

Jochen Reiser

List of Publications by Year in descending order

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Version: 2024-02-01

82
papers

11,660
citations

66343

42
h-index

60623

81
g-index

161
all docs

161
docs citations

161
times ranked

12353
citing authors

#	ARTICLE	IF	CITATIONS
1	A Conditionally Immortalized Human Podocyte Cell Line Demonstrating Nephric and Podocin Expression. <i>Journal of the American Society of Nephrology: JASN</i> , 2002, 13, 630-638.	6.1	932
2	The actin cytoskeleton of kidney podocytes is a direct target of the antiproteinuric effect of cyclosporine A. <i>Nature Medicine</i> , 2008, 14, 931-938.	30.7	837
3	Rearrangements of the Cytoskeleton and Cell Contacts Induce Process Formation during Differentiation of Conditionally Immortalized Mouse Podocyte Cell Lines. <i>Experimental Cell Research</i> , 1997, 236, 248-258.	2.6	810
4	Circulating urokinase receptor as a cause of focal segmental glomerulosclerosis. <i>Nature Medicine</i> , 2011, 17, 952-960.	30.7	750
5	Factors Associated With Death in Critically Ill Patients With Coronavirus Disease 2019 in the US. <i>JAMA Internal Medicine</i> , 2020, 180, 1436.	5.1	711
6	Synaptopodin: An Actin-associated Protein in Telencephalic Dendrites and Renal Podocytes. <i>Journal of Cell Biology</i> , 1997, 139, 193-204.	5.2	526
7	Modification of kidney barrier function by the urokinase receptor. <i>Nature Medicine</i> , 2008, 14, 55-63.	30.7	501
8	Induction of B7-1 in podocytes is associated with nephrotic syndrome. <i>Journal of Clinical Investigation</i> , 2004, 113, 1390-1397.	8.2	495
9	Podocin, a raft-associated component of the glomerular slit diaphragm, interacts with CD2AP and nephrin. <i>Journal of Clinical Investigation</i> , 2001, 108, 1621-1629.	8.2	491
10	Specialized roles for cysteine cathepsins in health and disease. <i>Journal of Clinical Investigation</i> , 2010, 120, 3421-3431.	8.2	478
11	Association Between Early Treatment With Tocilizumab and Mortality Among Critically Ill Patients With COVID-19. <i>JAMA Internal Medicine</i> , 2021, 181, 41.	5.1	385
12	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
13	Soluble Urokinase Receptor and Chronic Kidney Disease. <i>New England Journal of Medicine</i> , 2015, 373, 1916-1925.	27.0	338
14	Podocytes Respond to Mechanical Stress In Vitro. <i>Journal of the American Society of Nephrology: JASN</i> , 2001, 12, 413-422.	6.1	252
15	Synaptopodin regulates the actin-bundling activity of β -actinin in an isoform-specific manner. <i>Journal of Clinical Investigation</i> , 2005, 115, 1188-1198.	8.2	249
16	Proteinuria: an enzymatic disease of the podocyte?. <i>Kidney International</i> , 2010, 77, 571-580.	5.2	232
17	Circulating suPAR in Two Cohorts of Primary FSGS. <i>Journal of the American Society of Nephrology: JASN</i> , 2012, 23, 2051-2059.	6.1	202
18	Synaptopodin regulates the actin-bundling activity of β -actinin in an isoform-specific manner. <i>Journal of Clinical Investigation</i> , 2005, 115, 1188-1198.	8.2	184

