Wilhelm Kriz

List of Publications by Year
in descending order

74 papers	10,231 citations	57758 44 h-index	82547 72 g-index
75 all docs	75 docs citations	75 times ranked	7938 citing authors

#	Article	IF	Citations
1	A new view of macula densa cell microanatomy. American Journal of Physiology - Renal Physiology, 2021, 320, F492-F504.	2.7	13
2	Pathomorphological sequence of nephron loss in diabetic nephropathy. American Journal of Physiology - Renal Physiology, 2021, 321, F600-F616.	2.7	9
3	The Inability of Podocytes to Proliferate: Cause, Consequences, and Origin. Anatomical Record, 2020, 303, 2588-2596.	1.4	15
4	Herniation of the tuft with outgrowth of vessels through the glomerular entrance in diabetic nephropathy damages the juxtaglomerular apparatus. American Journal of Physiology - Renal Physiology, 2019, 317, F399-F410.	2.7	7
5	Upregulation of Tumor Susceptibility Gene 101 (TSG101) by mechanical stress in podocytes. Cellular and Molecular Biology, 2019, 65, 84-88.	0.9	0
6	Inverse correlation between vascular endothelial growth factor back-filtration and capillary filtration pressures. Nephrology Dialysis Transplantation, 2018, 33, 1514-1525.	0.7	7
7	Maintenance and Breakdown of Glomerular Tuft Architecture. Journal of the American Society of Nephrology: JASN, 2018, 29, 1075-1077.	6.1	14
8	Mechanical challenges to the glomerular filtration barrier: adaptations and pathway to sclerosis. Pediatric Nephrology, 2017, 32, 405-417.	1.7	61
9	Pals1 Haploinsufficiency Results in Proteinuria and Cyst Formation. Journal of the American Society of Nephrology: JASN, 2017, 28, 2093-2107.	6.1	33
10	Potential relevance of shear stress for slit diaphragm and podocyte function. Kidney International, 2017, 91, 1283-1286.	5.2	48
11	Accumulation of worn-out GBM material substantially contributes to mesangial matrix expansion in diabetic nephropathy. American Journal of Physiology - Renal Physiology, 2017, 312, F1101-F1111.	2.7	30
12	Combined use of electron microscopy and intravital imaging captures morphological and functional features of podocyte detachment. Pflugers Archiv European Journal of Physiology, 2017, 469, 965-974.	2.8	11
13	A Potential Role for Mechanical Forces in the Detachment of Podocytes and the Progression of CKD. Journal of the American Society of Nephrology: JASN, 2015, 26, 258-269.	6.1	214
14	Failed Tubule Recovery, AKI-CKD Transition, and Kidney Disease Progression. Journal of the American Society of Nephrology: JASN, 2015, 26, 1765-1776.	6.1	520
15	Structural Analysis of How Podocytes Detach from the Glomerular Basement Membrane Under Hypertrophic Stress. Frontiers in Endocrinology, 2014, 5, 207.	3.5	42
16	Structural Organization of the Mammalian Kidney. , 2013, , 595-691.		42
17	Renal epithelial injury and fibrosis. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2013, 1832, 931-939.	3.8	130
18	The podocyte's response to stress: the enigma of foot process effacement. American Journal of Physiology - Renal Physiology, 2013, 304, F333-F347.	2.7	231

