

List of Publications by Year in descending order

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54
papers

5,669
citations

147726

31
h-index

161767

54
g-index

55
all docs

55
docs citations

55
times ranked

7560
citing authors

#	ARTICLE	IF	CITATIONS
1	PACS-2 Ameliorates Tubular Injury by Facilitating Endoplasmic Reticulum-Mitochondria Contact and Mitophagy in Diabetic Nephropathy. <i>Diabetes</i> , 2022, 71, 1034-1050.	0.3	29
2	Mitochondrial DNA-dependent inflammation in kidney diseases. <i>International Immunopharmacology</i> , 2022, 107, 108637.	1.7	2
3	MAMs Protect Against Ectopic Fat Deposition and Lipid-Related Kidney Damage in DN Patients. <i>Frontiers in Endocrinology</i> , 2021, 12, 609580.	1.5	14
4	DsbA-L Ameliorates Renal Injury Through the AMPK/NLRP3 Inflammasome Signaling Pathway in Diabetic Nephropathy. <i>Frontiers in Physiology</i> , 2021, 12, 659751.	1.3	15
5	Mitochondria-Associated Membranes (MAMs): A Novel Therapeutic Target for Treating Metabolic Syndrome. <i>Current Medicinal Chemistry</i> , 2021, 28, 1347-1362.	1.2	21
6	Effects of HIF-1 α on renal fibrosis in cisplatin-induced chronic kidney disease. <i>Clinical Science</i> , 2021, 135, 1273-1288.	1.8	19
7	Mitophagy: A Novel Therapeutic Target for Treating DN. <i>Current Medicinal Chemistry</i> , 2021, 28, 2717-2728.	1.2	12
8	The Loss of Mitochondrial Quality Control in Diabetic Kidney Disease. <i>Frontiers in Cell and Developmental Biology</i> , 2021, 9, 706832.	1.8	20
9	Lipophagy deficiency exacerbates ectopic lipid accumulation and tubular cells injury in diabetic nephropathy. <i>Cell Death and Disease</i> , 2021, 12, 1031.	2.7	37
10	The CXCL1-CXCR2 Axis Mediates Tubular Injury in Diabetic Nephropathy Through the Regulation of the Inflammatory Response. <i>Frontiers in Physiology</i> , 2021, 12, 782677.	1.3	10
11	Design and validation of a scoring model for differential diagnosis of diabetic nephropathy and nondiabetic renal diseases in type 2 diabetic patients. <i>Journal of Diabetes</i> , 2020, 12, 237-246.	0.8	10
12	PACS-2: A key regulator of mitochondria-associated membranes (MAMs). <i>Pharmacological Research</i> , 2020, 160, 105080.	3.1	42
13	Mitochondria-Associated ER Membranes - The Origin Site of Autophagy. <i>Frontiers in Cell and Developmental Biology</i> , 2020, 8, 595.	1.8	75
14	Aristolochic acid induces renal fibrosis by arresting proximal tubular cells in G2/M phase mediated by HIF-1 α . <i>FASEB Journal</i> , 2020, 34, 12599-12614.	0.2	19
15	Tacrolimus ameliorates tubulointerstitial inflammation in diabetic nephropathy via inhibiting the NFATc1/TRPC6 pathway. <i>Journal of Cellular and Molecular Medicine</i> , 2020, 24, 9810-9824.	1.6	13
16	HIF-1 α ameliorates tubular injury in diabetic nephropathy via HO-1-mediated control of mitochondrial dynamics. <i>Cell Proliferation</i> , 2020, 53, e12909.	2.4	74
17	Mitochondria-Associated Endoplasmic Reticulum Membranes (MAMs) and Their Prospective Roles in Kidney Disease. <i>Oxidative Medicine and Cellular Longevity</i> , 2020, 2020, 1-21.	1.9	29
18	AdipoRon Protects against Tubular Injury in Diabetic Nephropathy by Inhibiting Endoplasmic Reticulum Stress. <i>Oxidative Medicine and Cellular Longevity</i> , 2020, 2020, 1-15.	1.9	6

