**Postdoctoral Position at the Columbia University**

The Lab of Dr. Choi in the Department of Pathology and Cell Biology at the Columbia University Medical Center is currently seeking a talented postdoctoral research scientist with interest in molecular-cellular mechanisms related to insulin signaling, trafficking and genome integrity. The candidate will employ a variety of techniques ranging from CRISPR screening, conventional biochemistry, cell biology and mouse genetics to achieve their project goals. The postdoctoral scientist will also be given opportunities to collaborate with experts in cryo-EM to understand the molecular mechanism of receptor tyrosine kinase activation.

Her research goal is to delineate mechanisms underlying the mutual regulation between cell division and metabolism by combining mouse genetics, cell biology, biochemistry and cryo-EM structure. Perturbation of this regulation leads to cancer and metabolic diseases. Dr. Choi has discovered a critical role of cell division regulators in insulin signaling through regulating insulin receptor endocytosis. These findings link aneuploidy-suppressing genes to insulin signaling and suggest a mechanism by which a circulating hormone may regulate genomic stability. Her laboratory will study the role of cell division regulators in insulin signaling and will expand it to other receptor tyrosine kinases to discover how systemic signaling communicates with cell division process to maintain both genomic stability and metabolic homeostasis.

Recently, collaboration with Xiaochen Bai in UTSW, she discovered that large-scale conformational change of insulin receptor driven by insulin binding relieves its auto-inhibition, triggering trans-autophosphorylation of the kinase domain and hence initiation of downstream signaling cascade. How this conformational change induces kinase activation, how the activated kinase selectively provokes the signaling branch, and how the active insulin receptor can be preferentially internalized are not fully understood. Her laboratory will reveal how insulin activates the receptor kinase and insulin signaling, and initiates the receptor endocytosis at the molecular level, and how this process maintains systemic homeostasis in vivo. Along the way, they hope to unravel new molecular targets that will be useful to treat two very prevalent diseases, Diabetes and Cancer.

For this position, general knowledge of molecular biology and cell biology is required. Experience in metabolic studies using mouse is ideal but not required.

Please send CV and the names of three academic references via email to: Eunhee Choi, PhD (EC3477@cumc.columbia.edu).

**Selected Publications**

Li J#., **Choi E.** #**\***., Yu H.**\***, Bai XC.**\*** **(2019)** Structural basis of the activation of type 1 insulin-like growth factor receptor. ***Nat. Commun.*** **10, 4567** (#Co-first author, **\***Co-corresponding author)

Uchikawa E.#, **Choi E.#\***, Shang G., Yu H.**\***, Bai XC.**\*** **(2019)** Activation mechanism of the insulin receptor revealed by cryo-EM structure of the fully liganded receptor–ligand complex. ***eLife 8, e48630.*** (#Co-first author, **\***Co-corresponding author)

**Choi E.**, Kikuchi S., Gao H., Brodzik K., Nassour I., Yopp A., Singal A., Zhu H., and Yu H. **(2019)**Mitotic regulators and the SHP2-MAPK pathway promote insulin receptor endocytosis and feedback regulation of insulin signaling. ***Nat. Commun.* 10, 1473**.

**Choi E.** and Yu H. **(2018)** Spindle checkpoint regulators in insulin signaling. ***Front. Cell Dev. Biol.*** 6:161.

Kim J., Hu Z., Cai L., Li K., **Choi E.**, Faubert B., Bezwada D., Rodriguez-Canales J., Villalobos P., Lin YF., Ni M., Huffman K., Girard L., Byers L., Kacmaz K., Pna C., Heymach J., Wauters E., Vansteenkiste J., Castrillon D., Chen B., Wistuba I., Lambrechts D., Xu J., Minna J., and DeBerardinis R. **(2017)** CPS1 maintains pyrimidine pools and DNA synthesis in KRAS/LKB1-mutant lung cancer cells. ***Nature* 546 (7656): 168-72.**

**Choi E.**, Zhang X., Xing C., and Yu H. **(2016)** Mitotic checkpoint regulators control insulin signaling and metabolic homeostasis. ***Cell* 166 (3): 567-81.**

**Choi E.** and Yu H. **(2015)**. Phosphorylation propels p31comet for mitotic exit. ***Cell Cycle* 14 (13) 1997-8.**

Park I., Lee HO., **Choi E.**, Lee Y-K., Kwon M-S., Min J., Park P-G., Lee S., Kong Y-Y., Gong G., and Lee H. **(2013)**. Loss of BubR1 acetylation causes defects in spindle assembly checkpoint signaling and promotes tumor formation. ***J. Cell Biol*. 202 (2): 295-309.**

**Choi E#**., Park P-G#., Lee HO#., Lee Y-K., Kang GH., Lee JW., Han W., Lee HC., Noh D-Y., Lekomtsev S., Gong GY., and Lee H. **(2012)**. BRCA2 fine-tunes the spindle assembly checkpoint through reinforcement of BubR1 acetylation. ***Dev. Cell* 22:295-308.** (#Co-first author)

**Choi E**., Choe H., Min J., Choi J-Y., Kim J., and Lee H. **(2009)**. BubR1 acetylation at prometaphase is required for modulating APC/C activity and timing in mitosis. ***EMBO J*. 28: 2077-2089.**

Lee Y., **Choi E**., Park P-G., Kim M. A., Park N-H, and Lee H. **(2009)**. BubR1 as a prognostic marker for recurrence-free survival rates in epithelial ovarian cancers. ***Brit. J. Cancer* 101: 504-510.**

**Choi E**. and Lee H. **(2008)**. Chromosome damage induces BubR1 activation and prometaphase arrest. ***FEBS Letters* 582:1700-1706.**

For a complete list of publications, please visit [PubMed.gov](https://www.ncbi.nlm.nih.gov/myncbi/1R7c7cLgmxhAy/bibliography/public/).