Christoph Korbmacher

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/8199416/publications.pdf

Version: 2024-02-01

62	2,748	159358 30	182168 51
papers	citations	h-index	g-index

64	64	64	2400
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	High baseline ROMK activity in the mouse late distal convoluted and early connecting tubule probably contributes to aldosterone-independent K⁺ secretion. American Journal of Physiology - Renal Physiology, 2022, 322, F42-F54.	1.3	10
2	Contributions of bile acids to gastrointestinal physiology as receptor agonists and modifiers of ion channels. American Journal of Physiology - Renal Physiology, 2022, 322, G201-G222.	1.6	11
3	Proteolytic activation of the epithelial sodium channel (ENaC) by factor VII activating protease (FSAP) and its relevance for sodium retention in nephrotic mice. Pflugers Archiv European Journal of Physiology, 2022, 474, 217-229.	1.3	17
4	Transmembrane serine protease 2 (TMPRSS2) proteolytically activates the epithelial sodium channel (ENaC) by cleaving the channelâ€™s Î³-subunit. Journal of Biological Chemistry, 2022, 298, 102004.	1.6	6
5	Two adjacent phosphorylation sites in the C-terminus of the channelâ€™s Î±-subunit have opposing effects on epithelial sodium channel (ENaC) activity. Pflugers Archiv European Journal of Physiology, 2022, 474, 681-697.	1.3	2
6	Zymogenâ€locked mutant prostasin (Prss8) leads to incomplete proteolytic activation of the epithelial sodium channel (ENaC) and severely compromises triamterene tolerance in mice. Acta Physiologica, 2021, 232, e13640.	1.8	18
7	A polycystin-2 protein with modified channel properties leads to an increased diameter of renal tubules and to renal cysts. Journal of Cell Science, 2021, 134, .	1.2	2
8	Critical role of the mineralocorticoid receptor in aldosterone-dependent and aldosterone-independent regulation of ENaC in the distal nephron. American Journal of Physiology - Renal Physiology, 2021, 321, F257-F268.	1.3	24
9	Inhibition of the epithelial sodium channel (ENaC) by connexin 30 involves stimulation of clathrin-mediated endocytosis. Journal of Biological Chemistry, 2021, 296, 100404.	1.6	9
10	Rebuttal to editorial: Sodium retention by uPA in nephrotic syndrome?. Acta Physiologica, 2020, 228, e13427.	1.8	3
11	Ubiquitination of renal ENaC subunits in vivo. American Journal of Physiology - Renal Physiology, 2020, 318, F1113-F1121.	1.3	16
12	Effects of syntaxins 2, 3, and 4 on rat and human epithelial sodium channel (ENaC) in Xenopus laevis oocytes. Pflugers Archiv European Journal of Physiology, 2020, 472, 461-471.	1.3	2
13	Prostaglandin E2 stimulates the epithelial sodium channel (ENaC) in cultured mouse cortical collecting duct cells in an autocrine manner. Journal of General Physiology, 2020, 152, .	0.9	13
14	Urokinaseâ€type plasminogen activator (uPA) is not essential for epithelial sodium channel (ENaC)â€mediated sodium retention in experimental nephrotic syndrome. Acta Physiologica, 2019, 227, e13286.	1.8	36
15	Bile acids inhibit human purinergic receptor P2X4 in a heterologous expression system. Journal of General Physiology, 2019, 151, 820-833.	0.9	9
16	The phosphorylation site T613 in the Î²-subunit of rat epithelial Na+ channel (ENaC) modulates channel inhibition by Nedd4-2. Pflugers Archiv European Journal of Physiology, 2018, 470, 649-660.	1.3	13
17	The degenerin region of the human bile acid-sensitive ion channel (BASIC) is involved in channel inhibition by calcium and activation by bile acids. Pflugers Archiv European Journal of Physiology, 2018, 470, 1087-1102.	1.3	8
18	Aprotinin prevents proteolytic epithelial sodium channel (ENaC) activation and volume retention in nephrotic syndrome. Kidney International, 2018, 93, 159-172.	2.6	77

