Systemic Lupus Erythematosus (SLE) is an autoimmune chronic disease characterized clinically by periods of flares and remission. Although its pathophysiology has not yet been well understood, it is well documented that SLE courses with systemic inflammation, as well as affection to specific organs [1]. Lupus nephritis (LN) is the most common complication and cause of death appearing in SLE patients, often leading to end-stage renal disease [2]. A type I interferon (IFN) signature is present in SLE, however, there is no consensus on which genes are expressed in LN. We performed a meta-analysis through integrative bioinformatics to identify differentially expressed genes (DEGs) in patients with LN.

**Background:** Systemic Lupus Erythematosus (SLE) is an autoimmune chronic disease characterized clinically by periods of flares and remission. Although its pathophysiology has not yet been well understood, it is well documented that SLE courses with systemic inflammation, as well as affection to specific organs [1]. Lupus nephritis (LN) is the most common complication and cause of death appearing in SLE patients, often leading to end-stage renal disease [2]. A type I interferon (IFN) signature is present in SLE, however, there is no consensus on which genes are expressed in LN. We performed a meta-analysis through integrative bioinformatics to identify differentially expressed genes (DEGs) in patients with LN.

**Objectives:** We aimed to identify and assess overlapped DEGs of patients with LN through integrative bioinformatics and functional enrichment analysis.

**Methods:** We designed a search strategy in the Gene Expression Omnibus platform to identify datasets of expression profiling by array of kidney samples of patients with LN. The inclusion criteria were: 1) Presence of healthy controls in the datasets, in which the IFN signature is present. 2) Analysis of data with GEO2R. The exclusion criteria were: 1) Patients with LN. The inclusion criteria were: 1) Presence of healthy controls in the datasets, in which the IFN signature is present. 2) Analysis of data with GEO2R. The exclusion criteria were: 1) Patients with LN. The inclusion criteria were: 1) Presence of healthy controls in the datasets, in which the IFN signature is present.

**Results:** 3 datasets fulfilled the inclusion criteria and 9 DEGs were identified. Prediction analysis yielded 23 co-expressed genes. David database recognized the principal biological processes involving those genes (Table 1). PPI analysis recognized the top 10 genes with the strongest interactions, and proposed STAT1 as the regulatory target (Figure 1).

**Conclusion:** We aimed to identify and assess DEGs of patients with LN and recognize the ones with the strongest interactions. Our results suggest that type I IFN is crucial on the pathophysiology of LN, and STAT1 can serve as a possible therapeutic target for treating LN. We propose further in vivo studies to validate these results, given that they can serve as diagnostic biomarkers and even possible therapeutic targets for LN treatment.

**Keywords:** Biomarkers, -Oomics, Systemic lupus erythematosus

**Disclosure of Interests:** None Declared.

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**Table 1.** Top 3 biological processes involving overlapped DEGs among the datasets, in which the IFN signature is present.

<table>
<thead>
<tr>
<th>Biological Processes</th>
<th>Gene Count</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Defense Response to Virus</td>
<td>21</td>
<td>1.6E-33</td>
</tr>
<tr>
<td>Response to Virus</td>
<td>15</td>
<td>3.4E-25</td>
</tr>
<tr>
<td>Negative Regulation of the Viral Genome</td>
<td>11</td>
<td>1.7E-20</td>
</tr>
</tbody>
</table>

**REFERENCES:**

