RNA was isolated from snap-frozen whole kidney, primary TECs, and BMMø using TRIzol (Life Technologies, Gaithersburg, MD). The RT reaction was performed on 100 µg of RNA using an oligo (dT) primer and Superscript II reverse transcriptase (Life Technologies). Relative quantitation with real-time two-step RT-PCR was performed with SYBR Green PCR reagents (Qiagen,

Valencia, CA) and an ABI PRISM 7700 sequence detection system (PE Applied Biosystems, Foster City, CA) according to the manufacturer's instructions. Reactions were performed using 1.0 µl of cDNA at a concentration of 100 ng/ml in a reaction volume of 25 µl. The PCR consists of HotStar *Taq* activation for 10 min at 95°C, followed by 40 cycles with heating to 95°C for 15 s and cooling to 60°C for 1 min. The mRNA levels were normalized to those of GAPDH. The data were analyzed by the ΔΔ-CT method. Primers are listed in Supplement Table 3.

SLE Patient's Disease Activity

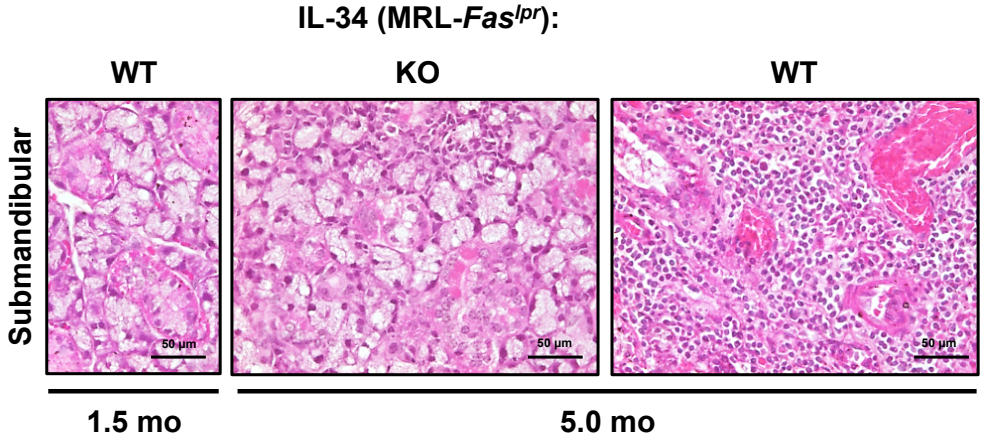
Disease activity was evaluated by the following standard clinical serological activity parameters: (complement 3 (C3c), complement 4 (C4), antinuclear antibodies (ANA), anti-double stranded DNA (dsDNA) antibodies and urine parameters: proteinuria (24h collection). The following standard values of serological activity markers were determined: C3 (0.9-1.8 g/l); C4 (0.1-0.4) by enzyme immunoassay; ANA (1:80-1:5120) by immunofluorescence; dsDNA (30 IU/ml - 200 IU/ml) by ELISA; creatinine (0.5-0.8 mg/dl) by isotope dilution mass spectrometry; proteinuria (<150 mg/24h) by immunoturbidimetric assay.

