Lawrence B Holzman

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Podocyte Depletion Causes Glomerulosclerosis: Diphtheria Toxin–Induced Podocyte Depletion in Rats Expressing Human Diphtheria Toxin Receptor Transgene. Journal of the American Society of Nephrology: JASN, 2005, 16, 2941-2952.	3.0	649
2	Positional cloning uncovers mutations in PLCE1 responsible for a nephrotic syndrome variant that may be reversible. Nature Genetics, 2006, 38, 1397-1405.	9.4	510
3	Podocin, a raft-associated component of the glomerular slit diaphragm, interacts with CD2AP and nephrin. Journal of Clinical Investigation, 2001, 108, 1621-1629.	3.9	491
4	mTORC1 activation in podocytes is a critical step in the development of diabetic nephropathy in mice. Journal of Clinical Investigation, 2011, 121, 2181-2196.	3.9	462
5	Wnt∫β-Catenin Signaling Promotes Podocyte Dysfunction and Albuminuria. Journal of the American Society of Nephrology: JASN, 2009, 20, 1997-2008.	3.0	356
6	Podocyte depletion and glomerulosclerosis have a direct relationship in the PAN-treated rat. Kidney International, 2001, 60, 957-968.	2.6	340
7	Podocyte-Selective Deletion of Dicer Induces Proteinuria and Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2008, 19, 2159-2169.	3.0	332
8	Nephrin ectodomain engagement results in Src kinase activation, nephrin phosphorylation, Nck recruitment, and actin polymerization. Journal of Clinical Investigation, 2006, 116, 1346-1359.	3.9	282
9	Podocyte-specific expression of cre recombinase in transgenic mice. Genesis, 2003, 35, 39-42.	0.8	275
10	Nephrin localizes to the slit pore of the glomerular epithelial cell. Kidney International, 1999, 56, 1481-1491.	2.6	268
11	Design of the Nephrotic Syndrome Study Network (NEPTUNE) to evaluate primary glomerular nephropathy by a multidisciplinary approach. Kidney International, 2013, 83, 749-756.	2.6	268
12	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.	1.1	239
13	Fyn Binds to and Phosphorylates the Kidney Slit Diaphragm Component Nephrin. Journal of Biological Chemistry, 2003, 278, 20716-20723.	1.6	209
14	Re-expression of the developmental gene Pax-2 during experimental acute tubular necrosis in mice1. Kidney International, 1999, 56, 1423-1431.	2.6	176
15	Protocadherin FAT1 binds Ena/VASP proteins and is necessary for actin dynamics and cell polarization. EMBO Journal, 2004, 23, 3769-3779.	3.5	168
16	Podocytes Populate Cellular Crescents in a Murine Model of Inflammatory Glomerulonephritis. Journal of the American Society of Nephrology: JASN, 2004, 15, 61-67.	3.0	166
17	Wnt/β-Catenin Pathway in Podocytes Integrates Cell Adhesion, Differentiation, and Survival. Journal of Biological Chemistry, 2011, 286, 26003-26015.	1.6	166
18	β1 integrin expression by podocytes is required to maintain glomerular structural integrity. Developmental Biology, 2008, 316, 288-301.	0.9	161

