METABOLIC SYNDROME AND SALT-SENSITIVE HYPERTENSION IN POLYGENIC OBESE TALLYHO/JNGJ MICE: THE ROLE OF NA/K-ATPASE SIGNALING
THE SIGNIFICANCE OF INTERLEUKIN-6 AND NA/K-ATPASE SIGNALING IN OBESITY HYPERTENSION

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INTRODUCTION

• Obesity is recognized as a low-grade chronic inflammatory disease. Interleukin-6 contributes highly to inflammation and hypertension.

• The renal proximal tubule is responsible for about 67% of filtered Na and water absorption.

• The molecular mechanism through which sodium is transported is not fully understood.

• There have been prior advances within the lab at Marshall to understand this process.
HYPOTHESIS

- Obesity stimulates the IL-6 signal pathway and sodium pump signaling pathway.
- What is IL-6’s specific involvement?
EXPERIMENT

• The TallyHo and B6 mice were measured and observed based on the following:
  - Body Weight
  - Blood Pressure
  - Blood Sugar
• A high salt diet was administered to the mice when they reach the optimum age range.
• Measurements of IL-6 expression, Stat3 expression, and Sodium pump signaling were analyzed from the kidney tissue of B6 and TallyHo mice.
BODY WEIGHT

C57BL/6J TALLYHO

BW B6 and TH

Note: **, P<.01 vs. B6 N= 8
NA/K-ATPASE SIGNALING WAS IMPAIRED IN TALLYHO MICE IN RESPONSE TO HIGH SALT DIET
REACTIVE OXYGEN SPECIES IN NA/K-ATPASE SIGNALING
CONCLUSION

- Impaired Na/K-ATPase signaling contributes to the increased salt sensitivity of obese hypertension and IL-6 appears to be involved in this process.
FUTURE ADVANCES

• To further explore the relationship between Na/K-ATPase and IL-6 signaling pathway will potentially lead to a new and innovative approach to improved treatment strategies for hypertension. Thus, reducing obesity-related health disparities.
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