Lawrence B Holzman

List of Publications by Year
in descending order

110	10,381	28274	32842
papers	citations	55 h-index	100 g-index

114	114	114	9240
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Quantification of Glomerular Structural Lesions: Associations With Clinical Outcomes and Transcriptomic Profiles in Nephrotic Syndrome. American Journal of Kidney Diseases, 2022, 79, 807-819.e1.	1.9	13
2	Kidney Biopsy Features Most Predictive of Clinical Outcomes in the Spectrum of Minimal Change Disease and Focal Segmental Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2022, 33, 1411-1426.	6.1	16
3	APOL1 genotype-associated morphologic changes among patients with focal segmental glomerulosclerosis. Pediatric Nephrology, 2021, 36, 2747-2757.	1.7	3
4	Phosphorylation of slit diaphragm proteins NEPHRIN and NEPH1 upon binding of HGF promotes podocyte repair. Journal of Biological Chemistry, 2021, 297, 101079.	3.4	4
5	Longitudinal Changes in Health-Related Quality of Life in Primary Glomerular Disease: Results From the CureGN Study. Kidney International Reports, 2020, 5, 1679-1689.	0.8	17
6	SHROOM3, the gene associated with chronic kidney disease, affects the podocyte structure. Scientific Reports, 2020, 10, 21103.	3.3	11
7	The longitudinal relationship between patient-reported outcomes and clinical characteristics among patients with focal segmental glomerulosclerosis in the Nephrotic Syndrome Study Network. CKJ: Clinical Kidney Journal, 2020, 13, 597-606.	2.9	14
8	Persistent Disease Activity in Patients With Long-Standing Glomerular Disease. Kidney International Reports, 2020, 5, 860-871.	0.8	2
9	Ultrastructural Characterization of Proteinuric Patients Predicts Clinical Outcomes. Journal of the American Society of Nephrology: JASN, 2020, 31, 841-854.	6.1	29
10	Health-related quality of life in glomerular disease. Kidney International, 2019, 95, 1209-1224.	5.2	38
11	The motor protein Myo1c regulates transforming growth factor-Î²â€“signaling and fibrosis in podocytes. Kidney International, 2019, 96, 139-158.	5.2	20
12	CureGN Study Rationale, Design, and Methods: Establishing a Large Prospective Observational Study of Glomerular Disease. American Journal of Kidney Diseases, 2019, 73, 218-229.	1.9	68
13	Reproducibility and Feasibility of Strategies for Morphologic Assessment of Renal Biopsies Using the Nephrotic Syndrome Study Network Digital Pathology Scoring System. Archives of Pathology and Laboratory Medicine, 2018, 142, 613-625.	2.5	21
14	Randomized Clinical Trial Design to Assess Abatacept in Resistant Nephrotic Syndrome. Kidney International Reports, 2018, 3, 115-121.	0.8	21
15	Clinical Characteristics and Treatment Patterns of Children and Adults With IgA Nephropathy or IgA Vasculitis: Findings From the CureGN Study. Kidney International Reports, 2018, 3, 1373-1384.	0.8	39
16	Digital pathology imaging as a novel platform for standardization and globalization of quantitative nephropathology. CKJ: Clinical Kidney Journal, 2017, 10, 176-187.	2.9	45
17	ARF6 mediates nephrin tyrosine phosphorylation-induced podocyte cellular dynamics. PLoS ONE, 2017, 12, e0184575.	2.5	8
18	An evolutionarily conserved mechanism for cAMP elicited axonal regeneration involves direct activation of the dual leucine zipper kinase DLK. ELife, 2016, 5, .	6.0	59

