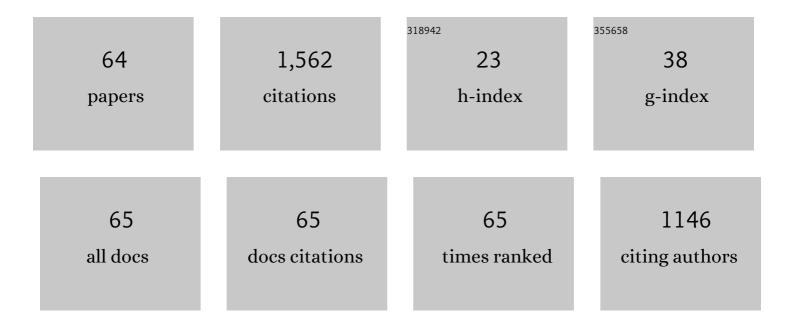
Dao-Hong Lin

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Activation of Kir4.1/Kir5.1 of DCT is essential for acute calcineurinâ€inhibitionâ€induced stimulation of NCC. FASEB Journal, 2022, 36, .	0.2	0
2	Role of inwardly rectifying K+ channel 5.1 (Kir5.1) in the regulation of renal membrane transport. Current Opinion in Nephrology and Hypertension, 2022, 31, 479-485.	1.0	6
3	Inwardly rectifying K ⁺ channels 4.1 and 5.1 (Kir4.1/Kir5.1) in the renal distal nephron. American Journal of Physiology - Cell Physiology, 2022, 323, C277-C288.	2.1	11
4	Deletion of renal Nedd4-2 abolishes the effect of high sodium intake (HS) on Kir4.1, ENaC, and NCC and causes hypokalemia during high HS. American Journal of Physiology - Renal Physiology, 2021, 320, F883-F896.	1.3	13
5	Deletion of Kir5.1 abolishes the effect of high Na ⁺ intake on Kir4.1 and Na ⁺ -Cl ^{â^`} cotransporter. American Journal of Physiology - Renal Physiology, 2021, 320, F1045-F1058.	1.3	11
6	Deletion of renal Nedd4-2 abolishes the effect of high K ⁺ intake on Kir4.1/Kir5.1 and NCC activity in the distal convoluted tubule. American Journal of Physiology - Renal Physiology, 2021, 321, F1-F11.	1.3	9
7	ROMK channels are inhibited in the aldosterone-sensitive distal nephron (ASDN) of renal-tubule Nedd4-2 deficient mice American Journal of Physiology - Renal Physiology, 2021, , .	1.3	5
8	Inhibition of AT2R and Bradykinin Type II Receptor (BK2R) Compromises High K ⁺ Intake-Induced Renal K ⁺ Excretion. Hypertension, 2020, 75, 439-448.	1.3	2
9	Effect of Angiotensin II on ENaC in the Distal Convoluted Tubule and in the Cortical Collecting Duct of Mineralocorticoid Receptor Deficient Mice. Journal of the American Heart Association, 2020, 9, e014996.	1.6	32
10	Renal Tubule Nedd4-2 Deficiency Stimulates Kir4.1/Kir5.1 and Thiazide-Sensitive NaCl Cotransporter in Distal Convoluted Tubule. Journal of the American Society of Nephrology: JASN, 2020, 31, 1226-1242.	3.0	18
11	Mg ²⁺ restriction downregulates NCC through NEDD4-2 and prevents its activation by hypokalemia. American Journal of Physiology - Renal Physiology, 2019, 317, F825-F838.	1.3	15
12	Deletion of Kir5.1 Impairs Renal Ability to Excrete Potassium during Increased Dietary Potassium Intake. Journal of the American Society of Nephrology: JASN, 2019, 30, 1425-1438.	3.0	40
13	Kir4.1/Kir5.1 Activity Is Essential for Dietary Sodium Intake–Induced Modulation of Na-Cl Cotransporter. Journal of the American Society of Nephrology: JASN, 2019, 30, 216-227.	3.0	30
14	Norepinephrine-Induced Stimulation of Kir4.1/Kir5.1 Is Required for the Activation of NaCl Transporter in Distal Convoluted Tubule. Hypertension, 2019, 73, 112-120.	1.3	22
15	Inhibition of AT2R and BK2R attenuates the effect of high K intake (HK) on thiazideâ€sensitive Na l cotransporter. FASEB Journal, 2019, 33, 824.23.	0.2	0
16	A case study of ciliary detachment with primary pulmonary hypertension. International Ophthalmology, 2018, 38, 375-379.	0.6	5
17	AT2R (Angiotensin II Type 2 Receptor)-Mediated Regulation of NCC (Na-Cl Cotransporter) and Renal K Excretion Depends on the K Channel, Kir4.1. Hypertension, 2018, 71, 622-630.	1.3	17
18	Potassium intake modulates the thiazide-sensitive sodium-chloride cotransporter (NCC) activity via the Kir4.1 potassium channel. Kidney International, 2018, 93, 893-902.	2.6	106

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19	Kir5.1 regulates Nedd4-2-mediated ubiquitination of Kir4.1 in distal nephron. American Journal of Physiology - Renal Physiology, 2018, 315, F986-F996.	1.3	27
20	Bradykinin Stimulates Renal Na ⁺ and K ⁺ Excretion by Inhibiting the K ⁺ Channel (Kir4.1) in the Distal Convoluted Tubule. Hypertension, 2018, 72, 361-369.	1.3	25