Supplement Figure 1



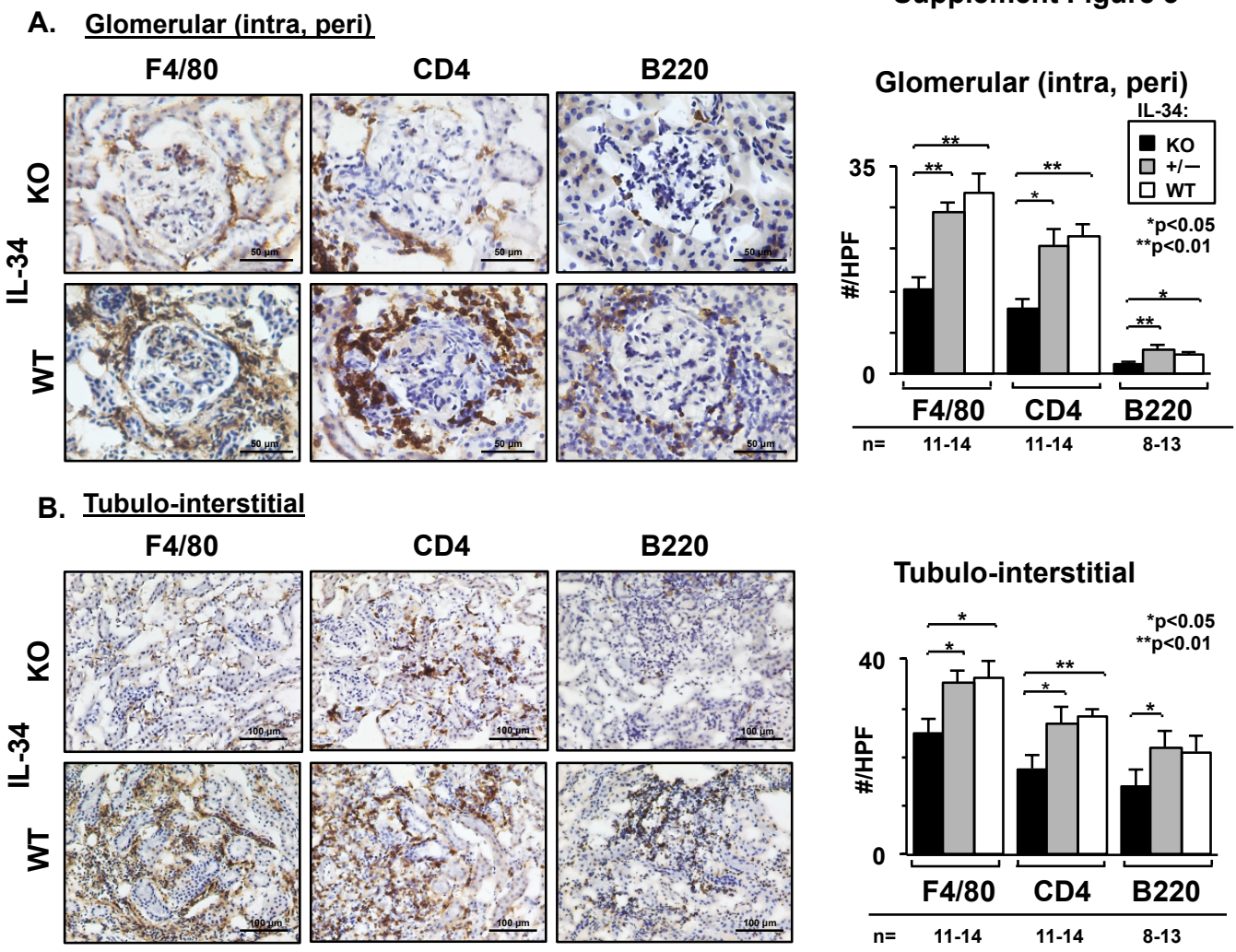
Supplement Figure 1. IL-34 and CSF-1 are expressed with advancing lupus nephritis in MRL-*Fas*^{lpr} mice. (A) IL-34 and CSF-1 transcript levels in MRL-*Fas*^{lpr} WT kidneys by qPCR. Values were normalized to GAPDH transcripts and expressed as relative ratio. (B) IL-34 and CSF-1 expression in kidney of MRL-*Fas*^{lpr} mice identified by reporter mouse (LacZ under control of IL-34 or CSF-1) stained for ß-galactosidase activity (X-gal). Graph of X-gal intensity comparing the cortex and medulla. (C) IL-34 in serum of MRL-*Fas*^{lpr} mice and B6 and BALB/c mice analyzed using an ELISA. (D) IL-34 in serum of IL-34 +/- and WT MRL-*Fas*^{lpr} mice at 5 mo of age using a different ELISA than in C. IL-34 KO= 0. Data are mean ± SEM. Mann-Whitney U test was used for statistical analysis.

Supplement Figure 2.