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19	Leucine Zipper-bearing Kinase promotes axon growth in mammalian central nervous system neurons. Scientific Reports, 2016, 6, 31482.	3.3	32
20	Structural Analysis of the Myo1c and Neph1 Complex Provides Insight into the Intracellular Movement of Neph1. Molecular and Cellular Biology, 2016, 36, 1639-1654.	2.3	34
21	Reproducibility of the NEPTUNE descriptor-based scoring system on whole-slide images and histologic and ultrastructural digital images. Modern Pathology, 2016, 29, 671-684.	5.5	56
22	Glomerular Diseases: Registries and Clinical Trials. Clinical Journal of the American Society of Nephrology: CJASN, 2016, 11, 2234-2243.	4.5	11
23	FAT1 mutations cause a glomerulotubular nephropathy. Nature Communications, 2016, 7, 10822.	12.8	99
24	Complete Remission in the Nephrotic Syndrome Study Network. Clinical Journal of the American Society of Nephrology: CJASN, 2016, 11, 81-89.	4.5	53
25	Nephrin Preserves Podocyte Viability and Glomerular Structure and Function in Adult Kidneys. Journal of the American Society of Nephrology: JASN, 2015, 26, 2361-2377.	6.1	93
26	Ret is critical for podocyte survival following glomerular injury in vivo. American Journal of Physiology - Renal Physiology, 2015, 308, F774-F783.	2.7	2
27	A reassessment of soluble urokinase-type plasminogen activator receptor in glomerular disease. Kidney International, 2015, 87, 564-574.	5.2	111
28	Podocyte-associated talin1 is critical for glomerular filtration barrier maintenance. Journal of Clinical Investigation, 2015, 125, 882-882.	8.2	0
29	Podocyte-specific deletion of NDST1, a key enzyme in the sulfation of heparan sulfate glycosaminoglycans, leads to abnormalities in podocyte organization in vivo. Kidney International, 2014, 85, 307-318.	5.2	19
30	The Kidney Research National Dialogue. Clinical Journal of the American Society of Nephrology: CJASN, 2014, 9, 1806-1811.	4.5	18
31	Crk1/2 and CrkL form a hetero-oligomer and functionally complement each other during podocyte morphogenesis. Kidney International, 2014, 85, 1382-1394.	5.2	37
32	Glomerular Disease. Clinical Journal of the American Society of Nephrology: CJASN, 2014, 9, 1138-1140.	4.5	14
33	Slit Diaphragm Protein Neph1 and Its Signaling. Journal of Biological Chemistry, 2014, 289, 9502-9518.	3.4	39
34	Podocyte-associated talin1 is critical for glomerular filtration barrier maintenance. Journal of Clinical Investigation, 2014, 124, 1098-1113.	8.2	122
35	Divergent functions of the Rho GTPases Rac1 and Cdc42 in podocyte injury. Kidney International, 2013, 84, 920-930.	5.2	125
36	Myo1c is an unconventional myosin required for zebrafish glomerular development. Kidney International, 2013, 84, 1154-1165.	5.2	14