21	Kir4.1 activity is essential for dietary Na ⁺ intake induced modulation of Na l cotransporter (NCC). FASEB Journal, 2018, 32, 620.6.	0.2	0
22	Potassium Sensing by Renal Distal Tubules Requires Kir4.1. Journal of the American Society of Nephrology: JASN, 2017, 28, 1814-1825.	3.0	133
23	ENaC and ROMK activity are inhibited in the DCT2/CNT of TgWnk4 ^{PHAII} mice. American Journal of Physiology - Renal Physiology, 2017, 312, F682-F688.	1.3	20
24	PGF _{2α} regulates the basolateral K channels in the distal convoluted tubule. American Journal of Physiology - Renal Physiology, 2017, 313, F254-F261.	1.3	6
25	Epoxyeicosatrienoic Acids Regulate Adipocyte Differentiation of Mouse 3T3 Cells, Via PGC-1α Activation, Which Is Required for HO-1 Expression and Increased Mitochondrial Function. Stem Cells and Development, 2016, 25, 1084-1094.	1.1	67
26	Disruption of KCNJ10 (Kir4.1) stimulates the expression of ENaC in the collecting duct. American Journal of Physiology - Renal Physiology, 2016, 310, F985-F993.	1.3	35
27	Caveolin-1 regulates corneal wound healing by modulating Kir4.1 activity. American Journal of Physiology - Cell Physiology, 2016, 310, C993-C1000.	2.1	7
28	KCNJ10 (Kir4.1) is expressed in the basolateral membrane of the cortical thick ascending limb. American Journal of Physiology - Renal Physiology, 2015, 308, F1288-F1296.	1.3	47
29	Src-family protein tyrosine kinase phosphorylates WNK4 and modulates its inhibitory effect on KCNJ1 (ROMK). Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, 4495-4500.	3.3	20
30	Caveolin-1 Deficiency Inhibits the Basolateral K+ Channels in the Distal Convoluted Tubule and Impairs Renal K+ and Mg2+ Transport. Journal of the American Society of Nephrology: JASN, 2015, 26, 2678-2690.	3.0	24
31	The Disruption of KCNJ10 (Kir4.1) Depolarizes the Membrane Potential of the CNT and Stimulates the Expression of ENaC in the Medullary Collecting Duct. FASEB Journal, 2015, 29, 844.7.	0.2	0
32	KCNJ10 determines the expression of the apical Na-Cl cotransporter (NCC) in the early distal convoluted tubule (DCT1). Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 11864-11869.	3.3	136
33	Cyp2c44 epoxygenase in the collecting duct is essential for the high K+ intake-induced antihypertensive effect. American Journal of Physiology - Renal Physiology, 2014, 307, F453-F460.	1.3	19
34	Kcnj10 is a major type of K+ channel in mouse corneal epithelial cells and plays a role in initiating EGFR signaling. American Journal of Physiology - Cell Physiology, 2014, 307, C710-C717.	2.1	7
35	MicroRNA-194 (miR-194) regulates ROMK channel activity by targeting intersectin 1. American Journal of Physiology - Renal Physiology, 2014, 306, F53-F60.	1.3	19
36	Src Family Protein Tyrosine Kinase Regulates the Basolateral K Channel in the Distal Convoluted Tubule (DCT) by Phosphorylation of KCNJ10 Protein. Journal of Biological Chemistry, 2013, 288, 26135-26146.	1.6	47

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37	Inhibition of miR-205 Impairs the Wound-Healing Process in Human Corneal Epithelial Cells by Targeting KIR4.1 (KCNJ10). , 2013, 54, 6167.		43
38	Srcâ€family tyrosine kinase (SFK) phosphorylates Withâ€No―Lysine Kinase4 (WNK4) and modulates the inhibitory effect of WNK4 on ROMK channels FASEB Journal, 2013, 27, 911.2.	0.2	0
39	HOâ€2 deletion impairs wound closure in Human Corneal Epithelial (HCE) cells by altering EGFR and FAK― mediated signaling pathways. FASEB Journal, 2013, 27, .	0.2	0
40	Srcâ€family protein tyrosine kinase (SFK) stimulates KCNJ10 K channels in the basolateral membrane of distal convoluted tubules (DCT) FASEB Journal, 2013, 27, 911.1.	0.2	0
41	Protein phosphatase 1 modulates the inhibitory effect of With-no-Lysine kinase 4 on ROMK channels. American Journal of Physiology - Renal Physiology, 2012, 303, F110-F119.	1.3	34