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19	The single nucleotide polymorphism rs11643718 in <i>SLC12A3</i> is associated with the development of diabetic kidney disease in Chinese people with type 2 diabetes. <i>Diabetic Medicine</i> , 2020, 37, 1879-1889.	1.2	8
20	DsbA-L deficiency exacerbates mitochondrial dysfunction of tubular cells in diabetic kidney disease. <i>Clinical Science</i> , 2020, 134, 677-694.	1.8	25
21	Metabolomics window into the role of acute kidney injury after coronary artery bypass grafting in diabetic nephropathy progression. <i>PeerJ</i> , 2020, 8, e9111.	0.9	4
22	A Glimpse of the Mechanisms Related to Renal Fibrosis in Diabetic Nephropathy. <i>Advances in Experimental Medicine and Biology</i> , 2019, 1165, 49-79.	0.8	82
23	DsbA-L ameliorates high glucose induced tubular damage through maintaining MAM integrity. <i>EBioMedicine</i> , 2019, 43, 607-619.	2.7	53
24	Disulfide-bond A oxidoreductase-like protein protects against ectopic fat deposition and lipid-related kidney damage in diabetic nephropathy. <i>Kidney International</i> , 2019, 95, 880-895.	2.6	54
25	Multiple microarray analysis for identification of hub genes involved in tubulointerstitial injury in diabetic nephropathy. <i>Journal of Cellular Physiology</i> , 2019, 234, 16447-16462.	2.0	43
26	Reactive oxygen species promote tubular injury in diabetic nephropathy: The role of the mitochondrial ros-txnip-nlrp3 biological axis. <i>Redox Biology</i> , 2018, 16, 32-46.	3.9	269
27	The Kidney Specific Protein myo-Inositol Oxygenase, a Potential Biomarker for Diabetic Nephropathy. <i>Kidney and Blood Pressure Research</i> , 2018, 43, 1772-1785.	0.9	7
28	Ectopic lipid accumulation: potential role in tubular injury and inflammation in diabetic kidney disease. <i>Clinical Science</i> , 2018, 132, 2407-2422.	1.8	53
29	The Susceptibility Genes in Diabetic Nephropathy. <i>Kidney Diseases (Basel, Switzerland)</i> , 2018, 4, 226-237.	1.2	51
30	Perturbations in mitochondrial dynamics by p66Shc lead to renal tubular oxidative injury in human diabetic nephropathy. <i>Clinical Science</i> , 2018, 132, 1297-1314.	1.8	36
31	The mitochondria-targeted antioxidant MitoQ ameliorated tubular injury mediated by mitophagy in diabetic kidney disease via Nrf2/PINK1. <i>Redox Biology</i> , 2017, 11, 297-311.	3.9	383
32	Normoalbuminuric diabetic kidney disease. <i>Frontiers of Medicine</i> , 2017, 11, 310-318.	1.5	85
33	Probucol ameliorates renal injury in diabetic nephropathy by inhibiting the expression of the redox enzyme p66Shc. <i>Redox Biology</i> , 2017, 13, 482-497.	3.9	43
34	Mitochondria: A Novel Therapeutic Target in Diabetic Nephropathy. <i>Current Medicinal Chemistry</i> , 2017, 24, 3185-3202.	1.2	58
35	Involvement of the NLR4-Inflammasome in Diabetic Nephropathy. <i>PLoS ONE</i> , 2016, 11, e0164135.	1.1	42
36	p66Shc: A novel biomarker of tubular oxidative injury in patients with diabetic nephropathy. <i>Scientific Reports</i> , 2016, 6, 29302.	1.6	36