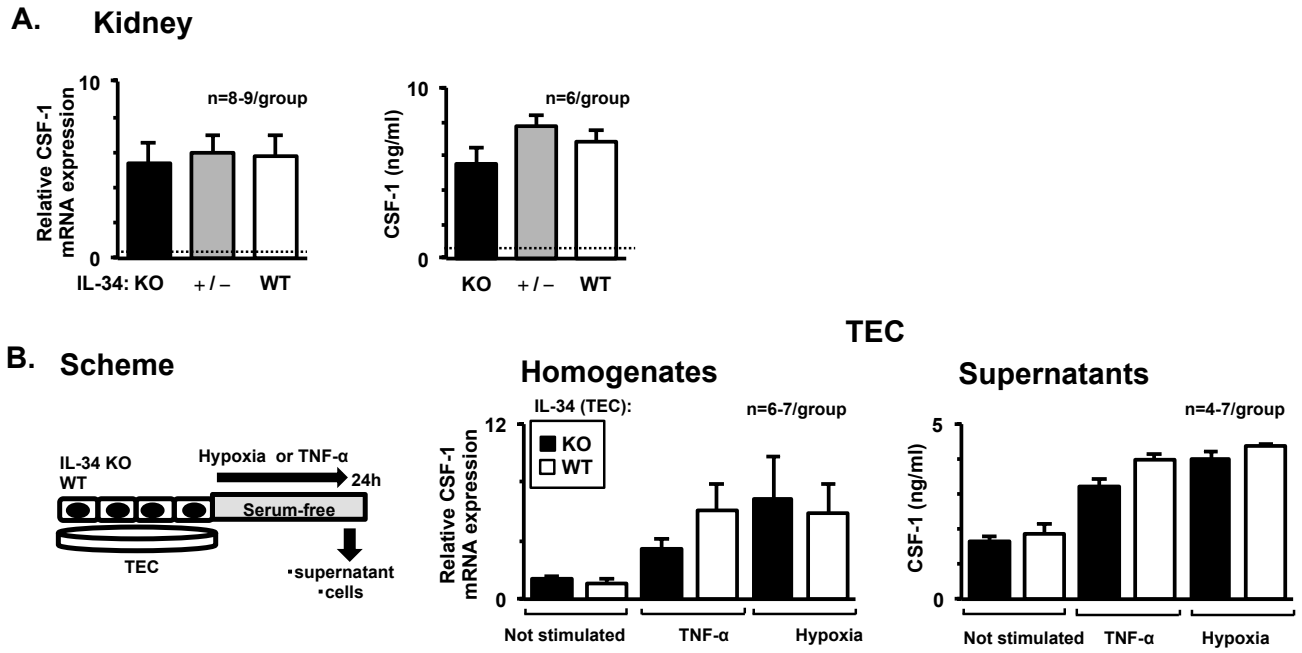
Supplement Figure 2. Submandibular inflammation (sialadenitis) is suppressed in IL-34 KO MRL-*Fas*^{lpr} mice. We analyzed the submandibular gland at 1.5 and 5 mo age in IL-34 KO and WT MRL-*Fas*^{lpr} mice. Representative photos (x 40) stained with H&E. Note the infiltrating cells in the WT, but not in the IL-34 KO mice. Data are mean ± SEM. Mann-Whitney U test was used for statistical analysis.

Supplement Figure 3



Supplement Figure 3. Mø, T and B cell accumulation is suppressed in IL-34 KO MRL-*Fas*^{lpr} mice. The following were compared: IL-34 KO, WT and +/- MRL-*Fas*^{lpr} mice (5 mo of age). Mø (F4/80), T cell (CD4), and $CD4^{-}CD8^{-}$ T cell and B cells (B220) were identified by immunostaining in (A) Glomeruli: Representative photos (x 40) and (B) tubulo-interstitium (x20). Adjacent graphs show quantification of 10 randomly selected HPF. Data are mean ± SEM. Mann-Whitney U test was used for statistical analysis.