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37	Design of the Nephrotic Syndrome Study Network (NEPTUNE) to evaluate primary glomerular nephropathy by a multidisciplinary approach. Kidney International, 2013, 83, 749-756.	5.2	268
38	Background Strain and the Differential Susceptibility of Podocyte-Specific Deletion of Myh9 on Murine Models of Experimental Glomerulosclerosis and HIV Nephropathy. PLoS ONE, 2013, 8, e67839.	2.5	25
39	Podocyte-specific knockout of myosin 1e disrupts glomerular filtration. American Journal of Physiology - Renal Physiology, 2012, 303, F1099-F1106.	2.7	39
40	Solution Structure Analysis of Cytoplasmic Domain of Podocyte Protein Neph1 Using Small/Wide Angle X-ray Scattering (SWAXS). Journal of Biological Chemistry, 2012, 287, 9441-9453.	3.4	13
41	Role of dynamin, synaptojanin, and endophilin in podocyte foot processes. Journal of Clinical Investigation, 2012, 122, 4401-4411.	8.2	137
42	Signaling From the Podocyte Intercellular Junction to the Actin Cytoskeleton. Seminars in Nephrology, 2012, 32, 307-318.	1.6	42
43	Inhibitory Effects of Robo2 on Nephrin: A Crosstalk between Positive and Negative Signals Regulating Podocyte Structure. Cell Reports, 2012, 2, 52-61.	6.4	53
44	Podocytes: Gaining a foothold. Experimental Cell Research, 2012, 318, 955-963.	2.6	34
45	Crk1/2-dependent signaling is necessary for podocyte foot process spreading in mouse models of glomerular disease. Journal of Clinical Investigation, 2012, 122, 674-692.	8.2	92
46	APOL1 Null Alleles from a Rural Village in India Do Not Correlate with Glomerulosclerosis. PLoS ONE, 2012, 7, e51546.	2.5	70
47	Lack of Nâ€Sulfation of Podocyte Cell Surface Heparan Sulfate Glycosaminoglycans Leads to Abnormalities in Podocyte Organization, Adhesion, and Migration. FASEB Journal, 2012, 26, 906.1.	0.5	0
48	mTORC1 activation in podocytes is a critical step in the development of diabetic nephropathy in mice. Journal of Clinical Investigation, 2011, 121, 2181-2196.	8.2	462
49	Vascular Endothelial Growth Factor Receptor 2 Direct Interaction with Nephrin Links VEGF-A Signals to Actin in Kidney Podocytes. Journal of Biological Chemistry, 2011, 286, 39933-39944.	3.4	58
50	Wnt/Î²-Catenin Pathway in Podocytes Integrates Cell Adhesion, Differentiation, and Survival. Journal of Biological Chemistry, 2011, 286, 26003-26015.	3.4	166
51	Podocyte-Specific Deletion of Myh9 Encoding Nonmuscle Myosin Heavy Chain 2A Predisposes Mice to Glomerulopathy. Molecular and Cellular Biology, 2011, 31, 2162-2170.	2.3	74
52	The inducible deletion of Drosha and microRNAs in mature podocytes results in a collapsing glomerulopathy. Kidney International, 2011, 80, 719-730.	5.2	105
53	Inhibition of Podocyte FAK Protects against Proteinuria and Foot Process Effacement. Journal of the American Society of Nephrology: JASN, 2010, 21, 1145-1156.	6.1	107
54	Actin-depolymerizing Factor Cofilin-1 Is Necessary in Maintaining Mature Podocyte Architecture. Journal of Biological Chemistry, 2010, 285, 22676-22688.	3.4	97

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55	Hepatocyte growth factor signaling ameliorates podocyte injury and proteinuria. Kidney International, 2010, 77, 962-973.	5.2	87
56	Podocytes require the engagement of cell surface heparan sulfate proteoglycans for adhesion to extracellular matrices. Kidney International, 2010, 78, 1088-1099.	5.2	23
57	Deletion of Von Hippel-Lindau in Glomerular Podocytes Results in Glomerular Basement Membrane Thickening, Ectopic Subepithelial Deposition of Collagen Î±1Î±2Î±1(IV), Expression of Neuroglobin, and Proteinuria. American Journal of Pathology, 2010, 177, 84-96.	3.8	30
58	Podocyte-specific overexpression of GLUT1 surprisingly reduces mesangial matrix expansion in diabetic nephropathy in mice. American Journal of Physiology - Renal Physiology, 2010, 299, F91-F98.	2.7	43
59	Initial Insight on the Determinants of Podocyte Polarity. Journal of the American Society of Nephrology: JASN, 2009, 20, 683-685.	6.1	1
60	Wnt/Î²-Catenin Signaling Promotes Podocyte Dysfunction and Albuminuria. Journal of the American Society of Nephrology: JASN, 2009, 20, 1997-2008.	6.1	356
61	Loss of heparan sulfate glycosaminoglycan assembly in podocytes does not lead to proteinuria. Kidney International, 2008, 74, 289-299.	5.2	83
62	Î²1 integrin expression by podocytes is required to maintain glomerular structural integrity. Developmental Biology, 2008, 316, 288-301.	2.0	161
63	A Mutation in the Mouse Chd2 Chromatin Remodeling Enzyme Results in a Complex Renal Phenotype. Kidney and Blood Pressure Research, 2008, 31, 421-432.	2.0	25
64	Ablation of developing podocytes disrupts cellular interactions and nephrogenesis both inside and outside the glomerulus. American Journal of Physiology - Renal Physiology, 2008, 295, F1790-F1798.	2.7	9
65	Podocyte-Selective Deletion of Dicer Induces Proteinuria and Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2008, 19, 2159-2169.	6.1	332
66	Ischemic Injury to Kidney Induces Glomerular Podocyte Effacement and Dissociation of Slit Diaphragm Proteins Neph1 and ZO-1. Journal of Biological Chemistry, 2008, 283, 35579-35589.	3.4	80
67	Neph1 Cooperates with Nephrin To Transduce a Signal That Induces Actin Polymerization. Molecular and Cellular Biology, 2007, 27, 8698-8712.	2.3	130
68	Identification of the Glomerular Podocyte as a Target for Growth Hormone Action. Endocrinology, 2007, 148, 2045-2055.	2.8	47
69	Differentially Spliced Isoforms of FAT1 Are Asymmetrically Distributed within Migrating Cells. Journal of Biological Chemistry, 2007, 282, 22823-22833.	3.4	29
70	Slit Diaphragm Junctional Complex and Regulation of the Cytoskeleton. Nephron Experimental Nephrology, 2007, 106, e67-e72.	2.2	29
71	Src Family Kinases Directly Regulate JIP1 Module Dynamics and Activation. Molecular and Cellular Biology, 2007, 27, 2431-2441.	2.3	27
72	The podocyte-specific inactivation of Lmx1b, Ldb1 and E2a yields new insight into a transcriptional network in podocytes. Developmental Biology, 2007, 304, 701-712.	2.0	60