#	Article	IF	Citations
19	Inhibitors of the proteasome stimulate the epithelial sodium channel (ENaC) through SGK1 and mimic the effect of aldosterone. Pflugers Archiv European Journal of Physiology, 2018, 470, 295-304.	1.3	12
20	ÎÎ²Î³-ENaC is inhibited by CFTR but stimulated by cAMP in Xenopus laevis oocytes. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2017, 312, L277-L287.	1.3	13
21	Bile acids potentiate protonâ€activated currents in Xenopus laevis oocytes expressing human acidâ€sensing ion channel (<scp>ASIC</scp>1a). Physiological Reports, 2017, 5, e13132.	0.7	11
22	In Liddle Syndrome, Epithelial Sodium Channel Is Hyperactive Mainly in the Early Part of the Aldosterone-Sensitive Distal Nephron. Hypertension, 2016, 67, 1256-1262.	1.3	34
23	Association of Plasminuria with Overhydration in Patients with CKD. Clinical Journal of the American Society of Nephrology: CJASN, 2016, 11, 761-769.	2.2	46
24	Activation of the Human Epithelial Sodium Channel (ENaC) by Bile Acids Involves the Degenerin Site. Journal of Biological Chemistry, 2016, 291, 19835-19847.	1.6	23
25	mTORC2 critically regulates renal potassium handling. Journal of Clinical Investigation, 2016, 126, 1773-1782.	3.9	37
26	Neutrophil Elastase Activates Protease-activated Receptor-2 (PAR2) and Transient Receptor Potential Vanilloid 4 (TRPV4) to Cause Inflammation and Pain. Journal of Biological Chemistry, 2015, 290, 13875-13887.	1.6	134
27	Mechanisms of Renal Control of Potassium Homeostasis in Complete Aldosterone Deficiency. Journal of the American Society of Nephrology: JASN, 2015, 26, 425-438.	3.0	66
28	Norepinephrine stimulates the epithelial Na⁺ channel in cortical collecting duct cells via Î±₂-adrenoceptors. American Journal of Physiology - Renal Physiology, 2015, 308, F450-F458.	1.3	20
29	Sensitisation of TRPV4 by PAR2 is independent of intracellular calcium signalling and can be mediated by the biased agonist neutrophil elastase. Pflugers Archiv European Journal of Physiology, 2015, 467, 687-701.	1.3	14
30	Cathepsin S Causes Inflammatory Pain via Biased Agonism of PAR2 and TRPV4. Journal of Biological Chemistry, 2014, 289, 27215-27234.	1.6	153
31	Proteolytic Activation of the Human Epithelial Sodium Channel by Trypsin IV and Trypsin I Involves Distinct Cleavage Sites. Journal of Biological Chemistry, 2014, 289, 19067-19078.	1.6	31
32	Pharmacological and electrophysiological characterization of the human bile acid-sensitive ion channel (hBASIC). Pflugers Archiv European Journal of Physiology, 2014, 466, 253-263.	1.3	23
33	Demonstration of Proteolytic Activation of the Epithelial Sodium Channel (ENaC) by Combining Current Measurements with Detection of Cleavage Fragments. Journal of Visualized Experiments, 2014, , .	0.2	4
34	A mutation in the Î²-subunit of ENaC identified in a patient with cystic fibrosis-like symptoms has a gain-of-function effect. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2013, 304, L43-L55.	1.3	25
35	Four Subunits (Îµ Î²Î³Î´) of the Epithelial Sodium Channel (ENaC) Are Expressed in the Human Eye in Various Locations. , 2012, 53, 596.		29
36	Plasmin and chymotrypsin have distinct preferences for channel activating cleavage sites in the Î³ subunit of the human epithelial sodium channel. Journal of General Physiology, 2012, 140, 375-389.	0.9	41