Supplement Figure 4

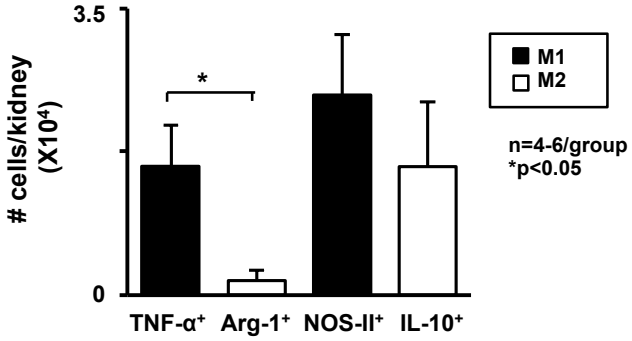


Supplement Figure 4. CSF-1 does not compensate for the absence of IL-34 in the kidney of IL-34 KO MRL-*Fas*^{lpr} mice. (**A**) Intra-renal CSF-1 transcript and protein levels analyzed by qPCR and ELISA in IL-34 KO, WT and +/- MRL-*Fas*^{lpr} mice (5 mo of age). Values are normalized to GAPDH transcript and expressed as relative quantification. Dotted lines are MRL-*Fas*^{lpr} at 1.5 mo of age (n=3). (**B**) Scheme, *in vitro* analysis of CSF-1 generated by TEC. CSF-1 in TEC from IL-34 KO and WT MRL-*Fas*^{lpr} mice analyzed by qPCR and ELISA, respectively. Data are mean ± SEM. Mann-Whitney U test was used for statistical analysis.

Supplement Figure 5

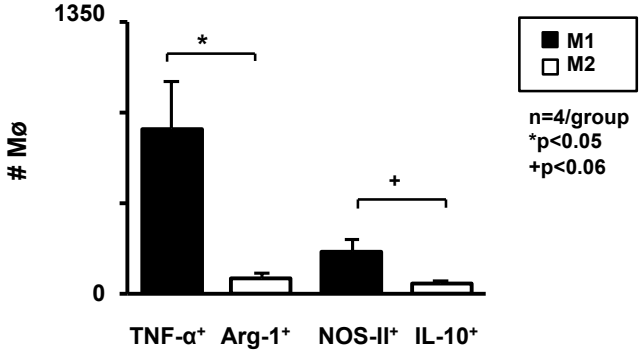
A. In vivo (Kidney- MRL-*Fas*^{lpr} at 5 mo of age)

Cell number (Gated on CD45$^+$CD11b$^+$Ly6G$^-$F4/80$^+$)



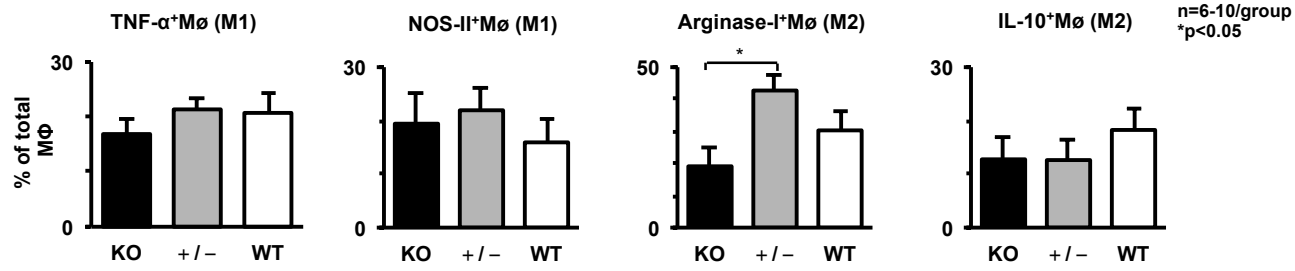
B. In vitro (MRL-*Fas*^{lpr} BM Mø co-cultured with hypoxic TEC)

