May 8th, 12:30 PM - 1:30 PM

Obesity-Induced Diabetes and Lower Urinary Tract Fibrosis Promote Urinary Voiding Dysfunction in a Mouse Model

Mehrnaz Gharaee–Kermani
University of Massachusetts Boston

Jose A. Rodriguez-Nieves
University of Massachusetts Boston

Jill A. Macoska
University of Massachusetts Boston

Follow this and additional works at: https://escholarship.umassmed.edu/cts_retreat

Part of the Cell Biology Commons, Endocrine System Diseases Commons, Male Urogenital Diseases Commons, and the Translational Medical Research Commons

This work is licensed under a Creative Commons Attribution-Noncommercial-Share Alike 3.0 License.

Gharaee–Kermani, Mehrnaz; Rodriguez-Nieves, Jose A.; and Macoska, Jill A., "Obesity-Induced Diabetes and Lower Urinary Tract Fibrosis Promote Urinary Voiding Dysfunction in a Mouse Model" (2013). UMass Center for Clinical and Translational Science Research Retreat. 52.
https://escholarship.umassmed.edu/cts_retreat/2013/posters/52

This material is brought to you by eScholarship@UMMS. It has been accepted for inclusion in UMass Center for Clinical and Translational Science Research Retreat by an authorized administrator of eScholarship@UMMS. For more information, please contact Lisa.Palmer@umassmed.edu.
Obesity-Induced Diabetes and Lower Urinary Tract Fibrosis Promote Urinary Voiding Dysfunction in a Mouse Model

Mehrnaz Gharae-Kermani, Jose A. Rodriguez-Nieves and Jill A. Macoska

Center for Personalized Cancer Therapy and Department of Biology, The University of Massachusetts, Boston,

**Background:** Progressive aging- and inflammation-associated fibrosis effectively remodels the extracellular matrix to increase prostate tissue stiffness and reduce urethral flexibility, resulting in urinary flow obstruction and Lower Urinary Tract Symptoms (LUTS). In the current study we sought to test whether senescence-accelerated mouse prone (SAMP)6 mice, which were reported to develop prostatic fibrosis, would also develop LUTS, and whether these symptoms would be exacerbated by diet-induced obesity and concurrent Type 2 Diabetes Mellitus (T2DM).

**Methods:** To accomplish this, SAMP6 and AKR/J background strain mice were fed regular mouse chow, low fat diet chow, or high fat diet chow for 8 months, then subjected to glucose tolerance tests, assessed for plasma insulin levels, evaluated for urinary voiding function, and assessed for lower urinary tract fibrosis.

**Results:** The results of these studies show that SAMP6 mice and AKR/J background strain mice develop diet-induced obesity and T2DM concurrent with urinary voiding dysfunction. Moreover, urinary voiding dysfunction was more severe in SAMP6 than AKR/J mice and was associated with pronounced prostatic and urethral tissue fibrosis.

**Conclusions:** Taken together, these studies suggest that obesity, T2DM, lower urinary tract fibrosis, and urinary voiding dysfunction are inextricably and biologically linked.

Supported by MICHR grant U034697 (MGK) and NIH/NIDDK grant 1P20DK090870-03 (JAM)