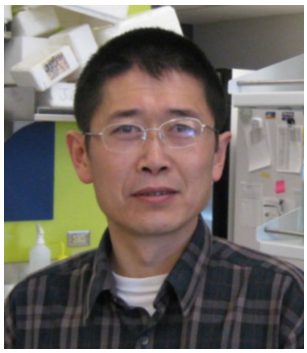
NCGG SEMINAR

Fat talks to Bone



Dr. Wei Zou

Assistant Professor

Department of Pathology and Immunology,
Washington University School of Medicine,
St. Louis, MO, USA

at 16:00~17:00 on 22 Nov, 2018 (Thu)

The 2nd floor Conference Hall Small,
1st Research building, NCGG

While obesity has been considered beneficial for skeletal health recent studies suggest the opposite. Because there is little mechanistic insight as to how fat, per se, regulates the skeleton, we generated "fat-free" (FF) mice completely lacking visible visceral, subcutaneous and brown fat. Due to robust osteoblastic activity, trabecular bone volume is enhanced 400-500% in these animals. As expected, FF mice are diabetic but normalization of glucose tolerance fails to alter their skeletal phenotype indicating their enhanced bone mass does not reflect the metabolic syndrome. Importantly, the skeletal phenotype of FF mice is completely rescued by transplantation of adipocyte precursors or various fat depots indicating adipocyte products regulate bone growth. Confirming such is the case, transplantation of fat derived from adiponectin and leptin double knockout mice, unlike that obtained from their WT counterparts, fails to normalize FF bone. Thus, fat suppresses bone formation and decreased adiposity may greatly enhance bone mass due to a paucity of adiponectin and leptin. These observations challenge the concept that obesity improves skeletal health and provides insight as to why lipodystrophic patients are osteosclerotic.

Contact: Sunao Takeshita, BJD (ext. 5047)