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19	A tripartite complex of suPAR, APOL1 risk variants and α 3 integrin on podocytes mediates chronic kidney disease. <i>Nature Medicine</i> , 2017, 23, 945-953.	30.7	176
20	A circulating antibody panel for pretransplant prediction of FSGS recurrence after kidney transplantation. <i>Science Translational Medicine</i> , 2014, 6, 256ra136.	12.4	172
21	Podocyte Biology and Pathogenesis of Kidney Disease. <i>Annual Review of Medicine</i> , 2013, 64, 357-366.	12.2	170
22	Podocyte Migration during Nephrotic Syndrome Requires a Coordinated Interplay between Cathepsin L and α 3 Integrin. <i>Journal of Biological Chemistry</i> , 2004, 279, 34827-34832.	3.4	155
23	Involvement of Lipid Rafts in Nephrin Phosphorylation and Organization of the Glomerular Slit Diaphragm. <i>American Journal of Pathology</i> , 2001, 159, 1069-1077.	3.8	142
24	Podocytes. <i>F1000Research</i> , 2016, 5, 114.	1.6	133
25	Defective podocyte insulin signalling through p85-XBP1 promotes ATF6-dependent maladaptive ER-stress response in diabetic nephropathy. <i>Nature Communications</i> , 2015, 6, 6496.	12.8	130
26	CD2AP in mouse and human podocytes controls a proteolytic program that regulates cytoskeletal structure and cellular survival. <i>Journal of Clinical Investigation</i> , 2011, 121, 3965-3980.	8.2	124
27	Bone marrow-derived immature myeloid cells are a main source of circulating suPAR contributing to proteinuric kidney disease. <i>Nature Medicine</i> , 2017, 23, 100-106.	30.7	121
28	Sphingomyelinase-Like Phosphodiesterase 3b Expression Levels Determine Podocyte Injury Phenotypes in Glomerular Disease. <i>Journal of the American Society of Nephrology: JASN</i> , 2015, 26, 133-147.	6.1	119
29	Role of Podocyte B7-1 in Diabetic Nephropathy. <i>Journal of the American Society of Nephrology: JASN</i> , 2014, 25, 1415-1429.	6.1	114
30	Prkdc participates in mitochondrial genome maintenance and prevents Adriamycin-induced nephropathy in mice. <i>Journal of Clinical Investigation</i> , 2010, 120, 4055-4064.	8.2	92
31	Human Immunodeficiency Virus-1 Induces Loss of Contact Inhibition in Podocytes. <i>Journal of the American Society of Nephrology: JASN</i> , 2001, 12, 1677-1684.	6.1	78
32	Danger Signaling by Glomerular Podocytes Defines a Novel Function of Inducible B7-1 in the Pathogenesis of Nephrotic Syndrome. <i>Journal of the American Society of Nephrology: JASN</i> , 2004, 15, 2246-2248.	6.1	71
33	Pathogenic Old World Hantaviruses Infect Renal Glomerular and Tubular Cells and Induce Disassembling of Cell-to-Cell Contacts. <i>Journal of Virology</i> , 2011, 85, 9811-9823.	3.4	68
34	Podocyte Effacement Closely Links to suPAR Levels at Time of Posttransplantation Focal Segmental Glomerulosclerosis Occurrence and Improves With Therapy. <i>Transplantation</i> , 2013, 96, 649-656.	1.0	58
35	A Podocyte-Based Automated Screening Assay Identifies Protective Small Molecules. <i>Journal of the American Society of Nephrology: JASN</i> , 2015, 26, 2741-2752.	6.1	53
36	Rituximab and Therapeutic Plasma Exchange in Recurrent Focal Segmental Glomerulosclerosis Postkidney Transplantation. <i>Transplantation</i> , 2018, 102, e115-e120.	1.0	50