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19	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.	1.6	157
20	Nephritogenic mAb 5-1-6 is directed at the extracellular domain of rat nephrin. Journal of Clinical Investigation, 1999, 104, 1559-1566.	3.9	154
21	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.	1.9	153
22	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.	3.0	137
23	Role of dynamin, synaptojanin, and endophilin in podocyte foot processes. Journal of Clinical Investigation, 2012, 122, 4401-4411.	3.9	137
24	Altered podocyte structure in GLEPP1 (Ptpro)-deficient mice associated with hypertension and low glomerular filtration rate. Journal of Clinical Investigation, 2000, 106, 1281-1290.	3.9	135
25	Neph1 Cooperates with Nephrin To Transduce a Signal That Induces Actin Polymerization. Molecular and Cellular Biology, 2007, 27, 8698-8712.	1.1	130
26	Stable expression of nephrin and localization to cell-cell contacts in novel murine podocyte cell lines. Kidney International, 2004, 66, 91-101.	2.6	125
27	Divergent functions of the Rho GTPases Rac1 and Cdc42 in podocyte injury. Kidney International, 2013, 84, 920-930.	2.6	125
28	Dual Leucine Zipper-bearing Kinase (DLK) Activates p46SAPK and p38 but Not ERK2. Journal of Biological Chemistry, 1996, 271, 24788-24793.	1.6	124
29	Podocyte-associated talin1 is critical for glomerular filtration barrier maintenance. Journal of Clinical Investigation, 2014, 124, 1098-1113.	3.9	122
30	A reassessment of soluble urokinase-type plasminogen activator receptor in glomerular disease. Kidney International, 2015, 87, 564-574.	2.6	111
31	Inhibition of Podocyte FAK Protects against Proteinuria and Foot Process Effacement. Journal of the American Society of Nephrology: JASN, 2010, 21, 1145-1156.	3.0	107
32	The inducible deletion of Drosha and microRNAs in mature podocytes results in a collapsing glomerulopathy. Kidney International, 2011, 80, 719-730.	2.6	105
33	FAT1 mutations cause a glomerulotubular nephropathy. Nature Communications, 2016, 7, 10822.	5.8	99
34	Actin-depolymerizing Factor Cofilin-1 Is Necessary in Maintaining Mature Podocyte Architecture. Journal of Biological Chemistry, 2010, 285, 22676-22688.	1.6	97
35	Two Gene Fragments that Direct Podocyte-Specific Expression in Transgenic Mice. Journal of the American Society of Nephrology: JASN, 2002, 13, 1561-1567.	3.0	96
36	Nephrin Preserves Podocyte Viability and Glomerular Structure and Function in Adult Kidneys. Journal of the American Society of Nephrology: JASN, 2015, 26, 2361-2377.	3.0	93

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37	The Mixed Lineage Kinase DLK Utilizes MKK7 and Not MKK4 as Substrate. Journal of Biological Chemistry, 1999, 274, 10195-10202.	1.6	92
38	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.	3.9	92
39	Hepatocyte growth factor signaling ameliorates podocyte injury and proteinuria. Kidney International, 2010, 77, 962-973.	2.6	87
40	Loss of heparan sulfate glycosaminoglycan assembly in podocytes does not lead to proteinuria. Kidney International, 2008, 74, 289-299.	2.6	83
41	Clinical impact of research on the podocyte slit diaphragm. Nature Clinical Practice Nephrology, 2006, 2, 271-282.	2.0	81
42	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.	1.6	80
43	Inducible Podocyte-Specific Gene Expression in Transgenic Mice. Journal of the American Society of Nephrology: JASN, 2003, 14, 1998-2003.	3.0	76
44	Phosphorylation of Pax2 by the c-Jun N-terminal Kinase and Enhanced Pax2-dependent Transcription Activation. Journal of Biological Chemistry, 2002, 277, 1217-1222.	1.6	75
45	Podocyte-Specific Deletion of Myh9 Encoding Nonmuscle Myosin Heavy Chain 2A Predisposes Mice to Glomerulopathy. Molecular and Cellular Biology, 2011, 31, 2162-2170.	1.1	74
46	Recruitment of JNK to JIP1 and JNK-dependent JIP1 Phosphorylation Regulates JNK Module Dynamics and Activation. Journal of Biological Chemistry, 2003, 278, 28694-28702.	1.6	70
47	APOL1 Null Alleles from a Rural Village in India Do Not Correlate with Glomerulosclerosis. PLoS ONE, 2012, 7, e51546.	1.1	70
48	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.	1.6	69
49	CureGN Study Rationale, Design, and Methods: Establishing a Large Prospective Observational Study of Glomerular Disease. American Journal of Kidney Diseases, 2019, 73, 218-229.	2.1	68
50	Evaluation of a New Tool for Exploring Podocyte Biology. Journal of the American Society of Nephrology: JASN, 2000, 11, 2306-2314.	3.0	66
51	GLUT-1 reduces hypoxia-induced apoptosis and JNK pathway activation. American Journal of Physiology - Endocrinology and Metabolism, 2000, 278, E958-E966.	1.8	65
52	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.	1.6	64
53	Cloning and Expression of the Rat Nephrin Homolog. American Journal of Pathology, 1999, 155, 907-913.	1.9	61
54	The podocyte-specific inactivation of Lmx1b, Ldb1 and E2a yields new insight into a transcriptional network in podocytes. Developmental Biology, 2007, 304, 701-712.	0.9	60

