

Wilhelm Kriz

List of Publications by Year in descending order

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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	Cell Biology of the Glomerular Podocyte. <i>Physiological Reviews</i> , 2003, 83, 253-307.	28.8	1,285
2	Progression of glomerular diseases: Is the podocyte the culprit?. <i>Kidney International</i> , 1998, 54, 687-697.	5.2	525
3	Failed Tubule Recovery, AKI-CKD Transition, and Kidney Disease Progression. <i>Journal of the American Society of Nephrology: JASN</i> , 2015, 26, 1765-1776.	6.1	520
4	The Dysregulated Podocyte Phenotype. <i>Journal of the American Society of Nephrology: JASN</i> , 1999, 10, 51-61.	6.1	459
5	Recruitment of Podocytes from Glomerular Parietal Epithelial Cells. <i>Journal of the American Society of Nephrology: JASN</i> , 2009, 20, 333-343.	6.1	418
6	Antibodies against Vascular Endothelial Growth Factor Improve Early Renal Dysfunction in Experimental Diabetes. <i>Journal of the American Society of Nephrology: JASN</i> , 2001, 12, 993-1000.	6.1	394
7	Mineralocorticoid receptor knockout mice: Pathophysiology of Na ⁺ metabolism. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 1998, 95, 9424-9429.	7.1	393
8	Pathways to nephron loss starting from glomerular diseases—Insights from animal models. <i>Kidney International</i> , 2005, 67, 404-419.	5.2	391
9	The Glomerular Slit Diaphragm Is a Modified Adherens Junction. <i>Journal of the American Society of Nephrology: JASN</i> , 2000, 11, 1-8.	6.1	384
10	Epithelial-mesenchymal transition (EMT) in kidney fibrosis: fact or fantasy?. <i>Journal of Clinical Investigation</i> , 2011, 121, 468-474.	8.2	382
11	Tubular Overexpression of Transforming Growth Factor- β 21 Induces Autophagy and Fibrosis but Not Mesenchymal Transition of Renal Epithelial Cells. <i>American Journal of Pathology</i> , 2010, 177, 632-643.	3.8	254
12	An efficient and versatile system for acute and chronic modulation of renal tubular function in transgenic mice. <i>Nature Medicine</i> , 2008, 14, 979-984.	30.7	253
13	Podocytes Respond to Mechanical Stress In Vitro. <i>Journal of the American Society of Nephrology: JASN</i> , 2001, 12, 413-422.	6.1	252
14	The podocyte's response to stress: the enigma of foot process effacement. <i>American Journal of Physiology - Renal Physiology</i> , 2013, 304, F333-F347.	2.7	231
15	Glomerular damage after uninephrectomy in young rats. II. Mechanical stress on podocytes as a pathway to sclerosis. <i>Kidney International</i> , 1992, 42, 148-160.	5.2	229
16	A Potential Role for Mechanical Forces in the Detachment of Podocytes and the Progression of CKD. <i>Journal of the American Society of Nephrology: JASN</i> , 2015, 26, 258-269.	6.1	214
17	A role for podocytes to counteract capillary wall distension. <i>Kidney International</i> , 1994, 45, 369-376.	5.2	193
18	Angiotensin II Type 1 Receptor Overexpression in Podocytes Induces Glomerulosclerosis in Transgenic Rats. <i>Journal of the American Society of Nephrology: JASN</i> , 2004, 15, 1475-1487.	6.1	186

