LEPTIN RECEPTOR EXPRESSION IN BREAST TUMORS AND CHROMATIN MODULATORS: A COMPARATIVE STUDY

Sara Pacheco* B.S, Evelyn Juarez* B.S, Sushmita Nandy, Ph.D.
*Equal contribution authors
Department of Biology, Chemistry, and Environmental Science, Northern New Mexico College, NM

San Antonio Breast Cancer Symposium® - December 8-11, 2020

Background

- Metabolic dysregulation and carcinogenesis are strongly linked.
- Leptin signaling acts as a metabolic switch that maintains body weight and energy homeostasis and gets impacted during metabolic dysregulation.
- Leptin signaling mediates its effect on breast cancer cells through downstream effectors like JAK-STAT, MAPK and PI3K pathways.
- At the molecular level, leptin exerts its effects through its receptor, LEPR, encoded by the LEPR gene.
- Leptin signaling has been shown to contribute towards progression of breast cancer.
- Obese post-menopausal women are a high-risk category for breast cancer and dysregulated leptin signaling contributes towards it.
- In this study, we focused on comparative molecular analysis of chromatin modulators in Leptin high (LEPRhi) and low (LEPRlow) expressing breast tumors.

Methods

- METABRIC Breast Cancer Study
- LEPRhi vs LEPRlow breast tumors
- Clinical
- RNA-seq
- Chromatin modulators (CR)
- Interactome

Results

I. Samples overlap

II. Survival curve LEPRhi vs LEPRlow (p<0.005)

III. Patients overlap

Conclusion

- This study provides insight into the differential expression of chromatin modulators in LEPRhi and LEPRlow expressing breast tumors.
- Based on the results, we hypothesize that leptin receptor signaling in breast cancer mediates epigenetic modifications of key genes that impact promotion and progression of breast cancer through alterations in the expression of chromatin modulators.

References