Abstract P95 Table 1 Laboratory and Clinical Outcomes for Patient 1 and 2 Through After CAR T-Cell Infusion

	Patient 1						Patient 2			
Parameter, units [reference range]	**Pre-LD**	**Day 0**	**Day 14**	**Day 28**	**Day 56**	**Day 90**	**Pre-LD**	**Day 0**	**Day 14**	**Day 28**
CD19+ B cells, cells/μL [107–698]	22	0	0	1	7	102	52	3	0	0
ANC, $\times 10^9$/L [1.80–7.00]	4.5	0.5	3.7	3.6	3.8	4.7	3.8	1.7	1.1	6.2
Hemoglobin, g/dL [11.5–15.5]	12	9.1	9.5	11.7	12.6	12.9	9	7.9	9.2	9.3
Platelets, $\times 10^9$/L [150–400]	93	54	78	103	175	201	341	213	245	453
CRP, mg/L [0–10]	83.3	67.5	4.8	2.8	11.8	9.7	10.9	7.6	6.4	5.4
Anti-dsDNA, IU [<25 = Negative]	297	294	356	304	216	241	293	432	386	281
C3, mg/dL [83–193]	54	59	65	94	135	146	62	58	82	100
C4, mg/dL [15.0–57.0]	9.2	13	18.7	28.8	44.2	41.6	4.9	5.3	9.7	12.8
Proteinuria, [UPCR]	1.4	1.5	0.6	0.9	0.5	0.5	1.3	1.9	1.2	0.6
SLEDAI-2K	19	22	14	13	8	8	12	8	16	10
IgG, mg/dL [757–1941]	620	546	452	429	291	287	1254	970	1017	873
IgM, mg/dL [33–393]	87	73	23	12	5	20	106	85	80	70
IgA, mg/dL [87–534]	118	113	62	46	37	37	370	307	275	190

ANC, absolute neutrophil count; C3, complement component 3; C4, complement component 4; CRP, C-reactive protein; dsDNA, double-stranded DNA; Ig, immunoglobulin; LD, lymphodepletion; SLEDAI-2K, Systemic Lupus Erythematosus Disease Activity Index 2000; UPCR, urine protein creatinine ratio.

Results Two patients have been treated at DL1 with 90 and 28 days of follow-up. Patient 1 is an 18-year-old female patient diagnosed with SLE at age 9 with class IV LN with persistent proteinuria refractory to multiple immunosuppressive therapies. Patient 2 is a 28-year-old female with SLE since 2021 who had failed numerous immunosuppressive therapies for persistently active class IV LN.

After CAR T cell infusion, both patients experienced grade 1 cytokine release syndrome consisting of fever (patient 1 on days 5 and 6; patient 2 on days 10 and 11). No immune effector cell-associated neurotoxicity syndrome, DLTs, or serious AEs occurred. KYV-101 rapidly expanded and B cell depletion was observed with evidence of B-cell recovery observed in patient 1 starting on day 56. Both patients showed improvement in LD associated cytopenias and normalization of CRP and complement levels paralleled their clinical improvement (table 1).

Conclusion While preliminary, these data demonstrate that KYV-101 was well tolerated with evidence of clinical improvement underscoring the potential of anti-CD19 CAR T-cell therapy for treating LN. To date, 9 autoimmune patients have been treated with KYV-101 and a phase 1/2 trial of LN in Europe, KYSA-3, is also ongoing.

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P96 PROGNOSTIC FACTORS FOR CHRONIC KIDNEY DISEASE IN PATIENTS WITH CLASS V LUPUS NEPHRITIS IN 4 REFERENCE CENTERS IN MEXICO

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Objective Analyze prognostic factors for chronic kidney disease progression in Mexican patients with class V lupus nephritis (LN), comparing them with mixed classes.

Methods Retrospective cohort, 74 patients 12–80 years old from 4 different hospitals: 33% LN class V, 23% LN class III +V and 43.2% LN class IV+V.

Results Sixty-two females (83.7%), mean age 33 years old (12–63), 59.5% had hypertension, 6.8% had type 2 diabetes and 8.1% had antiphospholipid antibody syndrome. Twelve-month follow-up eGFR <60 mL/min/1.73m^2 was similar among classes III+V and IV+V LN 40.8% vs class V LN 40%. Even with higher initial eGFR (61 vs 105 mL/min/1.73m^2 p=0.017) and despite adequate treatment, eGFR at 12 months follow-up in class V patients remained the same proportion (56 vs 96 mL/min/1.73m^2 p= 0.105). The percentage of patients with class III+V and IV+V vs class V with eGFR <60 mL/min/1.73m^2 at 12-month follow-up was similar: 40.8% vs 40% (p=0.946). eGFR <60 mL/min/1.73m^2 decline was seen in 55.1% of classes III+V and IV+V group and in 40% of class V group. There was no significant difference in factors for baseline proteinuria at 3, 6, 12, or 24 months in both groups, although those with higher levels of