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19	PTEN loss defines a TGF-Î²-induced tubule phenotype of failed differentiation and JNK signaling during renal fibrosis. American Journal of Physiology - Renal Physiology, 2012, 302, F1210-F1223.	2.7	99
20	Podocyte hypertrophy mismatch and glomerular disease. Nature Reviews Nephrology, 2012, 8, 618-619.	9.6	33
21	Epithelial-mesenchymal transition (EMT) in kidney fibrosis: fact or fantasy?. Journal of Clinical Investigation, 2011, 121, 468-474.	8.2	382
22	Tubular Overexpression of Transforming Growth Factor-Î²1 Induces Autophagy and Fibrosis but Not Mesenchymal Transition of Renal Epithelial Cells. American Journal of Pathology, 2010, 177, 632-643.	3.8	254
23	Recruitment of Podocytes from Glomerular Parietal Epithelial Cells. Journal of the American Society of Nephrology: JASN, 2009, 20, 333-343.	6.1	418
24	Palladin is a dynamic actin-associated protein in podocytes. Kidney International, 2009, 75, 214-226.	5.2	47
25	Effects of Increased Renal Tubular Vascular Endothelial Growth Factor (VEGF) on Fibrosis, Cyst Formation, and Glomerular Disease. American Journal of Pathology, 2009, 175, 1883-1895.	3.8	96
26	An efficient and versatile system for acute and chronic modulation of renal tubular function in transgenic mice. Nature Medicine, 2008, 14, 979-984.	30.7	253
27	Structural Organization of the Mammalian Kidney. , 2008, , 479-563.		22
28	CD2AP/CIN85 Balance Determines Receptor Tyrosine Kinase Signaling Response in Podocytes. Journal of Biological Chemistry, 2007, 282, 7457-7464.	3.4	33
29	Abrogation of Protein Uptake through Megalin-Deficient Proximal Tubules Does Not Safeguard against Tubulointerstitial Injury. Journal of the American Society of Nephrology: JASN, 2007, 18, 1824-1834.	6.1	87
30	New insights into structural patterns encountered in glomerulosclerosis. Current Opinion in Nephrology and Hypertension, 2007, 16, 184-191.	2.0	38
31	Ontogenetic Development of the Filtration Barrier. Nephron Experimental Nephrology, 2007, 106, e44-e50.	2.2	18
32	Podocytes are sensitive to fluid shear stress in vitro. American Journal of Physiology - Renal Physiology, 2006, 291, F856-F865.	2.7	115
33	Pathways to nephron loss starting from glomerular diseasesâ€"Insights from animal models. Kidney International, 2005, 67, 404-419.	5.2	391
34	TRPC6 â€“ a new podocyte gene involved in focal segmental glomerulosclerosis. Trends in Molecular Medicine, 2005, 11, 527-530.	6.7	40
35	Angiotensin II Type 1 Receptor Overexpression in Podocytes Induces Glomerulosclerosis in Transgenic Rats. Journal of the American Society of Nephrology: JASN, 2004, 15, 1475-1487.	6.1	186
36	Podocytes Populate Cellular Crescents in a Murine Model of Inflammatory Glomerulonephritis. Journal of the American Society of Nephrology: JASN, 2004, 15, 61-67.	6.1	166

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37	Adenosine and ATP: traffic regulators in the kidney. Journal of Clinical Investigation, 2004, 114, 611-613.	8.2	20
38	Progression of chronic renal failure in focal segmental glomerulosclerosis: consequence of podocyte damage or of tubulointerstitial fibrosis?. Pediatric Nephrology, 2003, 18, 617-622.	1.7	33
39	SCG/Kinjoh mice: A model of ANCA-associated crescentic glomerulonephritis with immune deposits. Kidney International, 2003, 64, 140-148.	5.2	55
40	Cell Biology of the Glomerular Podocyte. Physiological Reviews, 2003, 83, 253-307.	28.8	1,285
41	Pathways to Recovery and Loss of Nephrons in Anti-Thy-1 Nephritis. Journal of the American Society of Nephrology: JASN, 2003, 14, 1904-1926.	6.1	73
42	The pathogenesis of 'classic' focal segmental glomerulosclerosis--lessons from rat models. Nephrology Dialysis Transplantation, 2003, 18, 39vi-44.	0.7	53
43	Analysis of differential gene expression in stretched podocytes: osteopontin enhances adaptation of podocytes to mechanical stress. FASEB Journal, 2002, 16, 1-24.	0.5	78
44	Altered Expression Pattern of Polycystin-2 in Acute and Chronic Renal Tubular Diseases. Journal of the American Society of Nephrology: JASN, 2002, 13, 1855-1864.	6.1	13
45	Podocyte is the major culprit accounting for the progression of chronic renal disease. Microscopy Research and Technique, 2002, 57, 189-195.	2.2	151
46	The LIM-homeodomain transcription factor Lmx1b plays a crucial role in podocytes. Journal of Clinical Investigation, 2002, 109, 1073-1082.	8.2	100
47	Process formation of podocytes: morphogenetic activity of microtubules and regulation by protein serine/threonine phosphatase PP2A. Histochemistry and Cell Biology, 2001, 115, 255-266.	1.7	43
48	Podocyte injury underlies the progression of focal segmental glomerulosclerosis in the fa/fa Zucker rat. Kidney International, 2001, 60, 106-116.	5.2	85
49	Podocyte Bridges between the Tuft and Bowman's Capsule. Journal of the American Society of Nephrology: JASN, 2001, 12, 2060-2071.	6.1	122
50	Podocytes Respond to Mechanical Stress In Vitro. Journal of the American Society of Nephrology: JASN, 2001, 12, 413-422.	6.1	252
51	Tracer Studies in the Rat Demonstrate Misdirected Filtration and Peritubular Filtrate Spreading in Nephrons with Segmental Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2001, 12, 496-506.	6.1	74
52	Antibodies against Vascular Endothelial Growth Factor Improve Early Renal Dysfunction in Experimental Diabetes. Journal of the American Society of Nephrology: JASN, 2001, 12, 993-1000.	6.1	394
53	The Glomerular Slit Diaphragm Is a Modified Adherens Junction. Journal of the American Society of Nephrology: JASN, 2000, 11, 1-8.	6.1	384
54	Early Mechanisms of Renal Injury in Hypercholesterolemic or Hypertriglyceridemic Rats. Journal of the American Society of Nephrology: JASN, 2000, 11, 669-683.	6.1	159

