

# Transcriptomic Analysis Identifies ACSL4 And GPX4 As Potential Regulators Of Ferroptosis Sensitivity in Oral Squamous Cell Carcinoma In The MOCCA (Pakistani) Cohort



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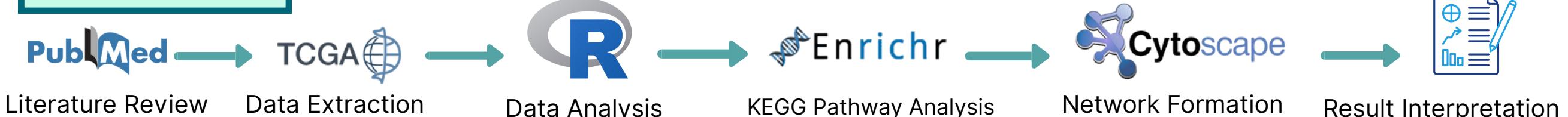
## INTRODUCTION

Oral squamous cell carcinoma is the most common cancer among males in Pakistan with a survival rate of around less than 50%, mainly because cancer cells are resistant to the currently available therapeutic options.<sup>1</sup> Ferroptosis, an iron-dependent form of regulated cell death driven by lipid peroxidation, has recently emerged as a potential therapeutic vulnerability in cancer.<sup>2</sup>