Cell number

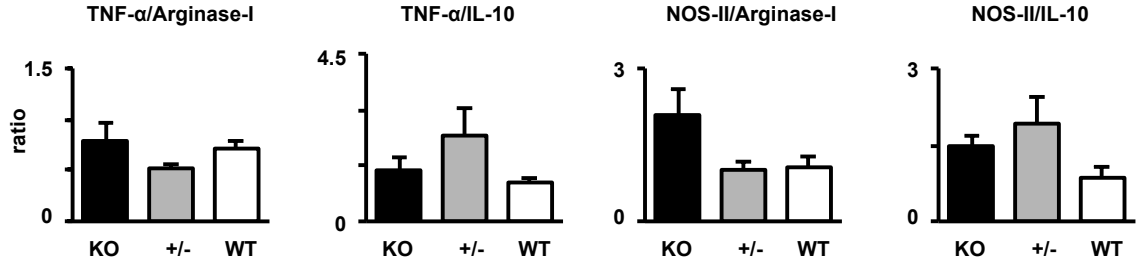


C. Polarization in kidney (MRL-*Fas*^{lpr} – 5 mo of age)

1. Frequency (Gated on CD45$^+$CD11b$^+$Ly6G$^-$F4/80$^+$)

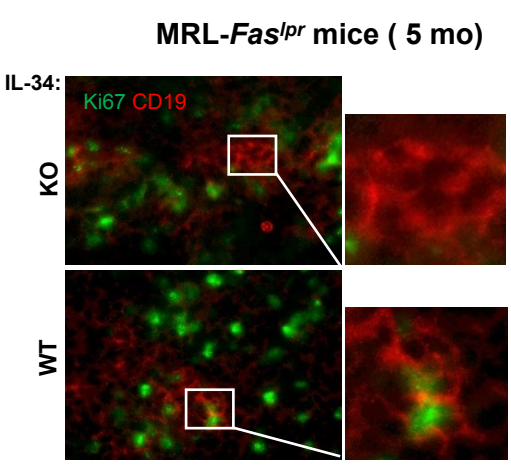


2. M1/M2 ratio



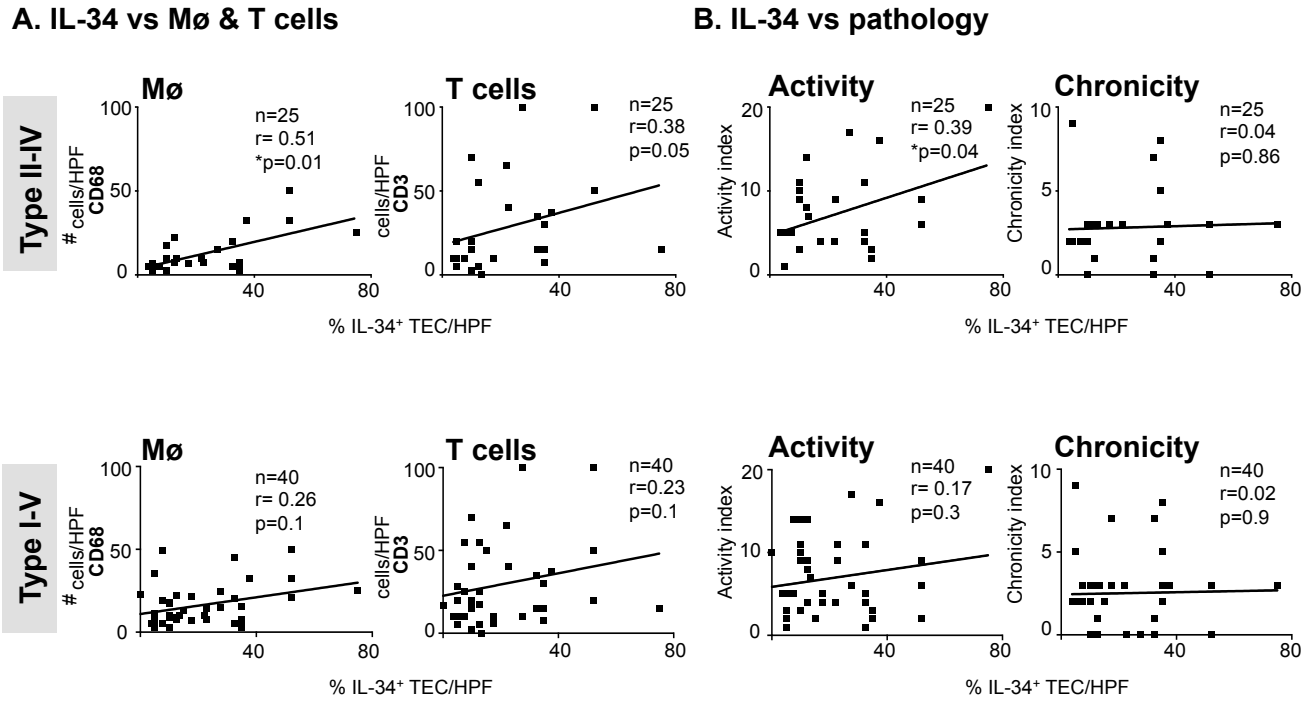
Supplement Figure 5. Mø skew towards cyto-destructive M1 phenotype during lupus nephritis in MRL-*Fas*^{lpr} mice and in BM Mø co-cultured with hypoxic TEC. Quantified cyto-destructive (M1, TNF–α, NOS-II$^+$) and cyto-protective (M2, Arg-1$^+$IL-10$^+$) Mø in (A) MRL-*Fas*^{lpr} mice (5 mo of age) and in (B) BM Mø co-cultured with hypoxic TEC analyzed by FACS. (C) Mø polarization in MRL-*Fas*^{lpr} kidney at 5 mo of age. 1. Frequency of M1 and M2 markers in IL-34 KO, WT and +/- MRL-*Fas*^{lpr} kidney. 2. M1/M2 ratio in IL-34 KO, WT and +/- MRL-*Fas*^{lpr} kidney. Data are mean $\pm$ SEM. Mann-Whitney U test was used for statistical analysis.

