



NORTHERN

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

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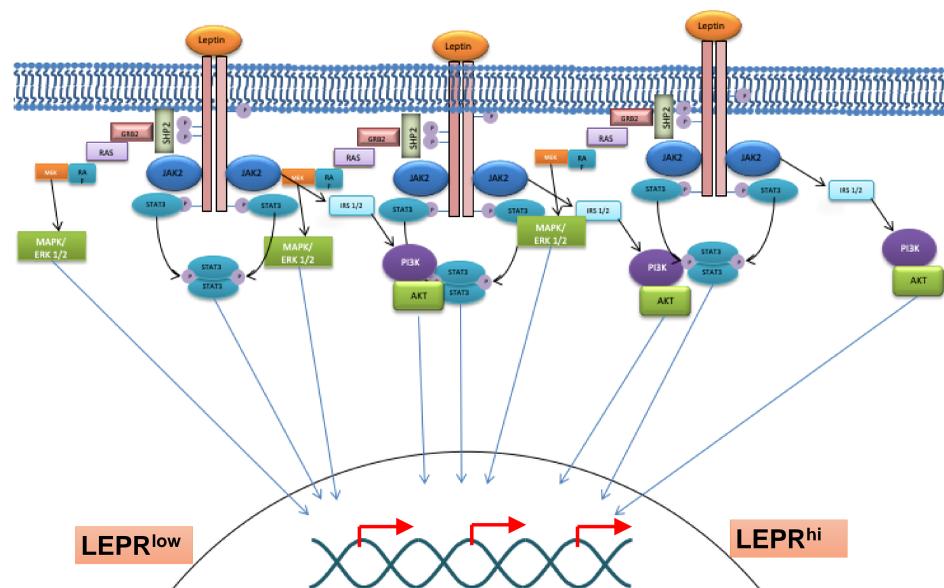
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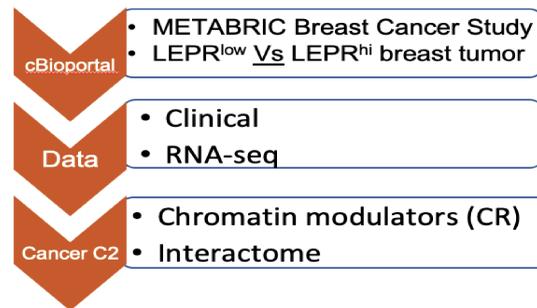
San Antonio Breast Cancer Symposium® - December 8-11, 2020

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 *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 ($LEPR^{hi}$) and low ($LEPR^{low}$) expressing breast tumors.



Methods



Results

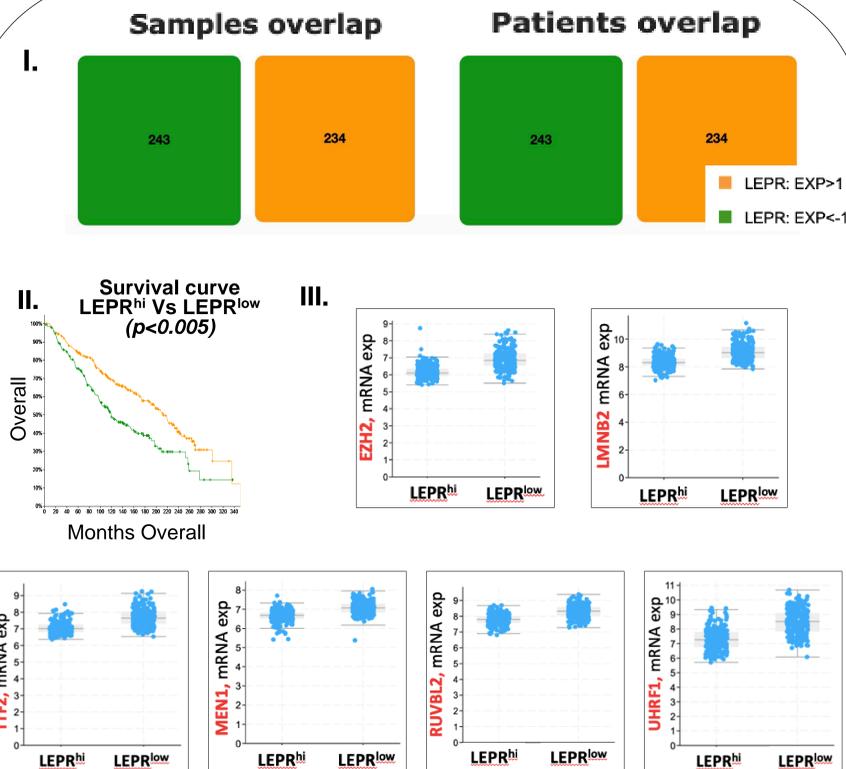


Figure 1) cBioportal-Breast cancer patient and tumor sample sizes from the METABRIC study expressing different levels of LEPR; II) Survival curve revealing a better prognosis for breast cancer tumors with higher expression as compared to low; III) cBioportal analysis of chromatin modulators-EZH2, LMNB2, TTF2, MEN1, RUVBL2 and UHRF1 in $LEPR^{hi}$ and $LEPR^{low}$ expressing breast tumor samples.

Results

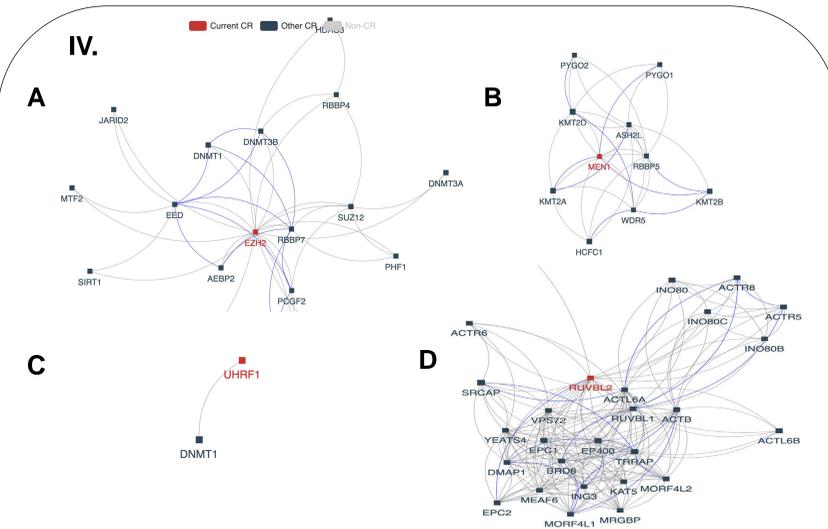


Figure IV) Cancer C2 analysis- Interactome demonstrating downstream molecules of chromatin modulators A) EZH2 B) MEN1 C) UHRF1 and D) RUVBL2.

Conclusion

- This study provides insight into the differential expression of chromatin modulators in $LEPR^{hi}$ and $LEPR^{low}$ 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

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