SSBH 2021 Curriculum Vitae	
Name	**Haejin Yoon**
Organization	**Harvard Medical School**
Position & Title	**Postdoctoral Fellow**
Educational background & Professional experience	
2011-2014	Department of Biomedical Sciences, College of Medicine, Seoul National University, Ph.D. Dissertation: Inhibitory roles of the lysyl-acetyltransferase ARD1 in bone development and regeneration: ARD1 controls osteoblast differentiation by inhibiting Runx2 in a feedback manner (Advisor: Professor Jong-Wan Park)
2009-2011	Department of Biomedical Sciences, College of Medicine, Seoul National University, M.S. Department of Biomedical Sciences, College of Medicine, Seoul National University Dissertation: Study on the regulation mechanism of HIF-1α N-terminal TAD (Advisor: Professor Jong-Wan Park)
2004-2009	Department of Biology, Kon-Kuk University, B.S. Advisor: Professor Kyungho Lee
2015-present	Harvard Medical School, Postdoctoral Fellow Research: Contributions of mitochondria to cell metabolism (Cell biology)
2014-2015	Ischemic/Hypoxic Disease Institute, Researcher Research: The role of histone modification and epigenetic modulation in hypoxia on ovarian cancer therapy (Cell biology, Epigenetics)
2009-2014	College of Medicine, Seoul National University, Ph.D. and Master Research Assistant Research: Roles of the post-translational modifications (PTMs) in human physiology and disease & Molecular mechanism of metabolic enzymes in hypoxia (Cell biology, Molecular biology, Bone physiology)
Research Interests	

1) The effects of metabolic enzymes PHD3 on energy homeostasis.

Dynamic control of cellular metabolism is critical for maintaining tissue homeostasis. In response to changing cellular energy levels, acetyl-CoA carboxylase 2 (ACC2) is post-translationally modified with phosphorylation or hydroxylation, leading to ACC2 enzymatic inhibition or activation. However, the biological consequences of ACC2 modulation are not fully understood, especially in the context of changing cellular

bioenergetics. I investigate the mechanism through which hydroxylation and phosphorylation of ACC2 coordinate fatty acid oxidation (FAO) *in vivo* (Yoon et al., 2020). Moreover, I utilized physiological conditions of AMP-activated protein kinase (AMPK) activation such as fasting and exercise challenge to demonstrate that ACC2 phosphorylation precludes PHD3 binding to ACC2 *in vivo*. These studies demonstrate that phosphorylation of ACC2 is sufficient to repress PHD3-mediated hydroxylation, and that this relationship is fundamental to the regulation of cellular lipid metabolism in normal physiology.

2) The contributions of mitochondria to cell metabolism.

While much research has examined the use of glucose and glutamine by tumor cells, many cancers instead prefer to metabolize fats. Despite the pervasiveness of this phenotype, knowledge of pathways that drive mitochondrial fat metabolism in cancer is limited. We reveal that PHD3 rapidly triggers the repression of fat catabolism in response to nutrient abundance (Yoon et al., 2020; German et al., 2016). Thus, PHD3 enables greater utilization of fatty acids but may also serve as a metabolic and therapeutic liability by indicating cancer cell susceptibility. In addition, I worked to develop the metabolic recycling of ammonia (Spinelli et al., 2017), and discover a glutamine metabolism pathway on mitochondria in cancer (Gonzalez et al., 2018). These studies will offer insight into a potential new node of mitochondrial metabolism that may be targeted pharmacologically in metabolic disease.

3) The roles of the post-translational modifications (PTMs) in human physiology.

After biosynthesis, every protein has different PTMs for modulating its enzymatic activity and stability. I developed the role of acetylation on major transcription factors in bone development (Yoon et al., 2014). This was an important contribution in bone biology that acetylation on transcription factors could be considered a potential strategy for facilitating bone formation. After, I worked on defining the role of methylation on protein in bone development (Kim et al., 2014). Still, PTMs regulation on transcription factor is very novel in bone biology, these studies suggest that different PTMs were novel biomarkers and clinical targets in bone metabolic disease.

4) The molecular mechanism of metabolic enzymes in hypoxia.

HIF-1α plays a central role in cellular adaptation to hypoxia, and is closely related to the pathogeneses of life-threatening disorders. I worked on the transcriptional activity of HIF-1α regulated by several metabolic enzymes, which determined cancer cell growth and tumor formation under hypoxia (Yoon et al., 2011; Yoon et al., 2014). These studies contributed a better understanding of the roles of metabolic enzymes in hypoxic responses and a basic concept for developing anticancer strategy. In addition, I investigated the new molecular mechanism of oxygen-dependent gene regulation and cancer metastasis in response to hypoxia (Kang, Shin and Yoon et al., 2018). These studies that reveal the molecular mechanism of HIF-1α modulation have direct impacts on cancer therapy.

Publications

1) Yoon H, Spinelli JB, Zaganjor E, Wong SJ, German NJ, Randall EC, Dean A, Clermont

A, Paulo JA, Garcia D, Li H, Rombold O, Agar NYR, Goodyear LJ, Shaw RJ, Gygi SP, Auwerx J, Haigis MC. PHD3 loss promotes exercise capacity and fat oxidation in skeletal muscle. Cell Metab. 2020. 32(2):215-228.e7. PMID: 32663458

2) Kang J*, Shin SH*, Yoon H*, Huh J, Shin HW, Chun YS, Park JW. FIH is an oxygen sensor in ovarian cancer for G9a/GLP-driven epigenetic regulation of metastasis-related genes. Cancer Res. 2018. 78(5):1184-1199. PMID: 29259012 [* co-first author]

3) Spinelli JB, Yoon H, Ringel AE, Jean-Favre S, Clish CB, Haigis MC. Metabolic recycling of ammonia via glutamate dehydrogenase supports breast cancer biomass. Science. 2017. 358(6365):941-946. PMID: 29025995

4) German NJ, Yoon H, Yusuf RZ, Murphy JP, Finley LW, Laurent G, Haas W, Satterstrom FK, Guarnerio J, Zaganjor E, Santos D, Pandolfi PP, Beck AH, Gygi SP, Scadden DT, Kaelin WG Jr, Haigis MC. PHD3 loss enables reliance on fat oxidation via deactivation of acetyl-CoA aarboxylase. 2016. Mol Cell. 63:1006-1020. PMID: 27635760

5) Yoon H, Kim HL, Chun YS, Shin DH, Lee KH, Shin CS, Lee DY, Kim HH, Lee ZH, Ryoo HM, Lee MN, Taeg Oh G, Park JW. NAA10 controls osteoblast differentiation and bone formation as a feedback regulator of Runx2. Nat Commun. 2014. 5:5176. PMID: 25376646

6) Yoon H, Shin SH, Shin DH, Chun YS, Park JW. Differential roles of Sirt1 in HIF-1α and HIF-2α mediated hypoxic responses. Biochem Biophys Res Commun. 2014. 444(1):36-43. PMID: 24423936

7) Yoon H, Lim JH, Cho CH, Huang LE, Park JW. CITED2 controls the hypoxic signaling by snatching p300 from the two distinct activation domains of HIF-1α. Biochim Biophys Acta. 2011. 1813(12):2008-16. PMID: 21925214

A full list of publications can be found:
http://www.ncbi.nlm.nih.gov/pubmed/?term=Haejin+Yoon