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55	An evolutionarily conserved mechanism for cAMP elicited axonal regeneration involves direct activation of the dual leucine zipper kinase DLK. ELife, 2016, 5, .	2.8	59
56	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.	1.6	58
57	Reproducibility of the NEPTUNE descriptor-based scoring system on whole-slide images and histologic and ultrastructural digital images. Modern Pathology, 2016, 29, 671-684.	2.9	56
58	Inhibitory Effects of Robo2 on Nephrin: A Crosstalk between Positive and Negative Signals Regulating Podocyte Structure. Cell Reports, 2012, 2, 52-61.	2.9	53
59	Complete Remission in the Nephrotic Syndrome Study Network. Clinical Journal of the American Society of Nephrology: CJASN, 2016, 11, 81-89.	2.2	53
60	Identification of the Glomerular Podocyte as a Target for Growth Hormone Action. Endocrinology, 2007, 148, 2045-2055.	1.4	47
61	Digital pathology imaging as a novel platform for standardization and globalization of quantitative nephropathology. CKJ: Clinical Kidney Journal, 2017, 10, 176-187.	1.4	45
62	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.	1.3	43
63	Signaling From the Podocyte Intercellular Junction to the Actin Cytoskeleton. Seminars in Nephrology, 2012, 32, 307-318.	0.6	42
64	Podocyte-specific knockout of myosin 1e disrupts glomerular filtration. American Journal of Physiology - Renal Physiology, 2012, 303, F1099-F1106.	1.3	39
65	Slit Diaphragm Protein Neph1 and Its Signaling. Journal of Biological Chemistry, 2014, 289, 9502-9518.	1.6	39
66	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.	0.4	39
67	Health-related quality of life in glomerular disease. Kidney International, 2019, 95, 1209-1224.	2.6	38
68	Crk1/2 and CrkL form a hetero-oligomer and functionally complement each other during podocyte morphogenesis. Kidney International, 2014, 85, 1382-1394.	2.6	37
69	Podocytes: Gaining a foothold. Experimental Cell Research, 2012, 318, 955-963.	1.2	34
70	Structural Analysis of the Myo1c and Neph1 Complex Provides Insight into the Intracellular Movement of Neph1. Molecular and Cellular Biology, 2016, 36, 1639-1654.	1.1	34
71	Leucine Zipper-bearing Kinase promotes axon growth in mammalian central nervous system neurons. Scientific Reports, 2016, 6, 31482.	1.6	32
72	Deletion of Von Hippel-Lindau in Glomerular Podocytes Results in Glomerular Basement Membrane Thickening, Ectopic Subepithelial Deposition of Collagen α1α2α1(IV), Expression of Neuroglobin, and Proteinuria. American Journal of Pathology, 2010, 177, 84-96.	1.9	30