## OBJECTIVES

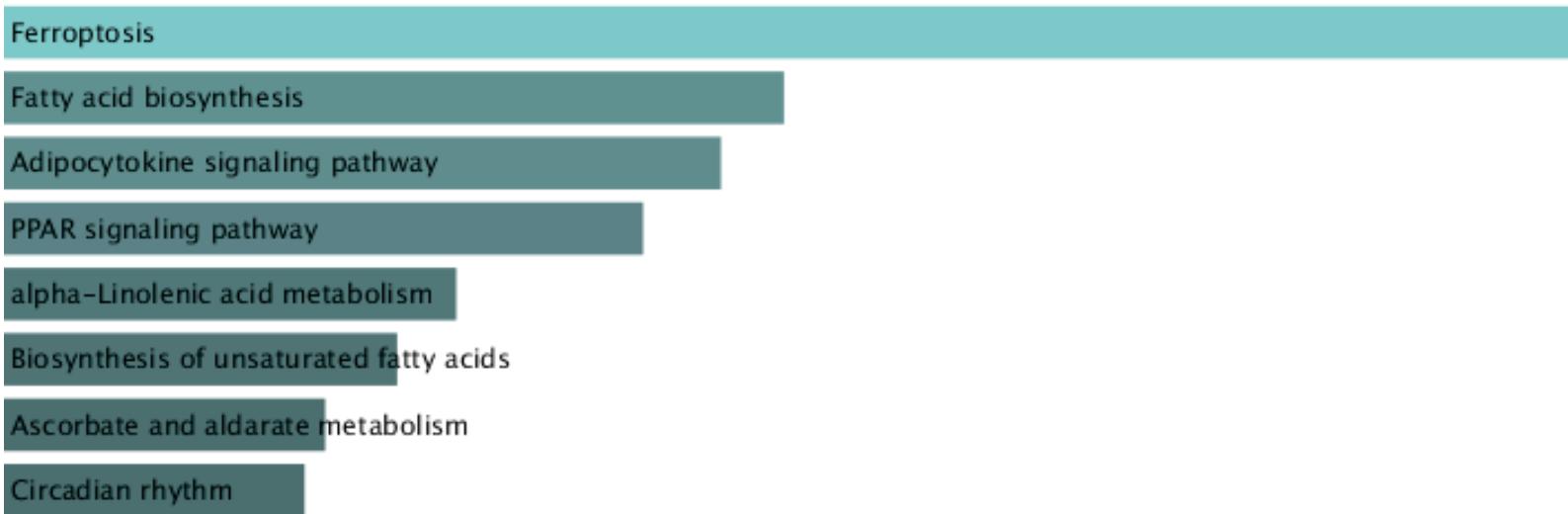
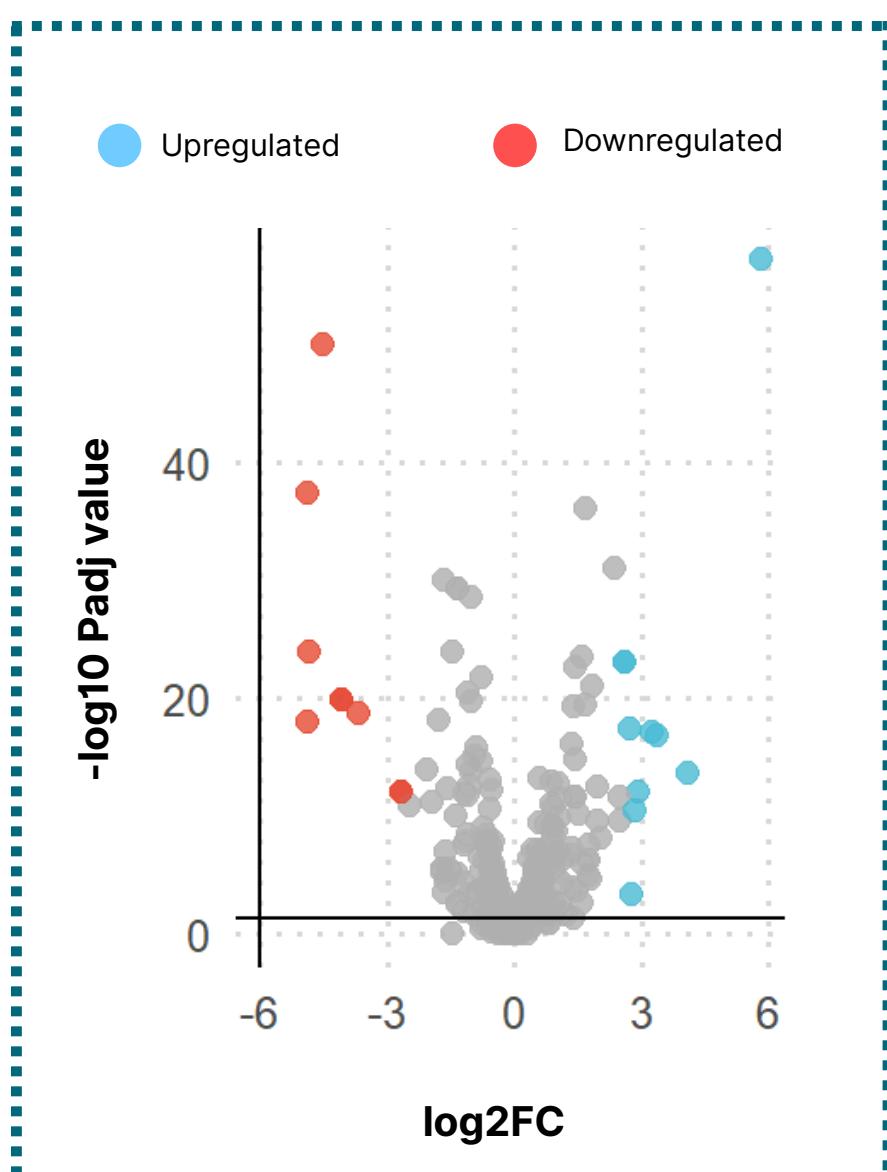
- Analyze transcriptomic data of Oral Squamous Cell Carcinoma (OSCC) for ferroptosis-associated gene expression patterns.
- Identify differentially expressed genes linked to ferroptosis sensitivity in OSCC.

