

Jian-Kang Chen

List of Publications by Year in descending order

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Version: 2024-02-01

19
papers

5,399
citations

840776

11
h-index

839539

18
g-index

19
all docs

19
docs citations

19
times ranked

14912
citing authors

#	ARTICLE	IF	CITATIONS
1	Blocking ribosomal protein S6 phosphorylation inhibits podocyte hypertrophy and focal segmental glomerulosclerosis. <i>Kidney International</i> , 2022, , .	5.2	3
2	The expression level of class III phosphatidylinositol-3 kinase controls the degree of compensatory nephron hypertrophy. <i>American Journal of Physiology - Renal Physiology</i> , 2020, 318, F628-F638.	2.7	6
3	Cre/loxP approach-mediated downregulation of Pik3c3 inhibits the hypertrophic growth of renal proximal tubule cells. <i>Journal of Cellular Physiology</i> , 2020, 235, 9958-9973.	4.1	4
4	Metformin effectively treats Tsc1 deletion-caused kidney pathology by upregulating AMPK phosphorylation. <i>Cell Death Discovery</i> , 2020, 6, 52.	4.7	13
5	The TNF-derived TIP peptide activates the epithelial sodium channel and ameliorates experimental nephrotoxic serum nephritis. <i>Kidney International</i> , 2019, 95, 1359-1372.	5.2	11
6	Dicer deficiency in proximal tubules exacerbates renal injury and tubulointerstitial fibrosis and upregulates Smad2/3. <i>American Journal of Physiology - Renal Physiology</i> , 2018, 315, F1822-F1832.	2.7	14
7	DNA methylation protects against cisplatin-induced kidney injury by regulating specific genes, including interferon regulatory factor 8. <i>Kidney International</i> , 2017, 92, 1194-1205.	5.2	43
8	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). <i>Autophagy</i> , 2016, 12, 1-222.	9.1	4,701
9	Blocking rpS6 Phosphorylation Exacerbates Tsc1 Deletion-Induced Kidney Growth. <i>Journal of the American Society of Nephrology: JASN</i> , 2016, 27, 1145-1158.	6.1	10
10	Overexpression of G-Protein-Coupled Receptor 40 Enhances the Mitogenic Response to Epoxyeicosatrienoic Acids. <i>PLoS ONE</i> , 2015, 10, e0113130.	2.5	19
11	Phosphorylation of ribosomal protein S6 mediates compensatory renal hypertrophy. <i>Kidney International</i> , 2015, 87, 543-556.	5.2	26
12	EGF Receptor Deletion in Podocytes Attenuates Diabetic Nephropathy. <i>Journal of the American Society of Nephrology: JASN</i> , 2015, 26, 1115-1125.	6.1	109
13	Phosphatidylinositol 3-kinase signaling determines kidney size. <i>Journal of Clinical Investigation</i> , 2015, 125, 2429-2444.	8.2	55
14	mVps34 Deletion in Podocytes Causes Glomerulosclerosis by Disrupting Intracellular Vesicle Trafficking. <i>Journal of the American Society of Nephrology: JASN</i> , 2013, 24, 198-207.	6.1	72
15	PIK3C3/VPS34, the class III PtdIns 3-kinase, plays indispensable roles in the podocyte. <i>Autophagy</i> , 2013, 9, 923-924.	9.1	2
16	Renal Collecting Duct Cell-specific mVps34 Deletion Decreases Nephron Number and Increases Nephron Size. <i>FASEB Journal</i> , 2013, 27, 705.11.	0.5	0
17	S6 kinase 1 knockout inhibits uninephrectomy- or diabetes-induced renal hypertrophy. <i>American Journal of Physiology - Renal Physiology</i> , 2009, 297, F585-F593.	2.7	68
18	Role of Mammalian Target of Rapamycin Signaling in Compensatory Renal Hypertrophy. <i>Journal of the American Society of Nephrology: JASN</i> , 2005, 16, 1384-1391.	6.1	120

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19	Epoxyeicosatrienoic Acids and Their Sulfonimide Derivatives Stimulate Tyrosine Phosphorylation and Induce Mitogenesis in Renal Epithelial Cells. <i>Journal of Biological Chemistry</i> , 1998, 273, 29254-29261.	3.4	123