Genetics Day Poster Submission

**Presenter**: Christina Kaszuba

**Category**: Graduate Student

**Authors**: Benjamin J. Rodems, CHRISTINA KAZSUBA, Sonali Sharma, Emily R. Quarato, Takashi Ito, Roman Eliseev, Laura M. Calvi, and Jeevisha Bajaj

**Title:** THE ROLE OF SLC6A6 (TAUT) IN BONE FORMATION AND MAINTENANCE

**Abstract:** Osteoporosis is a severe and chronic bone disorder that is responsible for nearly two million bone fractures annually in the United States alone. This disease is characterized by weak, brittle bones that increase the risk of fracture. A taurine-enriched diet has been shown to increase bone mass and protection against bone loss. This indicates that taurine, a non-essential amino acid, could play a functional role in osteogenesis. The majority of taurine is transported into the cells via the high-affinity-taurine transporter (TauT), encoded by the SLC6A6 gene, which functions as a sodium and ion-dependent transporter. While much work has focused on the supplementation of taurine on bone health, little is known about the functional effect of TauT loss on osteogenic differentiation and bone formation. To determine the effects of TauT loss in vivo, dual-energy X-ray absorptiometry (DEXA) and micro-CT was carried out on both wild type (WT) and knock out (KO) mice. Our data suggests that the loss of TauT decreases bone mineral quality and thickness. Biomechanical torsion and compression testing of both cortical and trabecular bone suggests there is a strength defect associated with TauT loss. In vitro osteogenic colony forming unit (CFU) assays of digested bone from WT and KO mice indicate that KO MSCs have a decreased rate of proliferation and differentiation, indicating a defect in KO bone forming ability. Our experiments show that TauT loss does not impair adipogenic differentiation and total fat deposition in the bone marrow. Collectively, our results identify TauT as a key regulator of bone formation and maintenance, and indicate that it may play a role in osteopenia and/or osteoporosis.