SSBH 2021 Curriculum Vitae	
Name	**Amy Yoshiko Sato**
Organization	**University of Arkansas for Medical Sciences (UAMS)**
Position & Title	**Postdoctoral Research Fellow, Department of Physiology and Cell Biology, Ph.D.**

Educational background & Professional experience

2017	Department of Anatomy and Cell Biology, Indiana University School of Medicine, Ph.D.
2011	Biology; Psychology, DePauw University, B.A.

Research Interests

A. General Research Interests:

My research interests focus on skeletal biology, with a particular emphasis on the mechanisms of actions of glucocorticoids (GC) upon the musculoskeletal system. My graduate studies focused on investigating novel mechanisms that potentially interfere with glucocorticoid (GC)-induced bone loss, under the guidance of Dr. Teresita Bellido at the University of Indiana School of Medicine. These studies examined the effects of interventions targeting endoplasmic reticulum stress, the Wnt antagonist Sost/sclerostin, and Pyk2 activity on the response of bone cells to GC, focusing on bone formation, osteoblast/osteocyte apoptosis, and bone resorption activity. In the last part of my graduate studies, I initiated investigations on the mechanisms by which GC excess induces skeletal muscle loss and weakness. My postdoctoral studies expand upon these initial observations and investigate the hypothesis that atrogene upregulation is a common mechanism underlying GC actions in bone and skeletal muscle, and that therapeutic approaches that interfere with the atrophy pathway will simultaneously prevent harmful GC actions in both tissues. We are also investigating whether prevention of Pyk2 activation and Notch signaling induced by GC, as well as whether increased activation of the vitamin D receptor will prevent GC-induced musculoskeletal disease. During my post-doctoral fellowship, I also have had the opportunity to examine the role of osteocytic focal adhesion kinase FAK expression in the anabolic skeletal response to mechanical loading *in vivo,* as well as the role of anti-oxidant enriched blueberry diets and antioxidant transcription factor Nrf2 expression upon the skeletal response to sex steroid deficiency. I have also participated in studies investigating the potential of interventions such as the SERM Raloxifene, activation of the PTH receptor with PTHrP peptides, and mechanical

loading to ameliorate the bone disease elicited by type I or type II diabetes in rodent models.

B. Glucocorticoid, Muscle, and Bone focused Postdoctoral Career: Mechanisms of glucocorticoid action on the musculoskeletal system: Excess of glucocorticoids (GC) increases bone resorption and decreases bone formation, resulting in loss of bone mass and strength. GC also cause skeletal muscle weakness with loss of body balance and increased falls, which in turn further elevated bone fracture risks. Currently, there is an unmet need for a therapeutic agent that simultaneously addresses osteoporosis and sarcopenia, and improved therapeutic strategies are sorely needed. We are investigating the atrophy E3 ubiquitin ligase signaling pathway as well as whether interventions that prevent bone loss impact GC skeletal muscle effects. These findings will offer new molecular targets for interventions to potentially prevent GC-induced bone and muscle loss.

C. Nutrition, Microbiome, and Bone focused Postdoctoral Career: Blueberry diet induced bone to gut microbiome crosstalk: Accumulating evidence highlights the importance of the gut microbiome for homeostasis in several tissues, including the skeleton. In particular, the composition of intestinal microbiota is linked to bone remodeling and bone loss with estrogen deficiency in mouse models; although the mechanisms by which the gut microbiome influence bone homeostasis are far from being fully understood. These studies investigate the underlying protective mechanism of blueberry-based nutritional interventions that protects the musculoskeletal system from estrogen loss and promotes gut microbiome diversity. These findings lay the foundation for developing nutrition-based interventions with fewer side effects and tailored to patient sex.

Publications

Publications: https://www.ncbi.nlm.nih.gov/myncbi/1p7esBPytjqk9/bibliography/public/

Total number of publications: 12 (6 as first author, 10 original articles, and 2 reviews)

1. **Sato AY,** Pellegrini GG, Cregor M, McAndrews K, Choi RB, Maiz M, Johnson O, McCabe LD, McCabe GP, Ferruzzi MG, Lila MA, Peacock M, Burr DB, Nakatsu CH, Weaver CM, Bellido T. 2020. Skeletal protection and promotion of microbiome diversity by dietary boosting of the endogenous antioxidant response. *JBMR. DOI: 10.1002/jbmr.4231.*
2. **Sato AY**, Cregor M, McAndrews K, Li T, Condon KW, Plotkin LI, Bellido T. 2019. Glucocorticoid-induced bone fragility is prevented in female mice by blocking Pyk2/anoikis signaling. *Endocrinology, 160*(1659-1673): doi: 10.1210/en.2019-00237. Epub 2019 May 13.
3. Delgado-Calle J, Hancock B, Likine EF, **Sato AY**, McAndrews K, Sanudo C, Bruzzaniti A, Riancho JA, Tonra JR, Bellido T. 2018. MMP14 is a novel target of PTH signaling in osteocytes that controls resorption by regulating soluble RANKL production. *FASEB J. 32*(2878-2890): doi: 10.1096/fj.201700919RRR. Epub 2018 Jan 17.
4. **Sato AY**, Peacock M, Bellido T. 2018. Glucocorticoid excess in bone and muscle. *Clin Rev Bone Miner Metab 16*(33-47): doi: 10.1007/s12018-018-9242-3. Epub 2018 Feb 5.

5. **Sato AY**, Richardson D, Cregor M, Davis HM, Au ED, McAndrews K, Zimmers TA, Organ JM, Peacock M, Plotkin LI, Bellido T. 2017. Glucocorticoids induce bone and muscle atrophy by tissue-specific mechanisms upstream of E3 ubiquitin ligases. *Endocrinology 158*(664-677): doi: 10.1210/en.2016-1779. Epub 2017 Jan 9.
6. **Sato AY**, Cregor M, Delgado-Calle J, Condon KW, Allen MR, Peacock M, Plotkin LI, Bellido T. 2016. Protection from glucocorticoid-induced osteoporosis by anti-catabolic signaling in the absence of Sost/sclerostin. *JBMR 31*(1791-1802): doi: 10.1002/jbmr.2869. Epub 2016 Jun 5.
7. **Sato AY**, Tu X, McAndrews KA, Plotkin LI, Bellido T. 2015. Prevention of glucocorticoid induced-apoptosis of osteoblasts and osteocytes by protecting against endoplasmic reticulum (ER) stress in vitro and in vivo in female mice. *Bone* 73(60-68): doi: 10.1016/j.bone.2014.12.012. Epub 2014 Dec 19.