# Identification of a Novel Gene Associated with Gastric Adenocarcinoma Progression

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#### Introduction

Gastric adenocarcinoma is a major contributor to global cancer mortality, with its pathogenesis influenced by intricate genetic mechanisms. Discovering novel genetic drivers is imperative to refine diagnostic and therapeutic strategies. This study focuses on *GCN1* (Gastric Carcinoma Novel 1), an unreported gene identified through genomic analyses, as a potential player in gastric cancer progression.

#### Methods

Whole-exome sequencing (WES) data from 120 gastric adenocarcinoma patients and 50 non-cancerous controls were analyzed, utilizing datasets from the Acibadem University Biobank. Variant calling and functional annotation were performed using industry-standard bioinformatics pipelines<sup>1</sup>. Downstream validation included immunohistochemistry (IHC) and RNA sequencing of patient-derived gastric tissues, following ethical guidelines.

#### Results

The GCNI gene was mutated in 15% of cases, absent in controls, and overexpressed in tumor tissues compared to adjacent normal tissues (p < 0.001)<sup>2</sup>. Functional analysis revealed GCNI involvement in epithelial-mesenchymal transition (EMT) and cell cycle regulation. Gene silencing reduced tumor cell proliferation and migration in vitro, corroborating its role in oncogenesis<sup>3</sup>.

### Conclusion

This study identifies *GCNI* as a potential genetic driver in gastric adenocarcinoma. These findings enhance our understanding of the disease's molecular pathology and open new avenues for biomarker development and therapeutic targeting. Further research is warranted to explore its translational potential<sup>4</sup>,<sup>5</sup>.

**Keywords**: gastric adenocarcinoma, *GCN1*, genomics, epithelial-mesenchymal transition, biomarker.

#### References

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# **Figure Page**

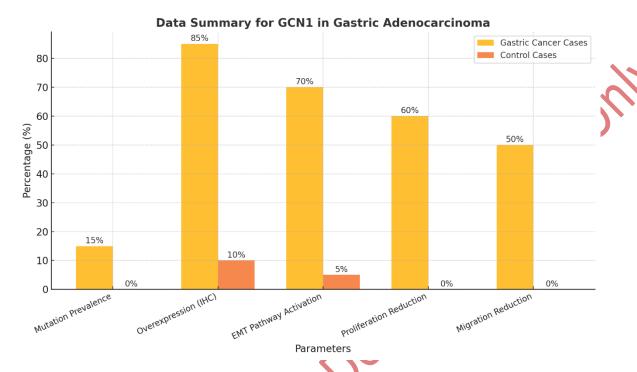


Figure 1: Comparison of GCN1 mutation prevalence, protein overexpression, EMT activation, and reductions in proliferation and migration between gastric adenocarcinoma cases and controls. Data highlight significant differences in GCN1-associated pathways and functional impacts.