Supplement Figure 6



Supplement Figure 6. Proliferating B cells are suppressed in IL-34 KO MRL-*Fas*^{lpr} mice with lupus nephritis. Representative photos of dual staining CD19 and Ki67 in IL-34 KO and WT MRL-*Fas*^{lpr} kidney cryosections at 5 mo of age (x40, inset x160).

Supplement Figure 7



Supplement Figure 7. IL-34 correlates with the number of Mø and T cells and histopathology disease activity in type II-IV lupus nephritis. (A) Correlation of intra-renal Mø ($CD68^+$) and T cells (CD3) with IL-34 in lupus nephritis in Type II-IV (n=25, corresponding demographic information in supplement Table 1) and Type I-V (n=40, corresponding demographic information in supplement Table 1). (B) Correlation of IL-34 with kidney histopathology activity and chronicity indices in lupus nephritis in Type II-IV and Type 1-V. IL-34, CSF-1, CD68 and CD3 are detected in renal biopsy by immunostaining. Statistical analysis using Spearman Correlation Coefficient.

Supplement Table 1. Study cohort demographic and clinical characteristics

		LN	Healthy control	Biopsy-proven LN	Healthy control	Biopsy-proven LN	SLE for WB*	Healthy control WB
		n=120	n=62	n=25	n=15	n=40	n=3	n=4
Race	White	105	62	23	15	35	3	4
	Asian	11	0	1	0	3	0	0
	Others	3	0	1	0	2	0	0
Sex	Female	109	51	22	10	35	3	4
	Male	11	11	3	5	5	0	0
Age (years) at study	Mean	44.1±2.1	48.5±1.9	32.3±2.1	40.7±3.7	34.1±2.7	42.1±2.3	33.5±4.1
	Range	22-75	23-76	17-69	22-67	17-69	25-58	25-41
Age at SLE diagnosis (years)	Mean	32.9±2.2		29.1±1.9		33.1±1.9	29.1±1.7	
	Range	12.1-72.3		10.7-67.1		10.7-67.1	18.1-43.4	
Age at diagnosis of LN (years)	Mean	38.6±4.2		32.3±2.1		34.1±2.7	29.1±1.7	
	Range	14.3-58.2		17.5-69.3		17.5-69.8	18.1-43.4	
Disease duration at time of LN onset (years)	Mean	9.1±1.9		4.3±1.1		4.2±1.1	0	
	Range	0-34.8		0-30.2		0-30.2	0	
Overall duration of SLE in this cohort (years)	Mean	10.4±1.4		8.7±1.1		9.2±1.2	11±1.8	
	Range	0.7-43.7		0.8-25		0.8-25	2.1-28.2	
Clinical parameter at the time of LN diagnosis								
Creatinine (mg/dl)		1.2±0.2	1.0±0.2	1.4±0.1	0.9±0.2	1.6±0.3	1.1±0.2	1.0±0.1
Proteinurie (24h collection) >0.5g	n	120		25	15	38	0	
	Mean	1.3±0.5		2.7±0.4	0.1±0.03	3.4±0.6	0.4±0.4	
	Range	0.2-14.5 g/d		0.4-14.5 g/d	0.01-0.4 g/d	0.4-14.5 g/d	0.4-1.2 g/d	
ANA positive	n	114		25		25	3	
anti-dsDNA positive	n	101		25		25	3	
Depressed serum C3 level	n	78		25		25	2	
	Mean	0.67±0.6		0.54±0.5		0.54±0.5	0.74±0.5	
	Range	0.2-1.4		0.2-1.4		0.2-1.4	0.4-1.5	
ISN/RPS class								
Class I	n	5		-		**2**	1	
Class II	n	12		**3**		**3**	1	
Class III	n	13		**7**		**7**	1	
Class IV	n	35		**15**		**15**	0	
Class V	n	42		-		**13**	0	
Medication								
Prednisolone dose (mg)	dose	3.8±0.7		7.8±0.6		6.3±0.7	5.4±0.4	
Prednisolone	n	102		20		31	3	
Mycophenolat mofetil	n	35		14		20	2	
Cyclophophamide (only as induction therapy)	n	24		13		13	0	
Azathioprin	n	32		4		4	0	
Hydroxychlororquin	n	81		20		32	3	
Angiotensin-blocking agent	n	53		14		26	2	
				includes only LN		includes only LN	*Western Blot (WB)	
				type II-IV		type I-V		