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37	Validation of the interstitial fibrosis and tubular atrophy on the new pathological classification in patients with diabetic nephropathy: A single-center study in China. <i>Journal of Diabetes and Its Complications</i> , 2016, 30, 537-541.	1.2	14
38	myo-Inositol Oxygenase Overexpression Accentuates Generation of Reactive Oxygen Species and Exacerbates Cellular Injury following High Glucose Ambience. <i>Journal of Biological Chemistry</i> , 2016, 291, 5688-5707.	1.6	27
39	Repression of Hox genes by LMP1 in nasopharyngeal carcinoma and modulation of glycolytic pathway genes by HoxC8. <i>Oncogene</i> , 2015, 34, 6079-6091.	2.6	50
40	Disruption of Renal Tubular Mitochondrial Quality Control by Myo-Inositol Oxygenase in Diabetic Kidney Disease. <i>Journal of the American Society of Nephrology: JASN</i> , 2015, 26, 1304-1321.	3.0	228
41	Insights into the Mechanisms Involved in the Expression and Regulation of Extracellular Matrix Proteins in Diabetic Nephropathy. <i>Current Medicinal Chemistry</i> , 2015, 22, 2858-2870.	1.2	156
42	Rap1 Ameliorates Renal Tubular Injury in Diabetic Nephropathy. <i>Diabetes</i> , 2014, 63, 1366-1380.	0.3	105
43	A Glimpse of Matrix Metalloproteinases in Diabetic Nephropathy. <i>Current Medicinal Chemistry</i> , 2014, 21, 3244-3260.	1.2	68
44	Mitochondrial dynamics: regulatory mechanisms and emerging role in renal pathophysiology. <i>Kidney International</i> , 2013, 83, 568-581.	2.6	298
45	Cyclic GMP-AMP Is an Endogenous Second Messenger in Innate Immune Signaling by Cytosolic DNA. <i>Science</i> , 2013, 339, 826-830.	6.0	1,778
46	A Glimpse of the Pathogenetic Mechanisms of Wnt/ β -Catenin Signaling in Diabetic Nephropathy. <i>BioMed Research International</i> , 2013, 2013, 1-7.	0.9	70
47	A Glimpse of Various Pathogenetic Mechanisms of Diabetic Nephropathy. <i>Annual Review of Pathology: Mechanisms of Disease</i> , 2011, 6, 395-423.	9.6	575
48	Epac1-Mediated, High Glucose-Induced Renal Proximal Tubular Cells Hypertrophy via the Akt/p21 Pathway. <i>American Journal of Pathology</i> , 2011, 179, 1706-1718.	1.9	28
49	Low-dose paclitaxel ameliorates fibrosis in the remnant kidney model by downregulating miR-192. <i>Journal of Pathology</i> , 2011, 225, 364-377.	2.1	105
50	Pathobiology of renal-specific oxidoreductase/myo-inositol oxygenase in diabetic nephropathy: its implications in tubulointerstitial fibrosis. <i>American Journal of Physiology - Renal Physiology</i> , 2010, 298, F1393-F1404.	1.3	21
51	p66Shc mediates high-glucose and angiotensin II-induced oxidative stress renal tubular injury via mitochondrial-dependent apoptotic pathway. <i>American Journal of Physiology - Renal Physiology</i> , 2010, 299, F1014-F1025.	1.3	95
52	Blockade of the erbB2 Receptor Induces Cardiomyocyte Death through Mitochondrial and Reactive Oxygen Species-dependent Pathways. <i>Journal of Biological Chemistry</i> , 2009, 284, 2080-2087.	1.6	152
53	Rap1b GTPase Ameliorates Glucose-Induced Mitochondrial Dysfunction. <i>Journal of the American Society of Nephrology: JASN</i> , 2008, 19, 2293-2301.	3.0	67
54	Modulation of renal-specific oxidoreductase/myo-inositol oxygenase by high-glucose ambience. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2005, 102, 17952-17957.	3.3	53