Justin Bromell

Biology Major Second Year ARCS Scholar Glenn Leadership Award



Obesity and Cancer

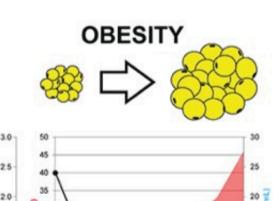
This work aims to understand how obesity promotes resistance to chemotherapeutics in prostate cancer.

Objectives

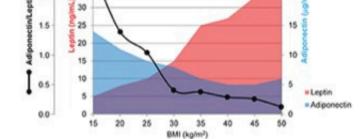
Objective 1: Determine the leptin signaling pathway responsible for leptin-induced chemoresistance.

Objective 2: Determine whether adiponectin and its mimetics can restore sensitivity to chemotherapeutics in leptin-protected prostate cancer.

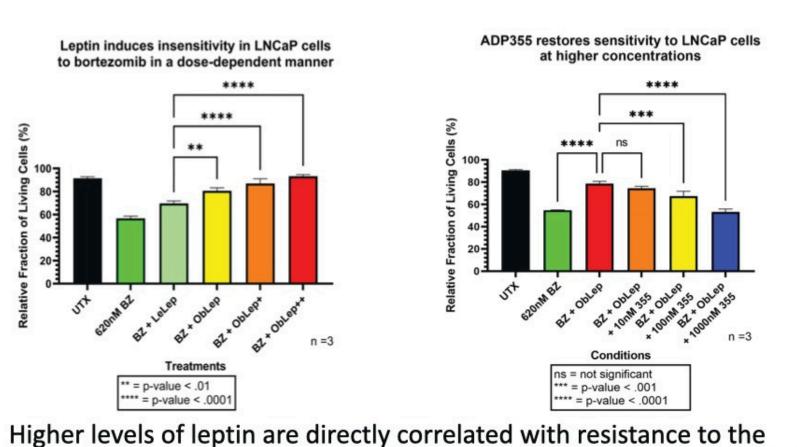
Obesity is Hormonally Regulated



• Fat cells, known as adipocytes, produce hormones called adipokines such as leptin or adiponectin

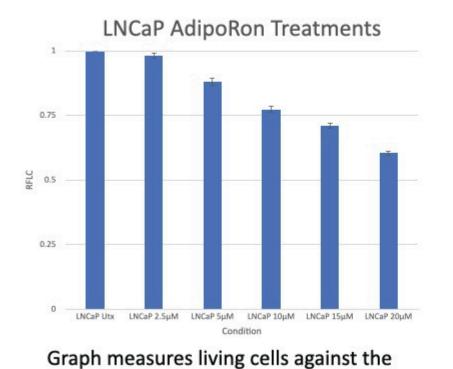


Obese people have higher • levels of circulating leptin in their bodies



chemotherapeutic bortezomib (BZ). Adding an adiponectin mimetic (ADP 355) seems to reverse this chemoresistance.

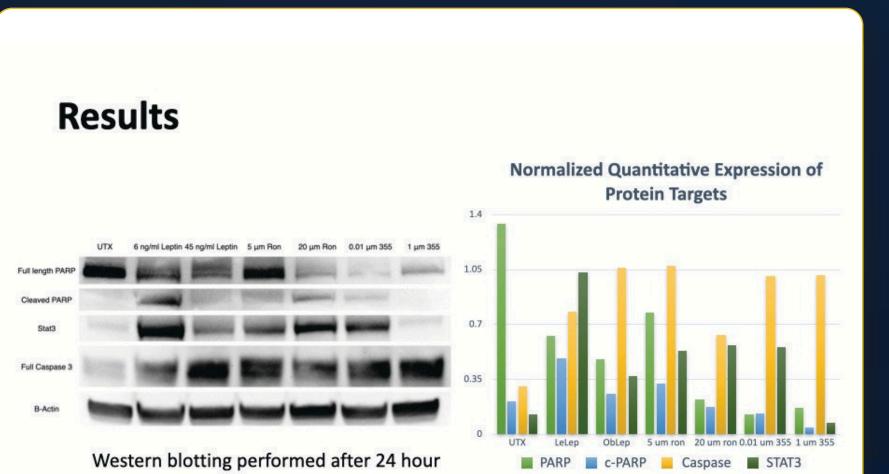
Adiponectin mimetics Cause Cancer Cell Death By Themselves



concentration of an adiponectin

previous assumption that adiponectin mimetics "restore" sensitivity to chemotherapeutics as adiponectin mimetics kill cancer by themselves

This data contradicts the



Conclusions

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- Leptin downregulates the STAT3 pathway

Future Directions

 Pharmacologically inhibit key parts of the leptin pathway and analyze cell death differences

treatment. B-actin was used as a loading control.

- The ADP 355 and Adiporon have different MOAs
- c-PARP/PARP ratios went up down as leptin concentration increased
- Leptin increases the expression of anti-apoptotic proteins

 Look for other targets in the leptin signaling pathways using western blotting

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Scholar-Awards Celebration

November 15, 2023



Igniting Innovation in Georgia