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19	The structural relationship between mesangial cells and basement membrane of the renal glomerulus. <i>Anatomy and Embryology</i> , 1987, 176, 373-386.	1.5	184
20	Long-term treatment of rats with FGF-2 results in focal segmental glomerulosclerosis. <i>Kidney International</i> , 1995, 48, 1435-1450.	5.2	173
21	Podocytes Populate Cellular Crescents in a Murine Model of Inflammatory Glomerulonephritis. <i>Journal of the American Society of Nephrology: JASN</i> , 2004, 15, 61-67.	6.1	166
22	Early Mechanisms of Renal Injury in Hypercholesterolemic or Hypertriglyceridemic Rats. <i>Journal of the American Society of Nephrology: JASN</i> , 2000, 11, 669-683.	6.1	159
23	Podocyte is the major culprit accounting for the progression of chronic renal disease. <i>Microscopy Research and Technique</i> , 2002, 57, 189-195.	2.2	151
24	The role of the podocyte in glomerulosclerosis. <i>Current Opinion in Nephrology and Hypertension</i> , 1999, 8, 489-497.	2.0	137
25	Glomerular damage after uninephrectomy in young rats. I. Hypertrophy and distortion of capillary architecture. <i>Kidney International</i> , 1992, 42, 136-147.	5.2	131
26	Renal epithelial injury and fibrosis. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2013, 1832, 931-939.	3.8	130
27	Podocyte Bridges between the Tuft and Bowman's Capsule. <i>Journal of the American Society of Nephrology: JASN</i> , 2001, 12, 2060-2071.	6.1	122
28	Age-related glomerulosclerosis and interstitial fibrosis in Milan normotensive rats: A podocyte disease. <i>Kidney International</i> , 1997, 51, 230-243.	5.2	117
29	Podocytes are sensitive to fluid shear stress in vitro. <i>American Journal of Physiology - Renal Physiology</i> , 2006, 291, F856-F865.	2.7	115
30	The LIM-homeodomain transcription factor Lmx1b plays a crucial role in podocytes. <i>Journal of Clinical Investigation</i> , 2002, 109, 1073-1082.	8.2	100
31	PTEN loss defines a TGF- β 2-induced tubule phenotype of failed differentiation and JNK signaling during renal fibrosis. <i>American Journal of Physiology - Renal Physiology</i> , 2012, 302, F1210-F1223.	2.7	99
32	Effects of Increased Renal Tubular Vascular Endothelial Growth Factor (VEGF) on Fibrosis, Cyst Formation, and Glomerular Disease. <i>American Journal of Pathology</i> , 2009, 175, 1883-1895.	3.8	96
33	Abrogation of Protein Uptake through Megalin-Deficient Proximal Tubules Does Not Safeguard against Tubulointerstitial Injury. <i>Journal of the American Society of Nephrology: JASN</i> , 2007, 18, 1824-1834.	6.1	87
34	Podocyte injury underlies the progression of focal segmental glomerulosclerosis in the fa/fa Zucker rat. <i>Kidney International</i> , 2001, 60, 106-116.	5.2	85
35	Analysis of differential gene expression in stretched podocytes: osteopontin enhances adaptation of podocytes to mechanical stress. <i>FASEB Journal</i> , 2002, 16, 1-24.	0.5	78
36	Tracer Studies in the Rat Demonstrate Misdirected Filtration and Peritubular Filtrate Spreading in Nephrons with Segmental Glomerulosclerosis. <i>Journal of the American Society of Nephrology: JASN</i> , 2001, 12, 496-506.	6.1	74