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73	Differentially Spliced Isoforms of FAT1 Are Asymmetrically Distributed within Migrating Cells. Journal of Biological Chemistry, 2007, 282, 22823-22833.	1.6	29
74	Slit Diaphragm Junctional Complex and Regulation of the Cytoskeleton. Nephron Experimental Nephrology, 2007, 106, e67-e72.	2.4	29
75	Ultrastructural Characterization of Proteinuric Patients Predicts Clinical Outcomes. Journal of the American Society of Nephrology: JASN, 2020, 31, 841-854.	3.0	29
76	Caveolar Structure and Protein Sorting Are Maintained in NIH 3T3 Cells Independent of Glycosphingolipid Depletion. Archives of Biochemistry and Biophysics, 2000, 373, 83-90.	1.4	28
77	Src Family Kinases Directly Regulate JIP1 Module Dynamics and Activation. Molecular and Cellular Biology, 2007, 27, 2431-2441.	1.1	27
78	Post-translational Processing and Renal Expression of Mouse Indian Hedgehog. Journal of Biological Chemistry, 1997, 272, 8466-8473.	1.6	26
79	A Mutation in the Mouse Chd2 Chromatin Remodeling Enzyme Results in a Complex Renal Phenotype. Kidney and Blood Pressure Research, 2008, 31, 421-432.	0.9	25
80	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.	1.1	25
81	Podocytes require the engagement of cell surface heparan sulfate proteoglycans for adhesion to extracellular matrices. Kidney International, 2010, 78, 1088-1099.	2.6	23
82	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.	1.2	21
83	Randomized Clinical Trial Design to Assess Abatacept in Resistant Nephrotic Syndrome. Kidney International Reports, 2018, 3, 115-121.	0.4	21
84	The motor protein Myo1c regulates transforming growth factor-β–signaling and fibrosis in podocytes. Kidney International, 2019, 96, 139-158.	2.6	20
85	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.	2.6	19
86	The Kidney Research National Dialogue. Clinical Journal of the American Society of Nephrology: CJASN, 2014, 9, 1806-1811.	2.2	18
87	SA Gene Expression in the Proximal Tubule of Normotensive and Hypertensive Rats. Hypertension, 1996, 27, 541-545.	1.3	18
88	Longitudinal Changes in Health-Related Quality of Life in Primary Glomerular Disease: Results From the CureGN Study. Kidney International Reports, 2020, 5, 1679-1689.	0.4	17
89	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.	3.0	16
90	Myo1c is an unconventional myosin required for zebrafish glomerular development. Kidney International, 2013, 84, 1154-1165.	2.6	14

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91	Glomerular Disease. Clinical Journal of the American Society of Nephrology: CJASN, 2014, 9, 1138-1140.	2.2	14
92	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.	1.4	14
93	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.	1.6	13
94	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.	2.1	13
95	An efficient system for tissue-specific overexpression of transgenes in podocytes in vivo. American Journal of Physiology - Renal Physiology, 2005, 289, F481-F488.	1.3	12
96	Imaging Podocyte Dynamics. Nephron Experimental Nephrology, 2006, 103, e69-e74.	2.4	12
97	Glomerular Diseases: Registries and Clinical Trials. Clinical Journal of the American Society of Nephrology: CJASN, 2016, 11, 2234-2243.	2.2	11
98	SHROOM3, the gene associated with chronic kidney disease, affects the podocyte structure. Scientific Reports, 2020, 10, 21103.	1.6	11
99	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.	1.3	9
100	ARF6 mediates nephrin tyrosine phosphorylation-induced podocyte cellular dynamics. PLoS ONE, 2017, 12, e0184575.	1.1	8
101	Glomerular Disease Workshop. Journal of the American Society of Nephrology: JASN, 2005, 16, 3472-3476.	3.0	6
102	Phosphorylation of slit diaphragm proteins NEPHRIN and NEPH1 upon binding of HGF promotes podocyte repair. Journal of Biological Chemistry, 2021, 297, 101079.	1.6	4
103	APOL1 genotype-associated morphologic changes among patients with focal segmental glomerulosclerosis. Pediatric Nephrology, 2021, 36, 2747-2757.	0.9	3
104	Ret is critical for podocyte survival following glomerular injury in vivo. American Journal of Physiology - Renal Physiology, 2015, 308, F774-F783.	1.3	2
105	Persistent Disease Activity in Patients With Long-Standing Glomerular Disease. Kidney International Reports, 2020, 5, 860-871.	0.4	2
106	Initial Insight on the Determinants of Podocyte Polarity. Journal of the American Society of Nephrology: JASN, 2009, 20, 683-685.	3.0	1
107	Nephritogenic mAb 5-1-6 is directed at the extracellular domain of rat nephrin. Journal of Clinical Investigation, 2000, 105, 125-125.	3.9	0
108	Podocyteâ€specific Vhlh loss demonstrates role for hypoxiaâ€inducible transcription factors (HIFs) in glomerular disease pathogenesis. FASEB Journal, 2007, 21, A504.	0.2	0

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109	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.	0.2	0
110	Podocyte-associated talin1 is critical for glomerular filtration barrier maintenance. Journal of Clinical Investigation, 2015, 125, 882-882.	3.9	0