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73	Disruption of Glomerular Basement Membrane Charge through Podocyte-Specific Mutation of Agrin Does Not Alter Glomerular Permselectivity. American Journal of Pathology, 2007, 171, 139-152.	3.8	153
74	Podocyteâ€specific Vhlh loss demonstrates role for hypoxiaâ€inducible transcription factors (HIFs) in glomerular disease pathogenesis. FASEB Journal, 2007, 21, A504.	0.5	0
75	Clinical impact of research on the podocyte slit diaphragm. Nature Clinical Practice Nephrology, 2006, 2, 271-282.	2.0	81
76	Positional cloning uncovers mutations in PLCE1 responsible for a nephrotic syndrome variant that may be reversible. Nature Genetics, 2006, 38, 1397-1405.	21.4	510
77	Podocyte-Specific Deletion of Integrin-Linked Kinase Results in Severe Glomerular Basement Membrane Alterations and Progressive Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2006, 17, 1334-1344.	6.1	137
78	Imaging Podocyte Dynamics. Nephron Experimental Nephrology, 2006, 103, e69-e74.	2.2	12
79	Nephrin ectodomain engagement results in Src kinase activation, nephrin phosphorylation, Nck recruitment, and actin polymerization. Journal of Clinical Investigation, 2006, 116, 1346-1359.	8.2	282
80	An efficient system for tissue-specific overexpression of transgenes in podocytes in vivo. American Journal of Physiology - Renal Physiology, 2005, 289, F481-F488.	2.7	12
81	Podocyte Depletion Causes Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2005, 16, 2941-2952.	6.1	649
82	Glomerular Disease Workshop. Journal of the American Society of Nephrology: JASN, 2005, 16, 3472-3476.	6.1	6
83	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
84	Stable expression of nephrin and localization to cell-cell contacts in novel murine podocyte cell lines. Kidney International, 2004, 66, 91-101.	5.2	125
85	Protocadherin FAT1 binds Ena/VASP proteins and is necessary for actin dynamics and cell polarization. EMBO Journal, 2004, 23, 3769-3779.	7.8	168
86	Podocyteâ€specific expression of cre recombinase in transgenic mice. Genesis, 2003, 35, 39-42.	1.6	275
87	Nephrin and Neph1 Co-localize at the Podocyte Foot Process Intercellular Junction and Form cis Hetero-oligomers. Journal of Biological Chemistry, 2003, 278, 19266-19271.	3.4	157
88	Recruitment of JNK to JIP1 and JNK-dependent JIP1 Phosphorylation Regulates JNK Module Dynamics and Activation. Journal of Biological Chemistry, 2003, 278, 28694-28702.	3.4	70
89	Fyn Binds to and Phosphorylates the Kidney Slit Diaphragm Component Nephrin. Journal of Biological Chemistry, 2003, 278, 20716-20723.	3.4	209
90	Inducible Podocyte-Specific Gene Expression in Transgenic Mice. Journal of the American Society of Nephrology: JASN, 2003, 14, 1998-2003.	6.1	76