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37	Transient Receptor Potential Channel 6 (TRPC6) Protects Podocytes during Complement-mediated Glomerular Disease. <i>Journal of Biological Chemistry</i> , 2013, 288, 36598-36609.	3.4	49
38	uPAR isoform 2 forms a dimer and induces severe kidney disease in mice. <i>Journal of Clinical Investigation</i> , 2019, 129, 1946-1959.	8.2	48
39	Synaptopodin Limits TRPC6 Podocyte Surface Expression and Attenuates Proteinuria. <i>Journal of the American Society of Nephrology: JASN</i> , 2016, 27, 3308-3319.	6.1	47
40	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
41	Podocyte injury-driven intracapillary plasminogen activator inhibitor type 1 accelerates podocyte loss via uPAR-mediated β 21-integrin endocytosis. <i>American Journal of Physiology - Renal Physiology</i> , 2015, 308, F614-F626.	2.7	45
42	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
43	Toward the development of podocyte-specific drugs. <i>Kidney International</i> , 2010, 77, 662-668.	5.2	42
44	Apoptosis and Compensatory Proliferation Signaling Are Coupled by Crkl-Containing Microvesicles. <i>Developmental Cell</i> , 2017, 41, 674-684.e5.	7.0	42
45	Recurrent Primary Focal Segmental Glomerulosclerosis Managed With Intensified Plasma Exchange and Concomitant Monitoring of Soluble Urokinase-Type Plasminogen Activator Receptor-Mediated Podocyte β 3-integrin Activation. <i>Transplantation</i> , 2015, 99, 2593-2597.	1.0	38
46	suPAR and chronic kidney disease—a podocyte story. <i>Pflügers Archiv European Journal of Physiology</i> , 2017, 469, 1017-1020.	2.8	36
47	Soluble urokinase receptor and focal segmental glomerulosclerosis. <i>Current Opinion in Nephrology and Hypertension</i> , 2012, 21, 428-432.	2.0	35
48	Phase I trial of donor-derived modified immune cell infusion in kidney transplantation. <i>Journal of Clinical Investigation</i> , 2020, 130, 2364-2376.	8.2	29
49	Signal integration at the PI3K-p85-XBP1 hub endows coagulation protease activated protein C with insulin-like function. <i>Blood</i> , 2017, 130, 1445-1455.	1.4	28
50	Soluble Urokinase Receptor and the Kidney Response in Diabetes Mellitus. <i>Journal of Diabetes Research</i> , 2017, 2017, 1-9.	2.3	28
51	Management of Severe Recurrent Focal Segmental Glomerulosclerosis Through Circulating Soluble Urokinase Receptor Modification. <i>American Journal of Therapeutics</i> , 2013, 20, 226-229.	0.9	26
52	IL-10 Dysregulation Underlies Chemokine Insufficiency, Delayed Macrophage Response, and Impaired Healing in Diabetic Wounds. <i>Journal of Investigative Dermatology</i> , 2022, 142, 692-704.e14.	0.7	22
53	Unwinding focal segmental glomerulosclerosis. <i>F1000Research</i> , 2017, 6, 466.	1.6	21
54	ACTH Gel in Resistant Focal Segmental Glomerulosclerosis After Kidney Transplantation. <i>Transplantation</i> , 2019, 103, 202-209.	1.0	21

