Ji-Bin Peng, PhD

Associate Professor
Department of Medicine, Division of Nephrology
ZRB 634 Zip 0007
Phone: 205-975-6130

Dr. Peng received his Ph.D. degree from Shanghai Institute of Cell Biology of the Chinese Academy of Sciences. Dr. Peng had his postdoctoral training at Brigham & Women's Hospital and Harvard Medical School during 1997 to 2000 (Mentored by Drs. Matthias Hediger and Edward Brown). He became an Instructor in Medicine at Harvard Medical School in 2000. Dr. Peng Joined the University of Alabama at Birmingham (UAB) as an Assistant Professor in 2003. He is currently an Associate Professor of Medicine at UAB.

Research/Clinical Interest

Calcium transport proteins and their roles in health and disease
Research in Dr. Peng's laboratory is centered on the mechanism and regulation of calcium transport pathways. Calcium is a major inorganic component of the skeleton and serves as a key extracellular and intracellular messenger. Intestinal absorption and renal reabsorption of calcium play crucial roles in maintaining calcium balance of the body. Defects in calcium transport may result in disorders such as kidney stone disease and osteoporosis. Towards a better understanding of calcium transport and its relation to hypercalciuria and hypertension, Dr. Peng's laboratory is investigating molecular mechanisms of calcium absorption and reabsorption. Two calcium channels, TRPV5 and TRPV6, are the focus of current investigation. Dr. Peng's group is also working on the mechanisms of WNK4 kinase activation by calcium ions. WNK4 is a protein kinase linked to pseudohypoaldosteronism type II, a genetic form of hypertension. It is expected that those studies will lead to novel therapeutic strategies for hypercalciuria and hypertension.

Selected Publications

1. Peng JB, Yan WM and Bao XZ. Plasmid and transposon transfer to Thiobacillus ferrooxidans. J. Bacteriol. 176: 2892-2897, 1994. 8188590 [1]
2. Zhang Y, Zhang Y, Bone RN, Cui W, Peng JB, Gene P. Siegal GP, Wang H, Wu H. Regeneration of Pancreatic non-β Endocrine Cells in Adult Mice Following a Single Diabetes-Inducing Dose of Streptozotocin. PLoS ONE, 7(5):e36675.2012. 22586489 [2]
3. Wu G, Zhang W, Na T, Jing H, Wu H, Peng JB. Suppression of intestinal calcium entry channel TRPV6 by OCRL, a lipid phosphatase associated with Lowe syndrome and Dent disease. Am J Physiol Cell Physiol. 302:C1479-91, 2012. 22378746 [3]

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8. Lieben L, Benn BS, Ajibade D, Stockmans I, Moermans K, Hediger MA, Peng JB, Christakos S, Bouillon R, Carmeliet G. Trpv6 mediates intestinal calcium absorption during calcium restriction and contributes to bone homeostasis. Bone. 47(2):301-8. 2010. 20399919 [8]
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12. Zhang W, Na T, Peng JB. WNK3 positively regulates epithelial calcium channels TRPV5 and TRPV6 via a kinase-dependent pathway. Am J Physiol Renal Physiol. 295:F1472-84. 2008. 18768590 [12]
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14. Suzuki Y, Kovacs CS, Takanaga H, Peng JB, Landowski CP, and Hediger MA. Calcium channel TRPV6 is involved in murine maternal-fetal calcium transport. J Bone Miner Res. 23(8):1249-56, 2008. 18348695 [14]
15. Benn BS, Ajibade D, Porta A, Dhawan P, Hediger M, Peng JB, Jiang Y, Oh GT, Jeung EB, Lieben L, Bouillon R, Carmeliet G, Christakos S: Active intestinal calcium transport in the absence of transient receptor potential vanilloid type 6 and calbindin-D9k. Endocrinology, 149:3196-205, 2008. 18325990 [15]
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