Supplement Table 2. Antibodies used for immunostaining.

Immunofluorescence			
Antigen	**Clone**	**Cat. No.**	**Supplier**
IgG	30-F11	55493	Cappel
C3	1A8	55500	Cappel
Ki-67	SP6	VP-RM04	Vector Laboratories
CD19	6D5	115508	BioLegend
F4/80	BM-8	MF48000	Invitrogen
Immunohistochemistry			
Mouse			
Antigen	**Clone**	**Cat. No.**	**Supplier**
F4/80	BM-8	MF48000	Invitrogen
CD4	RM4-5	14-0042-85	eBioscience
B220	RA3-6B2	14-0452-85	eBioscience
Cleaved Caspase-3	Polyclonal	9661	Cell Signaling Technology
CD3	SP7	16669-500	Abcam
F4/80	CI:A3-1	MCAP 497	Serotec
CD20	M-20	Sc-7735	Santa Cruz
Human			
Antigen	**Clone**	**Cat. No.**	**Supplier**
IL-34	C-19	sc-243072	Santa Cruz Biotechnology
CSF-1	N-16	sc-1324	Santa Cruz Biotechnology
c-FMS	C-20	sc-692	Santa Cruz Biotechnology
PTPRZ	Polyclonal	ab126497	Abcam
CD68	PG-M1	M0876	Dako
CD3	SP-7	RM-9107-S1	Neomarkers
CD19	Polyclonal	ab99965	abcam
Western blot			
Mouse			
Antigen	**Clone**	**Cat. No.**	**Supplier**
PTPRZ	3F8	-	Developmental Studies Hybridoma Bank
α-Tubulin	TU-02	sc8035	Santa Cruz
ERK2:p42 MAPK	Polyclonal	9108	Cell Signaling
GAPDH	0411	sc-47724	Santa Cruz
Human			
Antigen	**Clone**	**Cat. No.**	**Supplier**
PTPRZ	122.2	sc33664	Santa Cruz
GAPDH	14C10	2118	Cell Signaling
ß-Actin	13E5	4970	Cell Signaling
ERK: p44/42 MAPK	Polyclonal	9102	Cell Signaling

Supplement Table 3. qPCR primers to detect mRNAs.