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37	Pathways to Recovery and Loss of Nephrons in Anti-Thy-1 Nephritis. <i>Journal of the American Society of Nephrology: JASN</i> , 2003, 14, 1904-1926.	6.1	73
38	Mechanical challenges to the glomerular filtration barrier: adaptations and pathway to sclerosis. <i>Pediatric Nephrology</i> , 2017, 32, 405-417.	1.7	61
39	SCG/Kinjoh mice: A model of ANCA-associated crescentic glomerulonephritis with immune deposits. <i>Kidney International</i> , 2003, 64, 140-148.	5.2	55
40	Altered gene expression and functions of mitochondria in human nephrotic syndrome. <i>FASEB Journal</i> , 1999, 13, 523-532.	0.5	53
41	The pathogenesis of 'classic' focal segmental glomerulosclerosis—lessons from rat models. <i>Nephrology Dialysis Transplantation</i> , 2003, 18, 39vi-44.	0.7	53
42	Potential relevance of shear stress for slit diaphragm and podocyte function. <i>Kidney International</i> , 2017, 91, 1283-1286.	5.2	48
43	Palladin is a dynamic actin-associated protein in podocytes. <i>Kidney International</i> , 2009, 75, 214-226.	5.2	47
44	Nonuniform Microtubular Polarity Established by CHO1/MKLP1 Motor Protein Is Necessary for Process Formation of Podocytes. <i>Journal of Cell Biology</i> , 1998, 143, 1961-1970.	5.2	45
45	Process formation of podocytes: morphogenetic activity of microtubules and regulation by protein serine/threonine phosphatase PP2A. <i>Histochemistry and Cell Biology</i> , 2001, 115, 255-266.	1.7	43
46	Structural Organization of the Mammalian Kidney. , 2013, , 595-691.		42
47	Structural Analysis of How Podocytes Detach from the Glomerular Basement Membrane Under Hypertrophic Stress. <i>Frontiers in Endocrinology</i> , 2014, 5, 207.	3.5	42
48	TRPC6 “ a new podocyte gene involved in focal segmental glomerulosclerosis. <i>Trends in Molecular Medicine</i> , 2005, 11, 527-530.	6.7	40
49	New insights into structural patterns encountered in glomerulosclerosis. <i>Current Opinion in Nephrology and Hypertension</i> , 2007, 16, 184-191.	2.0	38
50	Progression of chronic renal failure in focal segmental glomerulosclerosis: consequence of podocyte damage or of tubulointerstitial fibrosis?. <i>Pediatric Nephrology</i> , 2003, 18, 617-622.	1.7	33
51	CD2AP/CIN85 Balance Determines Receptor Tyrosine Kinase Signaling Response in Podocytes. <i>Journal of Biological Chemistry</i> , 2007, 282, 7457-7464.	3.4	33
52	Podocyte hypertrophy mismatch and glomerular disease. <i>Nature Reviews Nephrology</i> , 2012, 8, 618-619.	9.6	33
53	Pals1 Haploinsufficiency Results in Proteinuria and Cyst Formation. <i>Journal of the American Society of Nephrology: JASN</i> , 2017, 28, 2093-2107.	6.1	33
54	Accumulation of worn-out GBM material substantially contributes to mesangial matrix expansion in diabetic nephropathy. <i>American Journal of Physiology - Renal Physiology</i> , 2017, 312, F1101-F1111.	2.7	30

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55	The Contractile Apparatus of Podocytes Is Arranged to Counteract GBM Expansion. Contributions To Nephrology, 1994, 107, 1-9.	1.1	26
56	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
57	Structural Organization of the Mammalian Kidney. , 2008, , 479-563.		22
58	Changes in glomerular structure following acute mesangial failure in the isolated perfused kidney. Kidney International, 1992, 41, 533-541.	5.2	21
59	Adenosine and ATP: traffic regulators in the kidney. Journal of Clinical Investigation, 2004, 114, 611-613.	8.2	20
60	Ontogenetic Development of the Filtration Barrier. Nephron Experimental Nephrology, 2007, 106, e44-e50.	2.2	18
61	The Inability of Podocytes to Proliferate: Cause, Consequences, and Origin. Anatomical Record, 2020, 303, 2588-2596.	1.4	15
62	Maintenance and Breakdown of Glomerular Tuft Architecture. Journal of the American Society of Nephrology: JASN, 2018, 29, 1075-1077.	6.1	14
63	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
64	A new view of macula densa cell microanatomy. American Journal of Physiology - Renal Physiology, 2021, 320, F492-F504.	2.7	13
65	New aspects of podocyte structure, function, and pathology. Clinical and Experimental Nephrology, 1998, 2, 85-99.	1.6	11
66	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
67	Glomerular Injury in Analbuminemic Rats after Subtotal Nephrectomy. Nephron, 1991, 59, 104-109.	1.8	9
68	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
69	Pathomorphological sequence of nephron loss in diabetic nephropathy. American Journal of Physiology - Renal Physiology, 2021, 321, F600-F616.	2.7	9
70	Inverse correlation between vascular endothelial growth factor back-filtration and capillary filtration pressures. Nephrology Dialysis Transplantation, 2018, 33, 1514-1525.	0.7	7
71	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
72	Scanning electron microscopy studies of the vascular pole of the rat glomerulus. The Anatomical Record, 1982, 204, 149-152.	1.8	6

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73	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
74	Upregulation of Tumor Susceptibility Gene 101 (TSG101) by mechanical stress in podocytes. Cellular and Molecular Biology, 2019, 65, 84-88.	0.9	0