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55	Altered gene expression and functions of mitochondria in human nephrotic syndrome. FASEB Journal, 1999, 13, 523-532.	0.5	53
56	The role of the podocyte in glomerulosclerosis. Current Opinion in Nephrology and Hypertension, 1999, 8, 489-497.	2.0	137
57	The Dysregulated Podocyte Phenotype. Journal of the American Society of Nephrology: JASN, 1999, 10, 51-61.	6.1	459
58	Whole-body hyperthermia combined with ifosfamide and carboplatin causes hypotension and nephrotoxicity. Journal of Cancer Research and Clinical Oncology, 1998, 124, 549-554.	2.5	9
59	New aspects of podocyte structure, function, and pathology. Clinical and Experimental Nephrology, 1998, 2, 85-99.	1.6	11
60	Progression of glomerular diseases: Is the podocyte the culprit?. Kidney International, 1998, 54, 687-697.	5.2	525
61	Nonuniform Microtubular Polarity Established by CHO1/MKLP1 Motor Protein Is Necessary for Process Formation of Podocytes. Journal of Cell Biology, 1998, 143, 1961-1970.	5.2	45
62	Mineralocorticoid receptor knockout mice: Pathophysiology of Na ⁺ metabolism. Proceedings of the National Academy of Sciences of the United States of America, 1998, 95, 9424-9429.	7.1	393
63	Kid-1 expression is high in differentiated renal proximal tubule cells and suppressed in cyst epithelia. American Journal of Physiology - Renal Physiology, 1998, 275, F928-F937.	2.7	5
64	Age-related glomerulosclerosis and interstitial fibrosis in Milan normotensive rats: A podocyte disease. Kidney International, 1997, 51, 230-243.	5.2	117
65	Long-term treatment of rats with FGF-2 results in focal segmental glomerulosclerosis. Kidney International, 1995, 48, 1435-1450.	5.2	173
66	A role for podocytes to counteract capillary wall distension. Kidney International, 1994, 45, 369-376.	5.2	193
67	The Contractile Apparatus of Podocytes Is Arranged to Counteract GBM Expansion. Contributions To Nephrology, 1994, 107, 1-9.	1.1	26
68	Glomerular damage after uninephrectomy in young rats. I. Hypertrophy and distortion of capillary architecture. Kidney International, 1992, 42, 136-147.	5.2	131
69	Glomerular damage after uninephrectomy in young rats. II. Mechanical stress on podocytes as a pathway to sclerosis. Kidney International, 1992, 42, 148-160.	5.2	229
70	Changes in glomerular structure following acute mesangial failure in the isolated perfused kidney. Kidney International, 1992, 41, 533-541.	5.2	21
71	Glomerular Injury in Analbuminemic Rats after Subtotal Nephrectomy. Nephron, 1991, 59, 104-109.	1.8	9
72	Role of mesangial cell contraction in adaptation of the glomerular tuft to changes in extracellular volume. Pflugers Archiv European Journal of Physiology, 1990, 415, 598-605.	2.8	23

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73	The structural relationship between mesangial cells and basement membrane of the renal glomerulus. Anatomy and Embryology, 1987, 176, 373-386.	1.5	184
74	Scanning electron microscopy studies of the vascular pole of the rat glomerulus. The Anatomical Record, 1982, 204, 149-152.	1.8	6