#	Article	IF	Citations
37	An Inhibitory Peptide Derived from the a-subunit of the Epithelial Sodium Channel (ENaC) Shows a Helical Conformation. Cellular Physiology and Biochemistry, 2012, 29, 761-774.	1.1	8
38	Proteolytic activation of the epithelial sodium channel (ENaC) by the cysteine protease cathepsin-S. Pflugers Archiv European Journal of Physiology, 2012, 464, 353-365.	1.3	54
39	Aldosterone-dependent and -independent regulation of the epithelial sodium channel (ENaC) in mouse distal nephron. American Journal of Physiology - Renal Physiology, 2012, 303, F1289-F1299.	1.3	82
40	Atomic Force Microscopy Reveals the Architecture of the Epithelial Sodium Channel (ENaC). Journal of Biological Chemistry, 2011, 286, 31944-31952.	1.6	53
41	A mutation of the epithelial sodium channel associated with atypical cystic fibrosis increases channel open probability and reduces Na⁺ self inhibition. Journal of Physiology, 2010, 588, 1211-1225.	1.3	83
42	Functional Characterization of a Partial Loss-of-Function Mutation of the Epithelial Sodium Channel (ENaC) Associated with Atypical Cystic Fibrosis. Cellular Physiology and Biochemistry, 2010, 25, 145-158.	1.1	27
43	Protein Kinase B Alpha (PKBα) Stimulates the Epithelial Sodium Channel (ENaC) Heterologously Expressed in <i>Xenopus laevis</i> Oocytes by Two Distinct Mechanisms. Cellular Physiology and Biochemistry, 2010, 26, 913-924.	1.1	25
44	The Î´-Subunit of the Epithelial Sodium Channel (ENaC) Enhances Channel Activity and Alters Proteolytic ENaC Activation. Journal of Biological Chemistry, 2009, 284, 29024-29040.	1.6	67
45	Cholesterol Depletion of the Plasma Membrane Prevents Activation of the Epithelial Sodium Channel (ENaC) by SGK1. Cellular Physiology and Biochemistry, 2009, 24, 605-618.	1.1	51
46	Plasmin in Nephrotic Urine Activates the Epithelial Sodium Channel. Journal of the American Society of Nephrology: JASN, 2009, 20, 299-310.	3.0	236
47	Regulated sodium transport in the renal connecting tubule (CNT) via the epithelial sodium channel (ENaC). Pflugers Archiv European Journal of Physiology, 2009, 458, 111-135.	1.3	142
48	Aldosterone responsiveness of the epithelial sodium channel (ENaC) in colon is increased in a mouse model for Liddle's syndrome. Journal of Physiology, 2008, 586, 459-475.	1.3	50
49	Cleavage in the Î³â€subunit of the epithelial sodium channel (ENaC) plays an important role in the proteolytic activation of nearâ€silent channels. Journal of Physiology, 2008, 586, 4587-4608.	1.3	87
50	Trypsin can activate the epithelial sodium channel (ENaC) in microdissected mouse distal nephron. American Journal of Physiology - Renal Physiology, 2008, 295, F1052-F1062.	1.3	44
51	Stimulation of the epithelial sodium channel (ENaC) by the serum- and glucocorticoid-inducible kinase (Sgk) involves the PY motifs of the channel but is independent of sodium feedback inhibition. Pflugers Archiv European Journal of Physiology, 2006, 452, 290-299.	1.3	27
52	Stimulation of the Epithelial Sodium Channel (ENaC) by cAMP Involves Putative ERK Phosphorylation Sites in the C Termini of the Channel's Î²- and Î³-Subunit. Journal of Biological Chemistry, 2006, 281, 9859-9868.	1.6	60
53	A Novel Pathway of Epithelial Sodium Channel Activation Involves a Serum- and Glucocorticoid-inducible Kinase Consensus Motif in the C Terminus of the Channel's Î±-Subunit. Journal of Biological Chemistry, 2004, 279, 38134-38142.	1.6	155
54	Extracellular Na + removal attenuates rundown of the epithelial Na + -channel (ENaC) by reducing the rate of channel retrieval. Pflugers Archiv European Journal of Physiology, 2004, 447, 884-894.	1.3	33

#	Article	IF	Citations
55	PGE2 stimulates Cl? secretion in murine M-1 cortical collecting duct cells in an autocrine manner. Pflugers Archiv European Journal of Physiology, 2004, 448, 411-21.	1.3	11
56	The role of individual Nedd4â€“2 (KIAA0439) WW domains in binding and regulating epithelial sodium channels. FASEB Journal, 2003, 17, 70-72.	0.2	96
57	Regulation of the Epithelial Sodium Channel by N4WBP5A, a Novel Nedd4/Nedd4-2-interacting Protein. Journal of Biological Chemistry, 2002, 277, 29406-29416.	1.6	85
58	Basolateral adrenoceptor activation mediates noradrenaline-induced Cl - secretion in M-1 mouse cortical collecting duct cells. Pflugers Archiv European Journal of Physiology, 2002, 445, 381-389.	1.3	8
59	Basolateral PARâ€2 receptors mediate KCl secretion and inhibition of Na + absorption in the mouse distal colon. Journal of Physiology, 2002, 539, 209-222.	1.3	58
60	Sulfonylurea receptors inhibit the epithelial sodium channel (ENaC) by reducing surface expression. Pflugers Archiv European Journal of Physiology, 2001, 442, 752-761.	1.3	32
61	ATP stimulates Cl â^' secretion and reduces amilorideâ€sensitive Na + absorption in Mâ€1 mouse cortical collecting duct cells. Journal of Physiology, 2000, 524, 77-90.	1.3	112
62	Basolateral proteinaseâ€activated receptor (PARâ€2) induces chloride secretion in Mâ€1 mouse renal cortical collecting duct cells. Journal of Physiology, 1999, 521, 3-17.	1.3	70