Apr 30th, 12:00 AM

Validation of a novel Trpm8 knockout mouse model

Bilan Mohamed
University of Southern Maine, bilan.mohamed@maine.edu

Title: Validation of *Trpm8* knockout mouse model by gene expression

Authors: Bilan Mohamed, Dr. Katherine Motyl,

Key Words: *Trpm8*, bone, osteoblast, osteoclast, *Cre,* loxP, mouse models

Recent studies suggest that the use of thermoregulatory treatment that impact brown fat may help curb obesity. However it is unknown, how these treatments may impact bone homeostasis. Our work has focused on the transient receptor potential melastatin (TRPM8) protein, which is responsible for detecting colder temperatures in sensory neurons. Previous work within the Motyl laboratory has found that *Trpm8* plays a role in bone acquisition. Mice with a global deletion of the *Trpm8* gene have reduced trabecular bone volume fraction due to reduced bone formation by osteoblasts. However, it is unclear whether sensory neuron or osteoblast-mediated expression of *Trpm8* is responsible for this finding. To test this, we generated mice with loxP sites surrounding the *Trpm8* gene. Using a constitutively expressed *Cre,* we generated a global knockout mouse model to confirm that the DNA in the new mouse model recombines as expected, and that the global deletion of *Trpm8* suppresses bone volume fraction and bone formation. Our aim is to confirm the *Trpm8* deletion by examining gene expression in the dorsal root ganglion. We will also examine the low bone mass phenotype using methods such as micro-computed tomography and dual energy x-ray absorptiometry. Future work will use the *Trpm8* sensory-neuron specific deletion to test the hypothesis that cold-temperature sensation supports bone homeostasis.