Introduction

- Gastric cancer is the third leading cause of cancer death globally
- *heyl* is upregulated in gastric cancer patients and significantly correlated to patient survival rates

Objective of the Study

To study the role of HEYL, a potential transcriptional repressor, in gastric cancer progression

Methods

- Transfected BGC cells with constructed plasmids containing *myc-heyl*
- Western Blot to confirm BGC cells' *heyl* overexpression
- Phenotypic assays to study the effects of *heyl* overexpression in BGC cells

Results

Western Blot

- Confirms BGC cells' *heyl* overexpression post-transfection

Transwell Migration and Invasion Assays

- Significant increase in BGC cell migration and invasion with *myc-heyl* overexpression 24 hours post-transfection
- BGC cells exhibited greater invasion abilities than migration abilities

MTT Assay

- BGC cells with *myc-heyl* overexpression have a slightly lower rate of MTT incorporation than BGC cells with control expression levels
- Thence, BGC cells with *myc-heyl* overexpression have slightly lower viability and metabolic activity compared to BGC cells with control expression levels

Discussion

- *heyl* upregulates gastric cancer migration and invasion
- MTT data contradicts previous MTT data for HGC cells' shRNA knockdown experiments; further repeats, preferably with stable cell line, are required to confirm results
- Speculate that the mechanism of HEYL involves epigenetic modifications, either through histone methylation or histone deacetylation
- Co-IP assays have been initiated to detect HEYL's interaction with histone deacetylase (HDAC)
- If HEYL immunoprecipitates with HDAC, future RNA-Seq analysis will help identify the genes affected

Questions

- Will the phenotypic assays of transfected BGC stable cell line confirm that of transiently transfected BGC cells?
- What is the mechanism of HEYL in gastric cancer progression?

Conclusion

- *heyl* upregulates gastric cancer migration and invasion

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