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A Systems Biology Approach to Unravel the Common Genes and Pathways in Amyotrophic Lateral Sclerosis and Traumatic Brain Injury

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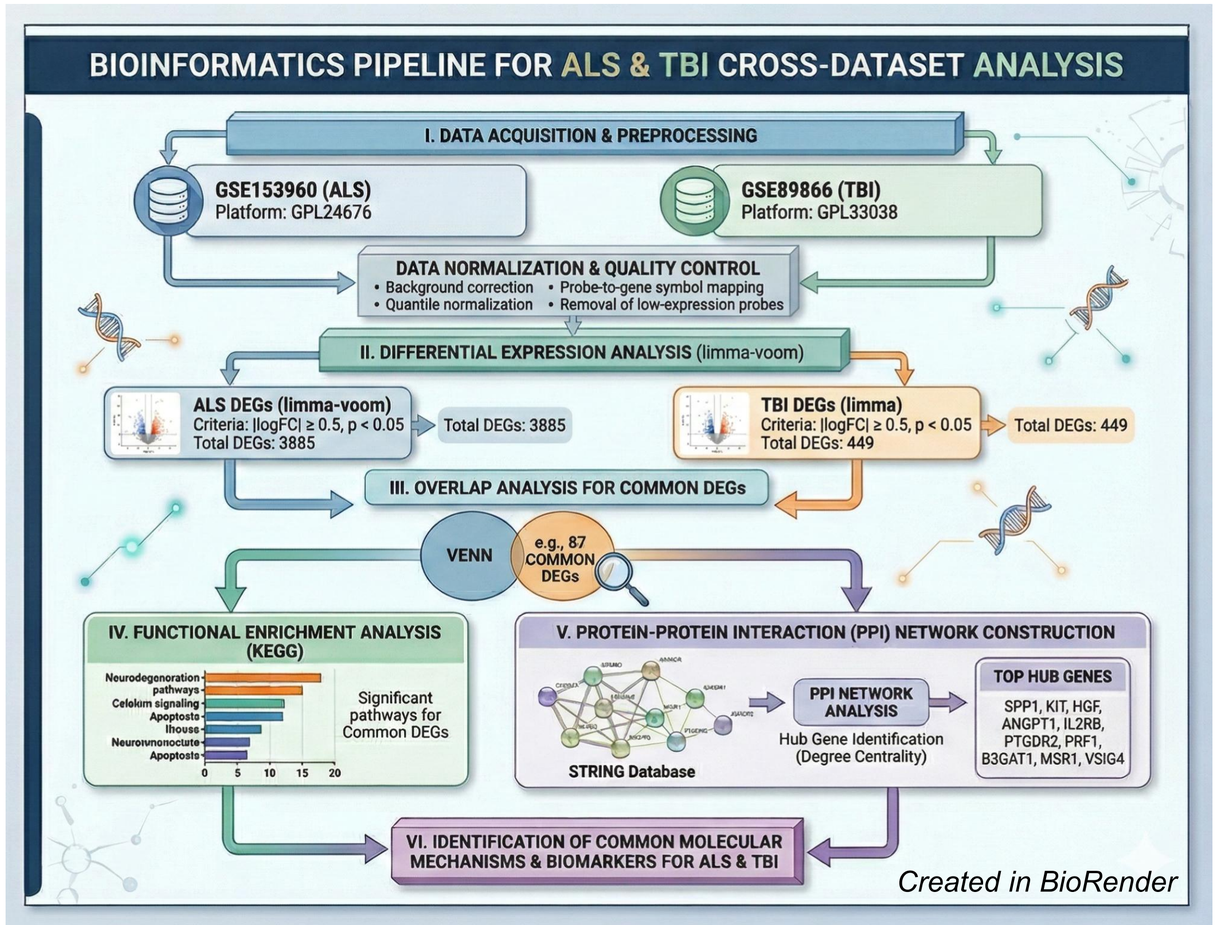
INTRODUCTION & AIM

• Amyotrophic Lateral Sclerosis (ALS) and Traumatic Brain Injury (TBI) are distinct neurological disorders.
• Both share overlapping pathological mechanisms including neuroinflammation, oxidative stress, and synaptic dysfunction.
• Understanding shared molecular signatures may reveal convergent pathways involved in neurodegeneration.
• A systems biology approach enables identification of shared gene signatures and pathway-level interactions that may not be evident through single-disease analysis.

