meaningful modules, including yellow, turquoise, grey60 and blue, were identified (Figure 1A,1B). And 183 overlapping gene were screened from the DEGs and the Hub genes in the four modules for further analysis. We final divided pSS patients into three subtypes, of which yellow and turquoise in Sub1, grey60 in Sub2 and blue in Sub3. Sub1 and Sub3 were related to cell metabolism, while Sub2 had connection with virus infection (Figure 1C,1D). Infiltrated immune cells were also different among these three types (Figure 1E,1F).

Conclusion: Patients with pSS could be classified into 3 subtypes, this classification might help for assessing prognosis and guiding precise treatment.

REFERENCES:

- [1] Ramos-Casals M, Brito-Zerón P, Sisó-Almirall A, et al. Primary Sjogren syndrome. *BMJ* (Clinical research ed) 2012;344:e3821. doi: 10.1136/bmj.e3821 [published Online First: 2012/06/16].
- [2] Brito-Zerón P, Baldini C, Bootsma H, et al. Sjogren syndrome. *Nat Rev Dis Primers* 2016;2:16047. doi: 10.1038/nrdp.2016.47 [published Online First: 2016/07/08].
- [3] Segal B, Bowman SJ, Fox PC, et al. Primary Sjogren's Syndrome: health experiences and predictors of health quality among patients in the United States. *Health Qual Life Outcomes* 2009;7:46. doi: 10.1186/1477-7525-7-46 [published Online First: 2009/05/29].

Figure: (A,B) Heatmap of the correlation between module eigengenes and clinical traits of SS. (C,D) Molecular typing of patients with SS based on ferroptosis-related gene expression profiles consensus clustering of GEO samples by NMF, a heatmap displaying the expression pattern of subtypes is shown. (Figure E,F) The scores of 23 immune cells are displayed in boxplots. Adjusted P values were showed as: ns, not significant; *P<0.05; **P<0.01; ***P<0.001.

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POS0110

ASSOCIATION OF miR-155 AND miR-34a EXPRESSION IN LUPUS NEPHRITIS RENAL TISSUE WITH DISEASE ONSET AND OUTCOMES

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Background: Epigenetic factors such as non-coding RNA (miRNA) have been shown to be deregulated in Systemic Lupus Erythematosus (SLE). In mouse models, different miRNAs have been associated with lupus nephritis (LN), one of the most severe manifestations of the disease¹.

Objectives: To evaluate the expression of miR-155 and miR-34a in renal tissues as biomarkers of organ involvement and inflammatory tissue activity in patients with LN.

Methods: Thirty-two SLE patients with LN (age: 32.2 ± 9.2 years) with active renal involvement undergoing ultrasound-guided renal biopsy were enrolled between 2010 and 2019. The nephritic manifestation was present in 13 (41%) patients at disease onset (early-LN SLE), while 19 (59%) patients showed a renal involvement after disease onset (long-LN SLE, mean disease duration at LN onset: 7.3 ± 5.7 years). Twelve age-matched patients with IgA nephropathy were enrolled as control group. Clinical, laboratory and demographic data were collected for each patient. Disease activity was recorded using SLE-DAI-2K and renal activity, using the total SLEDAI-2K fraction including the items related to the renal involvement. MiR-155 and miR-34a expression in renal tissues was carried out by extraction of total RNA from paraffin-preserved biopsies and was evaluated, after a retrotranscription protocol, using real-time PCR by relative quantification considering the ΔCt (Ct miRNA- Ct housekeeping gene)².

Results: MiR-155 and miR-34a expression in renal tissues was higher in LN-SLE patients as compared to IgA nephropathy patients (ΔCt miR-155: 9.4 ± 10.1 vs 21.9 ± 3.6, p<0.01; ΔCt miR-34a: 10.1 ± 9.8 vs 19.2 ± 3.1, p=0.02). MiR-155 and miR-34a expression in LN-SLE patients renal tissues was comparable in the different histological classes. Furthermore, a direct correlation was observed between the expression of miR-155 and miR-34a (r = 0.91, p <0.001). Dividing patients based on nephritic onset, SLE patients with long-LN showed higher expression of miR-155 (ΔCt 6.1 ± 8.7) and miR-34a (ΔCt 7.1 ± 9.0) as compared to patients with early-LN (miR-155: ΔCt 13.4 ± 10.6 p = 0.08; miR-34a: ΔCt 15.1 ± 9.5 p = 0.02) or patients with IgA nephropathy (miR-155 p<0.01 and miR-34a p<0.01). Moreover, in early-LN SLE it was observed an inverse correlation between miR-34a expression and C3 and C4 complement components (r=-0.72; p=0.05 and r=-0.86; p=0.01, respectively) and a direct correlation between miR-155 and 24h-UP (r=0.67; p=0.03). Considering SLE patients with early-LN, the expression of miR-34a was slightly significant in patients who had relapsed (ΔCt 8.2 ± 11.4 vs ΔCt 18.4 ± 7.9 p = 0.08), although no correlation emerged between the expression of miR-155 and miR-34a at the time of the biopsy and with disease activity indices.

Conclusion: MiR-155 and miR-34a may represent tissue biomarkers of inflammatory activation in SLE patients with LN; in particular, the higher expression of these miRNA in long-LN and the correlation between miR-155 expression with p24h-UP in early-LN could indicate a possible role of these biomarkers in renal involvement in patients with SLE with later renal onset. The increased expression of miR-34a could give indications of a disease recurrence suggesting a closer monitoring of patients.

REFERENCES:

- [1] Leiss H et al. *Plosone* 2017.
- [2] Alivernini S et al. *Nat Commun* 2018.

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POS0111

DEVELOPMENT OF DAMAGE AND MORTALITY IN AN INCEPTION COHORT OF SLE PATIENTS

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Background: There had been very limited data on the development of damage and mortality in an inception cohort of SLE patients who were recruited very soon after diagnosis.