In vitro differential gene expression in primary osteoblast cultures from osteogenesis imperfecta murine (*oim*) mice

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(OI) is a heritable connective tissue disorder most often characterized by

mutations in the genes which encode type I

include bone fragility and muscle weakness. There is no cure for OI, and current treatments including bisphosphonates and surgical rodding to stabilize long bones, most susceptible to fracture, have limited success. While there are multiple mouse models that mimic the pathology of this disorder, our lab

mutation in the *Col1a2* gene and model

Mesenchymal stromal cells (MSCs) can differentiate into osteoblasts through the up-regulation of fetal transcription factor *Runx2.* This allows the preosteoblast to enter a differentiation process in which the cells mature and become mineralized to produce bone, known as osteoblastogenesis. During this process, type I collagen is produced as it is a crucial component of bone, and it is expressed throughout the entire maturation of the osteoblast.

To determine if osteoblastogenesis is altered in *oim* mice and to evaluate differentiation and maturation of the cells, primary osteoblasts were collected from the calvaria of 7 day old wild-type (Wt) and homozygous *oim/oim* mice. These were digested and cultured in osteogenic media then differentiation media to promote differentiation of the preosteoblast to the mature osteoblast. Wt and *oim/oim* primary osteoblasts were plated and allowed to undergo differentiation in culture from pre- osteoblasts to mature osteoblasts. At days 1, 7, 14, and 21, a subset of the cells was harvested. RNA was isolated and then was performed to evaluate expression of specific osteoblast differentiation markers. Preliminary data suggest that both Wt and *oim/oim* preosteoblasts can differentiate in culture as evidenced by late upregulation of the key osteoblastogenic marker *Col1a1*.