Why Investigate ALS and TBI Together?
• TBI is a risk factor for later-life neurodegeneration
• Both disorders exhibit chronic neuroinflammation
• Shared molecular drivers may explain disease progression
• Cross-disease analysis can uncover conserved therapeutic targets

Aim:
To identify common differentially expressed genes (DEGs), hub genes, and enriched biological pathways linking ALS and TBI using an integrated systems biology approach.

METHOD



Data Source: NCBI GEO (https://www.ncbi.nlm.nih.gov/geo/)
– ALS: GSE153960
– TBI: GSE223245

Analysis Tool:

• **Differentially Expressed Genes (DEGs):-** limma package in R

• **Threshold :-** |logFC| ≥ 0.5 and p-value < 0.05

• **Protein–Protein Interaction (PPI) :-** STRING

• **Hub genes identification :-** Cytoscape (degree centrality method)

• **Functional enrichment analysis :-** clusterProfiler

RESULTS & DISCUSSION

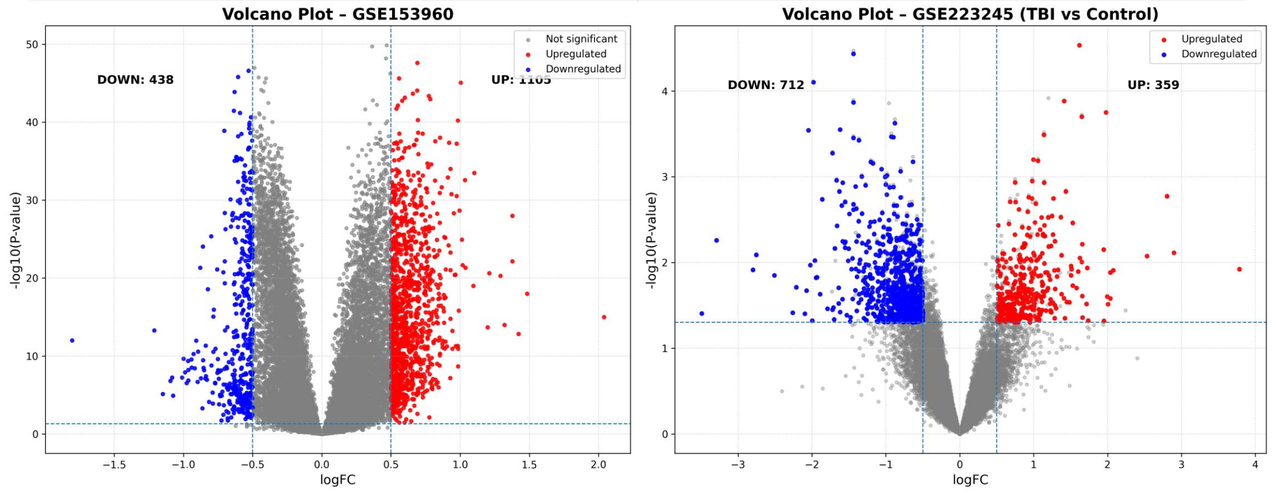


Figure 1. Significant transcriptomic alterations observed in ALS and TBI datasets