Gene	Forward primer sequence	Reverse primer sequence
β-actin	5'- GATTACTGCTCTGGCTCCTAGC -3'	5'-GACTCATCGTACTCCTGCTTG-3'
Bca-1/Cxcl13	5'- CTCTCCAGGCCACGGTATT -3'	5'- TAACCATTTGGCACGAGGAT -3'
Csf1	5′- GGCTTGGCTTGGGATGATTCT -3'	5′- GAGGGTCTGGCAGGTACTC -3′
Fms	5′- TGTCATCGAGCCTAGTGGC -3′	5′- CGGGAGATTCAGGGTCCAAG -3′
Gapdh	5'-AGGTCGGTGTGAACGGATTTG-3′	5'- TGTAGACCATGTAGTTGAGGTCA -3′
Ifn-γ	5'- AGCTCTTCCTCATGGCTGTT -3'	5'- TTTTGCCAGTTCCTCCAGAT -3'
Il-1β	5'- GCCTCGTGCTGTCGGACCCA -3'	5'- TGAGGCCCAAGGCCACAGGTAT -3'
Il-10	5'-GCTCTTACTGACTGGCATGAG-3'	5'- CGCAGCTCTAGGAGCATGTG-3'
Il34	5′- TTGCTGTAAACAAAGCCCCAT -3′	5′- CCGAGACAAAGGGTACACATTT -3′
Ip10/Cxcl10	5'- TGAAATTATTCCTGCAAGCCAA -3'	5'- CAGACATCTCTTCTCACCCTTCTTT -3'
I-tac/Cxcl11	5'- AGTAACGGCTGCGACAAAGT -3'	5'- GCATGTTCCAAGACAGCAGA -3'
Mcp-1/Ccl2	5'- GCTTGAGGTGGTTGTGGAAAA- 3'	5'- CTCACCTGCTGCTACTCATTC -3'
Mig/Cxc9	5'- TCCTTTTGGGCATCATCTTC -3'	5'- TTCCCCCTCTTTTGCTTTTT -3'
Mip1a/Ccl3	5'- TCTCCACCACTGCCCTTGCT -3'	5'- GGCGTGGAATCTTCCGGCTGT -3'
Ptprz1	5'- GGAGTATCCAACAGTTCAGAGGC -3'	5'- AAGTCAGGGCAGACACGATCAC -3'
Rantes/Ccl5	5'- TGCCAACCCAGAGAAGAAGT-3'	5'- AAGCTGGCTAGGACTAGAGCAA -3'
Tnf-α	5'- CCCTCACACTCAGATCATCTTCT -3'	5'- GCTACGACGTGGGCTACAG -3'

Supplement Table 4. Antibodies used for FACS.

Antigen	Clone	Fluorochrome	Supplier
CD45	30-F11	Pacific blue	BioLegend
Ly6G	1A8	APC-Cy7	BioLegend
CD11b	M1/70	Brillant Violet 510	BioLegend
F4/80	BM-8	PE-Cy7	BioLegend
CD19	6D5	APC	BioLegend
CD3e	145.2C11	PE	eBioscience
E-cadherin	DECMA-1	Alexa-Fluor 647	BioLegend
Annexin-V	-	APC	BioLegend
Ki-67	16A8	FITC	BioLegend
IL-10	JES5-16E3	PE-Cy7	BioLegend
TNFa	MP6-XT22	PerCP/Cy5.5	BioLegend
iNOS	CXNFT	PE	eBioscience
Arginase-1	Polyclonal	FITC	R&D Systems
CD86	GL-1	PE-Cy7	BioLegend
CD69	H1.2F3	APC-Cy7	BioLegend
CD23	B3B4	PE	eBioscience
BrdU	Bu20a	PE	BioLegend
Cleaved Caspase-3	Asp175	Unconjugated	Cell Signaling Technology
Rat IgG2a	RTK2758	PE/Cy7	BioLegend
Rat IgG2b	30-F11	Pacific Blue	BioLegend
Rat IgG1	G0114F7	PerCP/Cy5.5	BioLegend
Armenian Hamster IgG	HTK888	PE	BioLegend
Rat IgG2a	RTK2758	PE	BioLegend
Rat IgG2a	RTK2758	APC/Cy7	BioLegend
Rat IgG2a	eBR2a	APC	eBioscience
Rat IgG2a	RTK2758	FITC	BioLegend
Anti-rabbit IgG	Poly4064	PE	BioLegend