Abstract P96 Table 1

Variable	NL III+V/IV+V	NL V	p value
Age (years)	31.8 (13.2)	36 (12.4)	0.119
eGFR on admission (mL/min/1.73m^2)	61 (64)	105 (78)	0.017
eGFR 12 months follow-up (mL/min/1.73m^2)	73 (69)	94 (71)	0.128
Proteinuria on admission (g/day)	3.8 (5.0)	3.2 (5.8)	0.575
Proteinuria 12 months follow-up (g/day)	1.5 (2.6)	1.0 (3.0)	0.71
Hypertension	29 (59.2%)	15 (60%)	0.946
Complete remission	20 (40.8%)	16 (64%)	0.059
Partial remission	10 (20.4%)	3 (12%)	0.522
Relapse	3 (6.1%)	5 (20%)	0.11
No response	21 (42.9%)	6 (24%)	0.11
eGFR <60 mL/min at 12 months	20 (40.8%)	10 (40%)	0.946
Proteinuria >0.5 g/day at 12 months	35 (71.4%)	15 (60%)	0.32
Proteinuria >3.5 g/day at 12months	9 (18.4%)	5 (20%)	1.0

proteinuria had lower eGFR (p=0.0009) (table 1). Most patients received mycophenolic acid (92%), azathioprine (12%), and other immunosuppressive drugs, including tacrolimus (36%) and/or cyclophosphamide (56%). As for other medications, 56% received ACE inhibitors, 40% ARA2, 88% hydroxychloroquine and 80% statins. Only two thirds with class V LN had complete remission (64% vs 40.8% p=0.059), partial remission 12%, relapse 20% and no response 24%. Forty percent of patients had eGFR deterioration < 60 mL/min/1.73m^2 at 12 or more months (table 1).

Conclusion Mexican patients with class V LN present with greater deterioration of renal function, frequently with arterial hypertension (60%). eGFR either maintains or worsens at 12 or more months of follow-up, even with adequate/intensive treatment, compared to what is reported in the literature.

P97 LNMAP: A CURATED COMPUTATIONAL RESOURCE OF MOLECULAR PATHWAYS IN LUPUS NEPHRITIS

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Objective The generation of an open-access interactive lupus nephritis disease map (LNMap). The map will be a reference resource depicting the complex molecular pathways underlying LN pathogenesis and the key regulators of the disease. This graphical and computational repository will integrate existing knowledge, identify gaps and provide a scaffold for reproducible molecular signaling models of LN.

Methods Major review papers were used to identify the most important cell types and pathways associated with LN. Manual literature search focusing on the selected cell types was performed to detect inter- and intracellular interactions associated with LN. Molecular interactions between and within these cell types that have been associated with LN undergo manual literature curation. These interactions are depicted utilizing CellDesigner software and Systems Biology Graphical Notation and annotated using Minimum Information Required In the Annotation of Models (MIRIAM). Identified gaps in knowledge will be completed by data-driven, automated mining of literature and interaction databases.

Results LNMap will be comprised of molecular interactions between and within neutrophils, macrophages, mesangial cells, CD4+ T-cells, CD8+ T-cells, dendritic cells, monocytes, podocytes and B-cells. The map currently is comprised of a kidney compartment that contains sub-compartments for each selected cell type (figure 1A). At this stage, 164 publications are present in the database of the LNmap, focusing on dendritic (N=30) and mesangial (N=64) cells (figures 1B, C), CD4+ T cells (N=31), podocytes (N=16), neutrophils (N=11), macrophages (N=1), monocytes (N=4) and B cells (N=11).

Conclusions The LNmap, an ongoing dynamic project, will provide a useful resource of known pathways associated with the disease and will pinpoint gaps in our knowledge. The LNmap already constitutes a resource of molecular interactions with a multifaceted value aiming to assist anyone interested in the molecular landscape of LN. Future efforts are designed for the map to reach its end-users through the MINERVA Platform.

P98 BONE MORPHOGENETIC PROTEIN-7 AS TREATMENT IN LUPUS NEPHRITIS: GETTING FROM BENCH TO BEDSIDE

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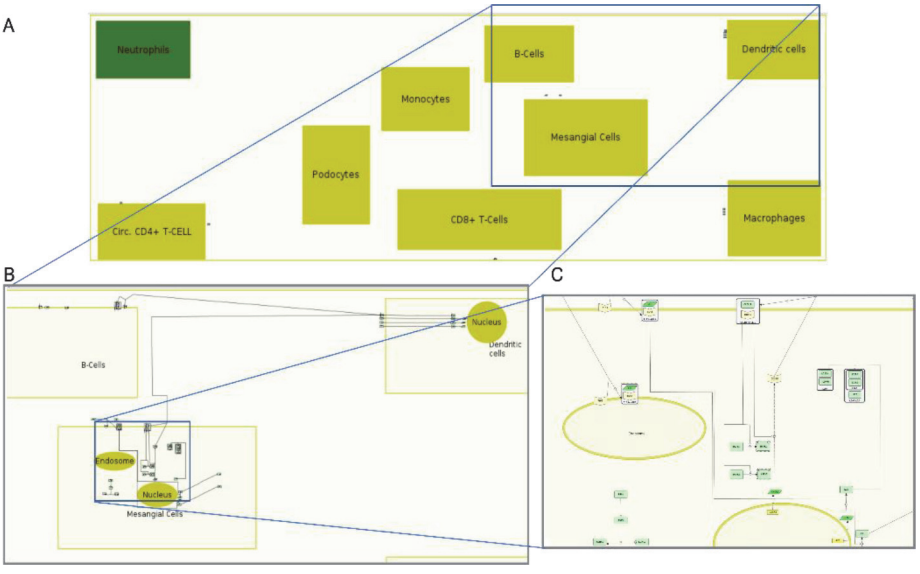
Objective A large body of literature suggests that prevention of fibrosis – which contributes to chronicity of lupus nephritis (LN) - may significantly improve long-term prognosis. Bone morphogenetic protein-7 (BMP-7) was first identified



Abstract P97 Figure 1 (A) The Lupus nephritis Map. (B) Interactions between dendritic cells, mesangial cells and B cells. (C) Mesangial cell interactions