Figure 2. Overlapping DEGs identified between ALS and TBI

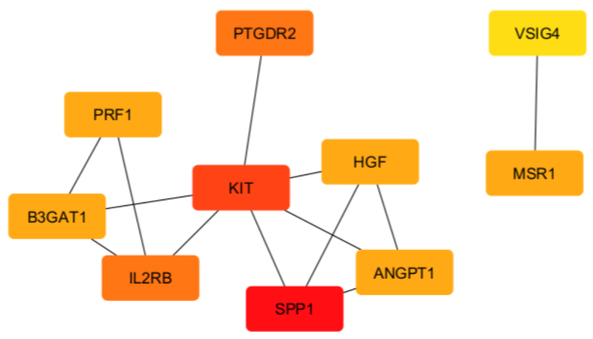


Figure 4. Top 10 hub genes

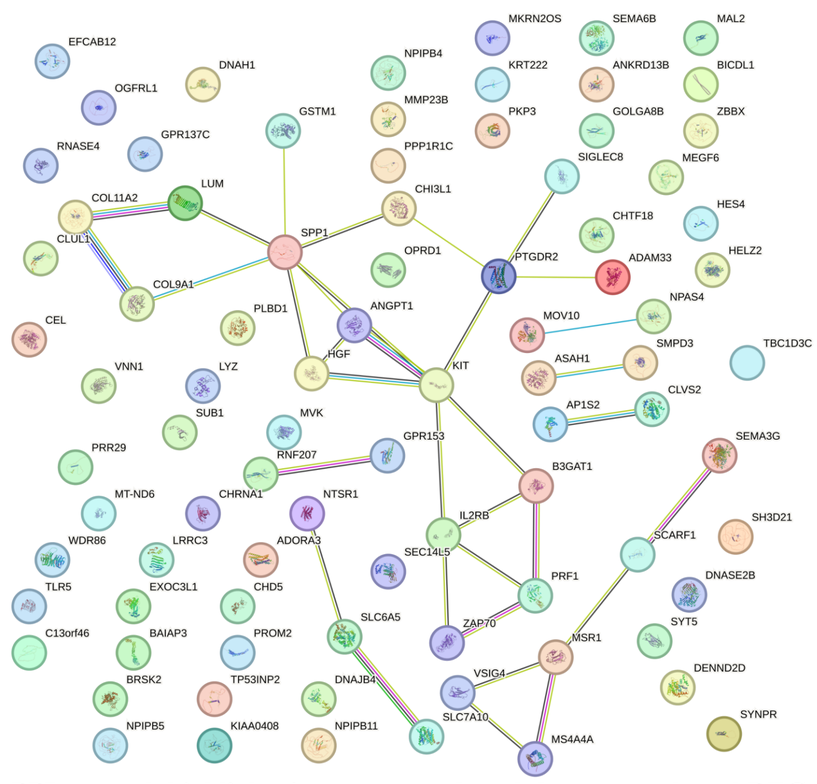


Figure 3. PPI network highlights interconnected modules among shared DEGs

Term	Overlap	P-value	Adjusted P-value	Old P-value	Old Adjusted P-value	Odds Ratio	Combined Score	Genes
NEUROACTIVE LIGAND SIGNALING	4/195	0.0104	0.37	0	0	4.9762	22.7208	OPRD1;SLC6A5;OGFRL1;ADORA3
SPHINGOLIPID SIGNALING PATHWAY	3/123	0.0165	0.37	0	0	5.8908	24.1629	OPRD1;ASAH1;ADORA3
LYSOSOME	3/132	0.0199	0.37	0	0	5.4773	21.4493	ASAH1;DNASE2B;AP1S2
NEUROACTIVE LIGAND-RECEPTOR INTERACTION	5/366	0.0218	0.37	0	0	3.3025	12.6394	CHRNA1;OPRD1;OGFRL1;ADORA3;NTSR1
SPHINGOLIPID METABOLISM	2/54	0.0231	0.37	0	0	8.9869	33.8526	SMPD3;ASAH1

CONCLUSION

• 87 shared DEGs identified between ALS and TBI
• Network analysis revealed 10 key hub regulators
• Enrichment highlights immune signalling and stress-response pathways as commonly enriched
• Shared molecular architecture suggests convergent neurodegenerative mechanisms

FUTURE WORK / REFERENCES

• Integration of additional transcriptomic datasets to enhance robustness.
• Exploration of therapeutic targeting of key hub genes and enriched pathways.
• Multi-omics integration (proteomics, epigenomics) to better understand regulatory mechanisms.

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