Potential Role of *heyl* in Gastric Cancer Progression

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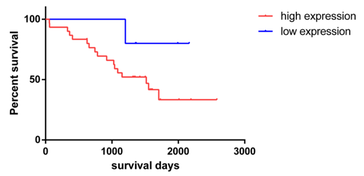
Introduction

• 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

Patient Survival Rate for Low and High Expressions of *heyl*

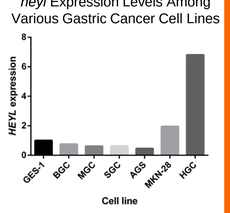


Objective of the Study

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

Methods

heyl Expression Levels Among Various Gastric Cancer Cell Lines



•Transfected BGC cells with constructed plasmids containing *myc-heyl*

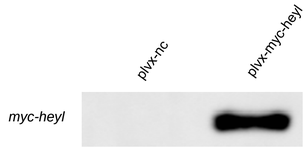
•Western Blot to confirm BGC cells' *heyl* overexpression

•Phenotypic assays to study the effects of *heyl* overexpression in BGC cells

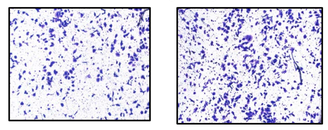
Results

Western Blot

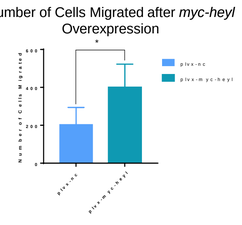
- Confirms BGC cells' ***heyl* overexpression** post-transfection



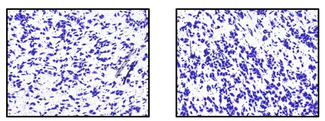
Crystal Violet Staining of Migrated Cells



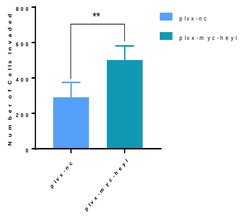
Number of Cells Migrated after *myc-heyl* Overexpression



Crystal Violet Staining of Invaded Cells



Number of Cells Invaded after *myc-heyl* Overexpression



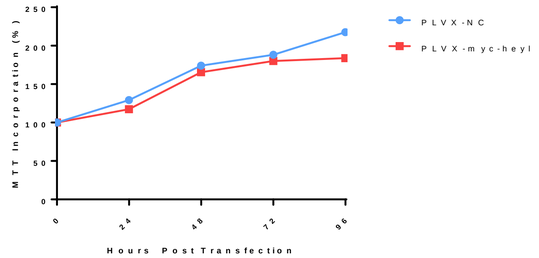
Transwell Migration and Invasion Assays

- 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

BGC-J PLVX-myc-heyl OE MTT Assay



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?

Acknowledgements

I would like to thank Professor Tianhua Zhou for welcoming me into his lab, Jiayu Zhang for being the most amazing mentor, and CHW for funding and providing me with this wonderful opportunity.

Conclusion

• *heyl* upregulates gastric cancer migration and invasion