Autophagy is a lysosomal proteostasis mechanism by which cells eliminate and recycle cytoplasmic content such as damaged or incorrectly folded proteins and organelles. Based on the delivery mechanisms utilized to transfer the targets to the lysosomes, autophagy is separated into three types: macroautophagy, chaperone-mediated autophagy (CMA), and microautophagy. We have previously shown that macroautophagy in osteoblast lineage is essential for skeletal health. However, whether other types of autophagy are relevant in bone, or what physiological/pathophysiological role they play in bone cells is unknown. In the work we presented in ASBMR 2020, we started addressing the role of CMA for skeletal remodeling. In these studies, we produced a murine model that lacks CMA in all tissues. By using this model, we showed that young adult mice lacking CMA have a low bone mass phenotype associated with increased production of bone resorbing cells. We also showed that calvarial bone forming cells from CMA-deficient mice did not have a drastic deficit in proteostasis likely due to compensatory upregulation of other proteostasis mechanisms. Our future studies will address the role of CMA for bone cells in response to cellular stressors such as aging.