## METHODOLOGY

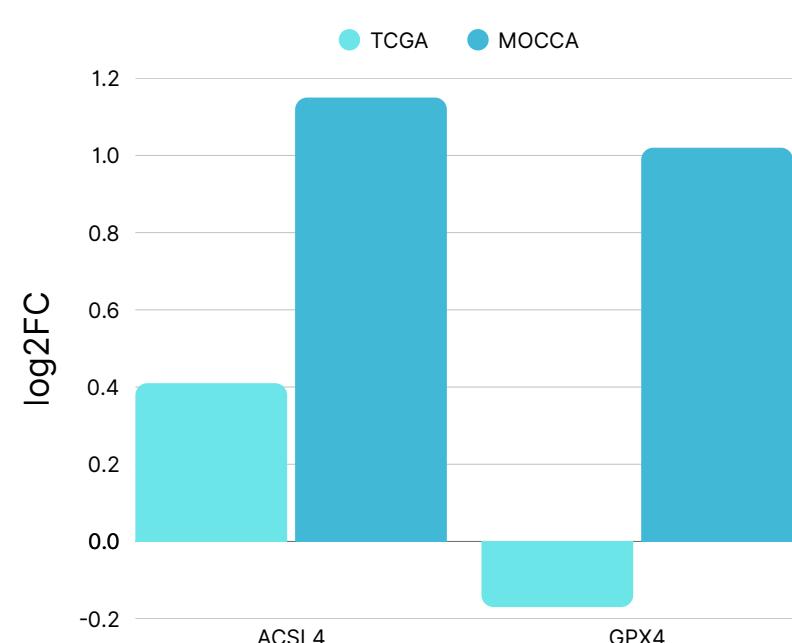
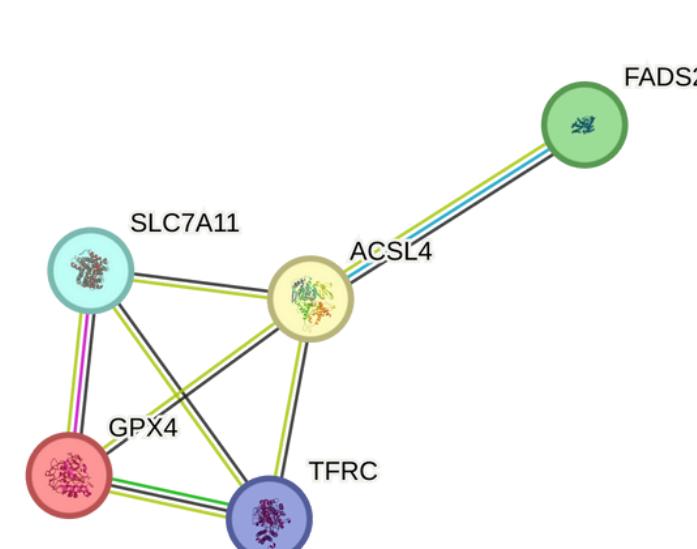


## RESULTS

### Pathway Enrichment Analysis



**Figure 2.** Bar Chart showing KEGG Pathway Enrichment Analysis of the significant genes. The most enriched pathway was found to be ferroptosis.



**Figure 3.** String network of genes involved in the process of ferroptosis, with ACSL4 being the central gene in this network.

**Figure 4.** Difference in the expression of ACSL4 and GPX4 in TCGA and MOCCA.  
MOCCA - PML's Multiomic data of local OSCC patients

## CONCLUSION

Oral squamous cell carcinoma exhibits sensitivity to ferroptosis, as reflected by distinct gene expression signatures. These transcriptomic insights highlight ferroptosis as a potential therapeutic vulnerability in OSCC.

## WAY FORWARD

Future directions include conducting wet-lab experiments to investigate how metabolic modulation of tumor cells influences ferroptosis sensitivity and overall viability, thereby providing functional validation of the transcriptomic findings.

## REFERENCES

- Tranby et al (2022) Oral Cancer Prevalence, Mortality, and Costs <https://doi.org/10.1158/1055-9965.EPI-22-0114>
- Dixon et al (2012) Ferroptosis: an iron-dependent form of nonapoptotic cell death <https://doi.org/10.1016/j.cell.2012.03.042>

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