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55	Cell Cycle Biomarkers and Soluble Urokinase-Type Plasminogen Activator Receptor for the Prediction of Sepsis-Induced Acute Kidney Injury Requiring Renal Replacement Therapy: A Prospective, Exploratory Study. <i>Critical Care Medicine</i> , 2019, 47, e999-e1007.	0.9	20
56	Nonimmune cell-derived ICOS ligand functions as a renoprotective α 23 integrin-selective antagonist. <i>Journal of Clinical Investigation</i> , 2019, 129, 1713-1726.	8.2	19
57	Predicting Mortality in African Americans With Type 2 Diabetes Mellitus: Soluble Urokinase Plasminogen Activator Receptor, Coronary Artery Calcium, and High-Sensitivity C-Reactive Protein. <i>Journal of the American Heart Association</i> , 2018, 7, .	3.7	18
58	Crkl/Abl phosphorylation cascade is critical for NLRC4 inflammasome activity and is blocked by <i>Pseudomonas aeruginosa</i> ExoT. <i>Nature Communications</i> , 2022, 13, 1295.	12.8	16
59	Virus- and cell type-specific effects in orthohantavirus infection. <i>Virus Research</i> , 2019, 260, 102-113.	2.2	14
60	Akt2 relaxes podocytes in chronic kidney disease. <i>Nature Medicine</i> , 2013, 19, 1212-1213.	30.7	13
61	Glomerular filtration barrier dysfunction in a self-limiting, RNA virus-induced glomerulopathy resembles findings in idiopathic nephrotic syndromes. <i>Scientific Reports</i> , 2020, 10, 19117.	3.3	13
62	High-content screening assay-based discovery of paullones as novel podocyte-protective agents. <i>American Journal of Physiology - Renal Physiology</i> , 2018, 314, F280-F292.	2.7	12
63	Soluble urokinase-type plasminogen activator receptor and incident end-stage renal disease in Chinese patients with chronic kidney disease. <i>Nephrology Dialysis Transplantation</i> , 2020, 35, 465-470.	0.7	12
64	Podocytes exhibit a specialized protein quality control employing derlin-2 in kidney disease. <i>American Journal of Physiology - Renal Physiology</i> , 2018, 314, F471-F482.	2.7	11
65	Is the LPS-mediated proteinuria mouse model relevant to human kidney disease?. <i>Nature Medicine</i> , 2009, 15, 133-134.	30.7	9
66	Rituximab: A Boot to Protect the Foot. <i>Journal of the American Society of Nephrology: JASN</i> , 2014, 25, 647-648.	6.1	8
67	Soluble urokinase plasminogen activation receptor and long-term outcomes in persons undergoing coronary angiography. <i>Scientific Reports</i> , 2019, 9, 475.	3.3	8
68	Bridges to cross, burn, and mend: cells of renin lineage as podocyte progenitors. <i>American Journal of Physiology - Renal Physiology</i> , 2015, 309, F499-F500.	2.7	6
69	New Insights into Diabetic Kidney Disease: The Potential Pathogenesis and Therapeutic Targets. <i>Journal of Diabetes Research</i> , 2017, 2017, 1-2.	2.3	5
70	Renal Dysfunction and Recovery following Initial Treatment of Newly Diagnosed Multiple Myeloma. <i>International Journal of Nephrology</i> , 2018, 2018, 1-6.	1.3	5
71	Renal cell markers: lighthouses for managing renal diseases. <i>American Journal of Physiology - Renal Physiology</i> , 2021, 321, F715-F739.	2.7	5
72	A High-Content Screening Technology for Quantitatively Studying Podocyte Dynamics. <i>Advances in Chronic Kidney Disease</i> , 2017, 24, 183-188.	1.4	4

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73	Stop that podocyte!. American Journal of Physiology - Renal Physiology, 2017, 312, F373-F374.	2.7	4
74	suPAR, a Circulating Kidney Disease Factor. Frontiers in Medicine, 2021, 8, 745838.	2.6	4
75	Characterization of a Trpc6 Transgenic Mouse Associated with Early Onset FSGS. British Journal of Medicine and Medical Research, 2015, 5, 1198-1212.	0.2	4
76	From Infancy to Fancy: A Glimpse into the Evolutionary Journey of Podocytes in Culture. Kidney360, 2021, 2, 385-397.	2.1	4
77	Therapeutic evaluation of immunomodulators in reducing surgical wound infection. FASEB Journal, 2022, 36, e22090.	0.5	4
78	The Grand Challenge of Nephrology. Frontiers in Medicine, 2014, 1, 28.	2.6	3
79	Extrarenal determinants of kidney filter function. Cell and Tissue Research, 2017, 369, 211-216.	2.9	3
80	Soluble Urokinase Receptor and Mortality in Kidney Transplant Recipients. Transplant International, 2021, 35, 10071.	1.6	2
81	Deiodinase-3 is a thyrostat to regulate podocyte homeostasis. EBioMedicine, 2021, 72, 103617.	6.1	1
82	SARS-CoV-2 pirates the kidneys: A scar(y) story. Cell Metabolism, 2022, 34, 352-354.	16.2	1