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91	Phosphorylation of Pax2 by the c-Jun N-terminal Kinase and Enhanced Pax2-dependent Transcription Activation. Journal of Biological Chemistry, 2002, 277, 1217-1222.	3.4	75
92	Two Gene Fragments that Direct Podocyte-Specific Expression in Transgenic Mice. Journal of the American Society of Nephrology: JASN, 2002, 13, 1561-1567.	6.1	96
93	Podocyte depletion and glomerulosclerosis have a direct relationship in the PAN-treated rat. Kidney International, 2001, 60, 957-968.	5.2	340
94	Podocin, a raft-associated component of the glomerular slit diaphragm, interacts with CD2AP and nephrin. Journal of Clinical Investigation, 2001, 108, 1621-1629.	8.2	491
95	GLUT-1 reduces hypoxia-induced apoptosis and JNK pathway activation. American Journal of Physiology - Endocrinology and Metabolism, 2000, 278, E958-E966.	3.5	65
96	Identification of Structural and Functional Domains in Mixed Lineage Kinase Dual Leucine Zipper-bearing Kinase Required for Complex Formation and Stress-activated Protein Kinase Activation. Journal of Biological Chemistry, 2000, 275, 7273-7279.	3.4	64
97	Caveolar Structure and Protein Sorting Are Maintained in NIH 3T3 Cells Independent of Glycosphingolipid Depletion. Archives of Biochemistry and Biophysics, 2000, 373, 83-90.	3.0	28
98	Altered podocyte structure in GLEPP1 (Ptpro)-deficient mice associated with hypertension and low glomerular filtration rate. Journal of Clinical Investigation, 2000, 106, 1281-1290.	8.2	135
99	Evaluation of a New Tool for Exploring Podocyte Biology. Journal of the American Society of Nephrology: JASN, 2000, 11, 2306-2314.	6.1	66
100	Nephritogenic mAb 5-1-6 is directed at the extracellular domain of rat nephrin. Journal of Clinical Investigation, 2000, 105, 125-125.	8.2	0
101	Requirement for Ras/Rac1-Mediated p38 and c-Jun N-Terminal Kinase Signaling in Stat3 Transcriptional Activity Induced by the Src Oncoprotein. Molecular and Cellular Biology, 1999, 19, 7519-7528.	2.3	239
102	The Mixed Lineage Kinase DLK Utilizes MKK7 and Not MKK4 as Substrate. Journal of Biological Chemistry, 1999, 274, 10195-10202.	3.4	92
103	Re-expression of the developmental gene Pax-2 during experimental acute tubular necrosis in mice1. Kidney International, 1999, 56, 1423-1431.	5.2	176
104	Nephrin localizes to the slit pore of the glomerular epithelial cell. Kidney International, 1999, 56, 1481-1491.	5.2	268
105	Cloning and Expression of the Rat Nephrin Homolog. American Journal of Pathology, 1999, 155, 907-913.	3.8	61
106	Nephritogenic mAb 5-1-6 is directed at the extracellular domain of rat nephrin. Journal of Clinical Investigation, 1999, 104, 1559-1566.	8.2	154
107	Post-translational Processing and Renal Expression of Mouse Indian Hedgehog. Journal of Biological Chemistry, 1997, 272, 8466-8473.	3.4	26
108	Characterization of Dual Leucine Zipper-bearing Kinase, a Mixed Lineage Kinase Present in Synaptic Terminals Whose Phosphorylation State Is Regulated by Membrane Depolarization via Calcineurin. Journal of Biological Chemistry, 1996, 271, 16888-16896.	3.4	69

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109	Dual Leucine Zipper-bearing Kinase (DLK) Activates p46SAPK and p38 but Not ERK2. Journal of Biological Chemistry, 1996, 271, 24788-24793.	3.4	124
110	SA Gene Expression in the Proximal Tubule of Normotensive and Hypertensive Rats. Hypertension, 1996, 27, 541-545.	2.7	18