42	MironRNAâ€194 (mirâ€194) regulates ROMk channel activity by downâ€regulation of intersectin 1 (ITSN1)â€WI pathway. FASEB Journal, 2012, 26, lb754.	NK 0.2	0
43	Inhibition of miRâ€205 impairs the woundâ€healing process in human corneal epithelial cells by stimulating Kir4.1(KCNJ10). FASEB Journal, 2012, 26, lb682.	0.2	0
44	MicroRNA 802 Stimulates ROMK Channels by Suppressing Caveolin-1. Journal of the American Society of Nephrology: JASN, 2011, 22, 1087-1098.	3.0	52
45	Decrease in dietary K intake stimulates the generation of superoxide anions in the kidney and inhibits K secretory channels in the CCD. American Journal of Physiology - Renal Physiology, 2010, 298, F1515-F1522.	1.3	19
46	Angiotensin II Stimulates ENaC and suppresses the inhibitory effect of 11,12â€EET on ENaC in the cortical collecting duct (CCD). FASEB Journal, 2010, 24, 606.7.	0.2	0
47	Micro RNA 802 (Mirâ€802) stimulates ROMK channels by suppressing caveolinâ€1 expression during a high potassium (K) intake. FASEB Journal, 2010, 24, 626.1.	0.2	0
48	Angiotensin II (AngII) diminishes the effect of SGK1 on WNK4â€mediated inhibition of ROMK1 channels. FASEB Journal, 2010, 24, 610.2.	0.2	0
49	POSH Stimulates the Ubiquitination and the Clathrin-independent Endocytosis of ROMK1 Channels. Journal of Biological Chemistry, 2009, 284, 29614-29624.	1.6	24
50	Src family protein tyrosine kinase (PTK) modulates the effect of SGK1 and WNK4 on ROMK channels. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 15061-15066.	3.3	36
51	POSH (Plenty of SH3) is an E3 ligase of ROMK (Kir1.1) channels in cortical collecting duct (CCD). FASEB Journal, 2009, 23, 998.40.	0.2	0
52	Src family Protein tyrosine kinase(PTK) modulates the effect of SGK1 and WNK4 on ROMK channels FASEB Journal, 2009, 23, 1000.10.	0.2	0
53	Src family protein tyrosine kinase (PTK) and MAPK are involved in mediating the effect of low potassium intake (LK) on ROMK channels. FASEB Journal, 2009, 23, 998.24.	0.2	0
54	Expression of Tetraspan Protein CD63 Activates Protein-tyrosine Kinase (PTK) and Enhances the PTK-induced Inhibition of ROMK Channels. Journal of Biological Chemistry, 2008, 283, 7674-7681.	1.6	21

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55	POSH decreases ROMK1 channel activity through stimulating clatharinâ€independent and dynaminâ€dependent endocytosis FASEB Journal, 2008, 22, 1180.1.	0.2	0
56	Expression of tetraspanin protein CD63 enhances the PTKâ€induced inhibition of ROMK channels. FASEB Journal, 2007, 21, A1331.	0.2	0
57	Role of superoxide anions in mediating the effect of Kâ€restriction on ROMK channels and renal K excretion. FASEB Journal, 2007, 21, A1331.	0.2	0
58	The Protein Tyrosine Kinase-Dependent Pathway Mediates the Effect of K Intake on Renal K Secretion. Physiology, 2005, 20, 140-146.	1.6	26
59	ROMK1 channel activity is regulated by monoubiquitination. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 4306-4311.	3.3	45
60	Protein tyrosine kinase is expressed and regulates ROMK1 location in the cortical collecting duct. American Journal of Physiology - Renal Physiology, 2004, 286, F881-F892.	1.3	61
61	Inhibition of Protein-tyrosine Phosphatase Stimulates the Dynamin-dependent Endocytosis of ROMK1. Journal of Biological Chemistry, 2002, 277, 4317-4323.	1.6	50
62	Protein Kinase C (PKC)-induced Phosphorylation of ROMK1 Is Essential for the Surface Expression of ROMK1 Channels. Journal of Biological Chemistry, 2002, 277, 44278-44284.	1.6	50
63	K depletion increases protein tyrosine kinase-mediated phosphorylation of ROMK. American Journal of Physiology - Renal Physiology, 2002, 283, F671-F677.	1.3	44
64	Effect of dietary K intake on apical small-conductance K channel in CCD: role of protein tyrosine kinase. American Journal of Physiology - Renal Physiology, 2001, 281, F206-F212.	1.3	76